

# A Case of Acute Pericardial Abscess Due to *Klebsiella Pneumoniae*

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**Abstract:** *Klebsiella pneumoniae* (KPn) is a gram-negative bacterium, which is an opportunistic pathogen commonly found in the natural environment, and the infection rate of *Klebsiella pneumoniae* is increased in patients with underlying diseases such as diabetes mellitus, malignant tumors, liver and gallbladder diseases, and those who have received glucocorticoid therapy for a long period of time [1]-[4], which can cause urinary, respiratory, digestive, hematological, neurological and other multi-system infections with high morbidity and mortality rates [5]. *Klebsiella pneumoniae* infection involving the heart leading to purulent pericardial effusion has a lower incidence, and the literature in this regard reported extremely rare [6], mostly secondary to other parts of the infection, once suspected of pericardial abscess should be immediately performed pericardiocentesis to drain adequately, the application of saline to flush the pericardial cavity, can be thrombolysis of pericardial cavity with antibiotics or urokinase to speed up the process of treatment, to avoid the formation of constrictive pericarditis [7]-[9]. In order to improve clinicians' understanding of this disease, this article reports the diagnosis and treatment of a case of pericardial abscess caused by *Klebsiella pneumoniae*, with the aim of providing more case references for the clinic.

**Keywords:** *Klebsiella pneumoniae*, Pericardial abscess, Multipoint puncture drainage, Endoluminal drug therapy, Constrictive pericarditis.

Clinical Information: Male, 54 years old, diabetes mellitus, hypertension, history of cerebral infarction, long-term smoking and drinking, complained of poor blood glucose control. 2023.03.22, he visited the hospital with the main complaint of "recurrent fever for more than 20 days", and completed the relevant examinations: blood routine: leukocytes:  $23.07 \times 10^9/L \uparrow$ , neutrophil percentage: 86.00 % $\uparrow$ . 86.00 % $\uparrow$ . Serum amyloid A:  $>300mg/L \uparrow$ , PCT: 0.59ng/ml $\uparrow$ , CRP:  $>200mg/L \uparrow$ , ESR: 89mm/h $\uparrow$ . Urine routine: urinary glucose: +4, urinary ketone bodies: +1, microscopic erythrocytes: 2.4/HP, microscopic leukocytes: 81.45/HP, bacterial count: 544.5/u l. BNP: 156.7pg/ml. Chest CT (03.23): Bilateral pulmonary edema; focal left lung distension insufficiency; large cardiac shadows, pericardial effusion, bilateral pleural effusions; cardiac ultrasound (03.23): EF: 54%, thickening of the interventricular septum; small pericardial effusion (fluid dark area echoes were seen anteriorly on the anterior wall of the right ventricle at a depth of about 15 mm and posteriorly on the posterior wall of the left ventricle at a depth of about 15 mm). Diagnosed with urinary tract infection and pericardial effusion, ampicillin sulbactam, moxifloxacin and meropenem were given to fight infection, and the effusion was sent for examination after pericardial puncture and drainage, suggesting exudate, and no pathogen growth was seen. After treatment, the blood test was better than before, and the imaging showed that the lung atelectasis and pericardial effusion were better than before, but there were still intermittent fever, urinary frequency and urgency, urinary pain, and chest tightness and shortness of breath after activity, so he further visited our hospital (2023.04.24), and combined with the results of the outside hospital, levofloxacin was given to empirical anti-infective on admission, and the rest of the symptomatic supportive treatment. At the same time, blood and imaging related examinations were completed: blood routine: leukocytes  $10.21 \times 10^9/L$ , neutrophils absolute value  $7.37 \times 10^9/L$ . Liver biochemistry: ALT: 74.46 U/L, AST: 136.41 U/L, Alb 27.97 g/L, TB 27.13  $\mu mol/L$ . CRP 212.07 mg/L. urine routine+. Urine sediment quantification (ward): WBC/uL 470.25/uL,

WBC/HP 106.88/HP, U\_PC 11.00/uL. sent urine culture suggests *Klebsiella pneumoniae pneumoniae* subspecies Ultra broad-spectrum beta-lactamase negative, drug sensitivity antibiotics all sensitive, replacement of Ertapenem anti-infection treatment. Although actively preserving the liver and reducing enzymes, the patient still intermittently felt nausea and discomfort, and the monitoring of liver enzymes showed progressive elevation. 04.27 morning, the patient suddenly suffered chest tightness and wheezing, finger pulse oximetry decreased, and blood pressure could not be measured for several times, and was given active resuscitation treatment, the patient's vital signs stabilized, and the bedside cardiac ultrasound was immediately improved: LVEF: 50%, and a cystic echogenic area was seen in front of the right anterior wall of the right ventricle (about 13.1\*6.3cm in size). Fluid was seen in the left pleural cavity. Pericardial effusion puncture drainage was performed immediately, and a large amount of coffee-colored purulent fluid was drained. At the same time, PET-CT results returned showed pericardial encapsulated effusion (size about 123\*65mm); consider the possibility of pericarditis or pericardial tumor; consider the possibility of hepatic stasis due to large liver. The fluid showed *Klebsiella pneumoniae* subspecies, mucus type, ultra broad-spectrum beta-lactamase negative, and antibiotic sensitivity. NGS: *Klebsiella pneumoniae*. On the basis of adequate drainage, gentamicin 2 ml was given to flush the pus cavity daily, and a total of about 600 ml was drained, and the patient's symptoms of chest tightness and shortness of breath improved compared with before. Chest CT was repeated on the 7th and 12th postoperative days, suggesting a large amount of effusion on the left side with left lung distension, a small amount of pericardial effusion, and an aneurysm of the right proximal segment of the coronary artery with an attached wall thrombus? Because of the obvious fibrous segregation in the thoracic cavity, there were 4 times of thoracocentesis and drainage, and actively performed thoracic lavage, and sent the pleural fluid suggesting acute inflammation, and cultured *A. baumannii* complex, and the antibiotics were all sensitive. 05.08 sent the urine culture

showed: *Enterococcus faecalis*, and adjusted the antibiotics to meropenem 1.0g q8h. In accordance with the opinion of the MDT, continue to meropenem for anti-infections, and suspend pericardial effusion with gentamicin irrigation, and continue to give warm salt water lavage, and continue to give warm saline irrigation, emphasizing the importance of multi-point puncture. After the patient's condition improved, his temperature stabilized, and the infection index decreased, he was adjusted to amikacin combined with piperacillin tazobactam to fight infection. Repeated delivery of pleural fluid (previous cultures of all-sensitive *Acinetobacter baumannii* existed non-pathogenic possibility) did not show bacterial growth. Repeat chest CT at the local hospital on the 7th day after discharge showed no significant increase in left pleural effusion and pericardial effusion. One month after discharge, he visited our hospital with symptoms of chest tightness and shortness of breath and blood biochemistry tests that were significantly better than before. Cardiac ultrasound: LVEF: 54%; localized calcification of the left ventricular pericardium (possible constrictive pericarditis); decreased amplitude of motion of the left ventricular anterior and lateral walls, and diastolic restriction. Chest CT: left pleural effusion decreased compared to the previous slice, moderate pericardial effusion; nodular shadow at the beginning of the right coronary artery.

**Discussion:** Tuberculous and neoplastic pericardial effusions accounted for nearly 70% of patients with moderate to large pericardial effusions requiring pericardiocentesis drainage in China [10]. The 2021 National Bacterial Resistance Detection Report shows that *Klebsiella pneumoniae* is the second most common pathogen after *Escherichia coli*, with isolation rates of up to 14.12% [11]. However, pericardial abscess caused by *Klebsiella pneumoniae* is rarely reported, and the clinical manifestations are mainly fever, chest tightness, chest pain, shortness of breath, etc. If combined with a large amount of pericardial effusion, it may cause Beck's triad, which is manifested by low blood pressure, low and distant heart sounds, and jugular venous distension [12]. The present study is based on the following findings. Current treatment strategies for symptomatic massive pericardial effusion are centered on etiologic treatment, rational and timely drainage of pericardial effusion by puncture, and reduction of the long-term morbidity of constrictive pericarditis. The main focus of the treatment of symptomatic massive pericardial effusions is to minimize long-term morbidity [10].

The main antibiotics used in the anti-infective treatment of *Klebsiella pneumoniae* are beta-lactams, quinolones, aminoglycosides, polymyxins, tigecycline [13]. With the unregulated use of special grade antimicrobial drugs in recent years, the detection rate of drug-resistant strains has been increasing year by year, and the mechanisms of resistance include the absence of outer membrane pore proteins, the formation of biofilm, the active exocytosis of antimicrobial drugs, and the decrease in the affinity for penicillin-binding proteins [13]-[15]. When strains are isolated from clinical specimens, infection or colonization should be distinguished, and appropriate effective antimicrobial drugs should be selected according to the results of drug sensitivity. The patient was fully sensitive to the drug sensitivity, and therefore could be treated with targeted anti-infective therapy,

but when the strain was insensitive to all the drugs, a high-dose combination of the drugs that showed MIC values closer to the sensitivity cut-off point of the strain could be selected, and the dosing regimen should be decided according to pharmacokinetic and pharmacodynamic characteristics. The decision should be based on pharmacokinetic and pharmacodynamic characteristics, and the drug dose should be adjusted appropriately for hepatic and renal liver damage and elderly patients. In patients with hepatic and renal impairment and in the elderly, the dose should be adjusted [16]. Meropenem and imipenem are commonly used carbapenem antimicrobial drugs, ertapenem because of the lack of activity against *Pseudomonas aeruginosa* and *Pseudomonas aeruginosa*, the use of the clinical use of limited, the case of the patient's initial drug sensitivity results returned in a timely manner after the adjustment to ertapenem 1g, ivvg, qd, and then repeatedly sent to examine the pleural fluid specimens suggesting that the complex group of *A. baumannii* infections, can not be ruled out the causative organisms or the sample contamination of the In this case, the specimen was repeatedly sent for examination and the antibiotic was promptly adjusted to meropenem 1g, ivgtt, q8h. Relevant studies have shown that aminoglycoside monoclonal antimicrobials have achieved good efficacy in patients with carbapenem-resistant *Klebsiella pneumoniae*, who are 80% bloodstream infected. This class of antimicrobials is usually used in combination with other drugs for infections caused by XDR Enterobacteriaceae, *Pseudomonas aeruginosa*, or *Acinetobacter baumannii*. In many countries, the recommended dose of amikacin is 15mg/kg per day, but in our country, the dose is lower. In severely infected patients with normal renal function, 0.8g/day, qd is recommended. After this patient's improvement on ertapenem treatment, he was given amikacin 0.6g, ivgtt, q12h in combination with piperacillin tazobactam 4.5g, ivgtt, q8h for anti-infective therapy. There are a limited number of antimicrobial agents available for use against extensively resistant Gram-negative bacilli; tigecycline and polymyxin are most effective against XDR-GNB based on in vitro susceptibility, but relevant clinical studies have shown a high rate of clinical failure for the above 2 agents.

Early puncture and drainage of symptomatic large pericardial effusion is critical to alleviate the symptoms of cardiac tamponade, while understanding the nature of the pericardial effusion and identifying the cause of the disease is particularly important. In addition to common complications such as perforation of the cardiac chambers, hemothorax, pneumothorax, and cardiac arrhythmia, the formation of a fistula between the pericardial cavity and the pleural cavity, which leads to leakage of pericardial fluid into the pleural cavity and the formation of pericardiothoracic fistula, this is a very rare complication of pericardiocentesis [17]. In this patient, the first review of lung CT after pericardiocentesis was more than that of the left lung at the time of admission, and the pericardio-pleural fistula could not be ruled out except for the sepsis. As far as the management and prognosis of pericardial effusion is concerned, if the etiology of the pericardial effusion is clear, medication should be directed to the etiology of the effusion, and the most commonly used are nonsteroidal anti-inflammatory drugs (NSAIDs), colchicine, and glucocorticoids, and relevant studies have shown that the use of glucocorticosteroids is an independent risk factor for

the recurrence of pericardial effusion, probably because glucocorticoids interfere with the efficacy of colchicine. [18]. Therefore, the use of glucocorticoids as a second-line agent in patients with acute pericarditis in whom NSAIDs and colchicine are contraindicated or in whom treatment has failed should be recommended only for patients with pericarditis due to immune or connective tissue diseases. There was a 32.3% reduction in effusion recurrence to 10.7% after standard therapy plus colchicine treatment [9]-[10]. Intrapericardial thrombolysis is an option because pericarditis leads to fibrin exudation, hyperplasia, adhesions and calcification, and inflammatory damage to pericardial interstitial cells leads to decreased secretion of tissue-type activator of fibrinolytic enzymes, which reduces the degradation of deposited fibrin and induces pericardial constriction [7]. Relevant studies have shown that in patients with septic pericarditis, early intrapericardial urokinase lavage helps to drain the pericardial effusion completely, reduce pericardial thickness, alleviate pericardial adhesions, and effectively prevent the occurrence of constrictive pericarditis [19]. Irreversible pericardial injury characterizes chronic constrictive pericarditis (CP), which makes pericardiectomy the only effective treatment for the disease, and the timing and extent of pericardiectomy are critical to the patient's prognosis. The optimal time for surgery is 3-6 months after constriction. For CP due to septic pericarditis, the left anterolateral open thoracotomy approach should be preferred due to the risk of concomitant pyothorax and sternal infection. The extent of stripping is still controversial, and enlarged pericardial stripping is recommended because of its low recurrence rate, significant cardiac recovery and symptomatic relief, and low dependence on diuretics [20]. This patient was discharged from the hospital 1 month later to review the cardiac ultrasound: localized calcification of the left ventricular pericardium, but LVEF: 54%, daily activities are slightly limited, after communicating with the patient, surgical operation was not considered for the time being, and close follow-up was performed.

The experience of this treatment success case is summarized as follows: (1) For patients who present with sudden chest tightness with dyspnea, clinical attention should be paid to blood glucose, cardiac ultrasound and chest imaging to be alert to the emergence of pericardial pus leading to cardiac tamponade. (2) The patient had progressive elevation of liver enzymes after admission to the hospital, but the patient had no history of hepatitis, no history of alcohol consumption, no history of application of drugs for liver damage, and no obvious abnormalities in immune indexes, so that hepatic stasis due to posthepatic disease (cardiogenic) should be considered after temporarily ruling out hepatogenic factors. [21]. (3) If the patient has a history of sepsis, the patient should be considered to be suffering from a posthepatic disease (cardiogenic). (3) If sepsis or suspected pericardial abscess is present, ideally, pericardial fluid should be drained before starting antibiotic therapy to increase the potential diagnostic yield, and empiric broad-spectrum antibiotic therapy should be given initially afterward, with timely adjustment of antimicrobials based on drug sensitivity results, and to prevent the development of drug-resistant bacteria. (4) Diabetes mellitus is a common causative agent of *Klebsiella pneumoniae* infection, and the treatment is especially critical with puncture and drainage of multiple plasma cavity

effusions, sustained and potent anti-infection, nutritional support, and enhancement of immunity; (5) In this case, a large amount of pericardial effusion was formed after the colonization of the pericardium by *Klebsiella pneumoniae*, which indicated that the infection was systemic and the prognosis was poor, but the drug sensitivities were all sensitive without drug-resistant organisms and the number of times to give the drugs were determined according to the drug's time-dependency and concentration. The number of times the drug is given is decided according to the time dependence and concentration dependence of the drug in order to achieve the best therapeutic effect [22]. The number of doses should be determined according to the time dependence and concentration dependence of the drug to achieve the best therapeutic effect [22]. (6) In addition to strengthening hospital-acquired infection prevention and control measures and the management of the clinical application of antimicrobial drugs, multidisciplinary active participation of clinical pharmacists should be encouraged in the diagnosis and treatment of patients [23].

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