A Rare Case of Rush Progression of Purulent Pericarditis by Escherichia coli in a Patient with Malignant Lymphoma

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Summary
Purulent pericarditis is a rare disease in the antibiotic era. The common pathogens of purulent pericarditis are gram-positive species such as Staphylococcus aureus. Streptococcus pneumoniae, Salmonella, Haemophilus, fungal pathogens/tuberculosis can also result in purulent pericarditis. We report an old male case of purulent pericarditis by Escherichia coli. He came to our hospital suffering from leg edema for 3 months. Echocardiography revealed the large amount of pericardial effusion, and he was admitted to test the cause of pericardial effusion without high fever, tachycardia, and shock vital signs. On the third day, he suddenly presented vital shock. We performed emergency cardiopulmonary resuscitation and pericardiocentesis. Appearance of pericardial effusion was hemorrhagic and purulent. The gram stain revealed remarkable E. coli invasion to pericardial space. Antibiotic therapy was immediately started; however, he died on sixth day with septic shock. The cytological examination of pericardial effusion suggested the invasion of malignant lymphoma to pericardium. This case showed subacute or chronic process of pericarditis without severe clinical and laboratory signs before admission. Nevertheless, bacterial purulent pericarditis usually shows acute clinical manifestation; the first process of this case was very silent. Immunosuppression of malignant lymphoma might make E. coli translocation from gastrointestinal tract to pericardial space, and bacterial pericarditis was progressed to purulent pericarditis. In the latter process, this case showed unexpected rush progression to death by sepsis from purulent pericarditis. Immediate pericardiocentesis should be performed for a prompt diagnosis of purulent pericarditis, and it might have improved the outcome of this case.

Key words: Sepsis, Infectious pericarditis, Purulent effusion

Purulent pericarditis is defined as a localized infection of pericardium characterized by macroscopic or microscopic purulence (>20 leukocytes per oil immersion field). Purulent pericarditis is not synonymous with infectious pericarditis, and only a few cases proceed to purulent effusions.⁷ Before antibiotics have become popular, purulent pericarditis was a frequent disease accompanied with pneumococcal pneumonia. In antibiotic era, purulent pericarditis is now a very rare disease, and a few cases were reported associated with immunosuppression, thoracic surgery, and bloodstream infection. Generally, clinical manifestation of purulent pericarditis is acutely ill with high fever, tachycardia, cough, chest pain, and cardiac tamponade.⁵ The major cause of pathogen is commonly gram-positive bacteria such as Staphylococcus aureus.⁵ The pathogen of subacute or chronic purulent pericarditis is usually tuberculosis.⁷ We report here a case of uncommon pathogen, E. coli and acute on chronic process of purulent pericarditis in an old patient with malignant lymphoma.

Case Report
An 86-year-old man was admitted to our hospital presenting leg edema and general fatigue. His symptom began and gradually progressed for 3 months. He had a medical history of diabetes mellitus (casual blood glucose 264 mg/dL, HbA1c 7.7%). He was afebrile (36.5 °C) on admission, and blood pressure was normal (118/62 mmHg) without pulsus paradoxus. A chest radiograph revealed cardiomegaly (cardiothoracic ratio 68%) and dull costphrenic angle without congestion (Figure 1). Electrocardiography showed normal sinus rhythm (75 bpm), complete right bundle branch block, and low voltage in limb leads (Figure 2). Echocardiography revealed the normal left ventricular function without dilatation; however, a large amount of pericardial effusion was found. Rightsided chamber collapse was not observed at the time of
admission (Figure 3). A chest computed tomography (CT) scan also revealed a large amount of pericardial effusion, swollen hilar lymph nodes, and mediastinum mass (Figure 4). Whole-body CT scan showed no evidence of purulent diseases in abdominal and pelvic organs. Urea and electrolyte tests, liver function tests, thyroid function tests, and cardiac enzyme tests were within normal range. Lactate dehydrogenase (LDH) was elevated at 397 IU/L, and C-reactive protein was slightly elevated (1.4 mg/dL). Serum albumin level was decreased (2.8 g/dL). Full blood count showed mild anemia (Hb 10.3 g/dL), with normal counts of WBC (5,300/μL, neutro 67.3%, eosino 3.4%, baso 0.2%, mono 9.8%, and lymph 19.3%) and platelets (21.7 \times 10^4/μL).

On the admission day, his vital signs were stable, and he did not show any signs of cardiac tamponade. We had planned pericardiocentesis on the third day. However, he suddenly suffered from high fever and sudden cardiac arrest on the third day. We suspected cardiogenic shock due to cardiac tamponade and immediately performed emergency echo-guided pericardiocentesis. Subsequently, a large amount of bloody pus was drained, and his circulation was improved; however, vasopressors were necessary to manage shock state. Direct examination of pericardial fluid smear by gram stain showed a large number of gram-negative bacilli, which were phagocytosed by neutrophils, as seen in Figure 5. The character of pericardial effusion was exudative fluid (LDH 8120 IU/L, albumin 2.1 g/dL). The glucose level of pericardial effusion was extremely low (<1 mg/dL), which was compatible with purulent pericarditis. The examination of blood culture also revealed gram-negative bacilli. He was diagnosed as bacterial sepsis from purulent pericarditis. We immediately started broad antimicrobial therapy (meropenem 1 g intravenously every 12 hours), because the both cultures of pericardial effusion and blood were soon revealed to be positive for *E. coli*, which was sensitive to meropenem. Mycobacterium smear and culture of pericardial effusion was negative. Bacterial polymicrobial infection including mycobacteria was denied. The culture of urine was negative for *E. coli*. He had no manifestation of abdominal infection and abdominal CT revealed no significant sign of abdominal infection. Finally, the source of *E. coli* bactemia was unclear; however, we suspected bacterial translocation from gastrointestinal tract.

Furthermore, the pericardial fluid analysis detected a population of atypical lymphoid cells. Immunohistochemical stains were positive for B-cell-associated antigen; CD79a but negative for CD3, CD20, and CD138. Serum sIL2 receptor was elevated (1880 U/mL). These findings suggested that he had mediastinal B-cell lymphoma. Swollen lymph nodes, posterior mediastinum mass, and pericardial effusion infiltrated by lymphoid cells were only in the supradiaphragmatic side on CT scan. The CT scan findings suggested that his lymphoma was suspected as stage II.

Finally, infection and shock vital could not be controlled and he died on sixth day.

**Discussion**

This is a rare case of purulent pericarditis that is uncommon disease in modern time. Although the major causes of bacterial pericarditis are such as *Staphylococcus*, *Streptococcus*, *Haemophilus*, and *Mycobacterium tuberculosis*, in this case, purulent pericarditis was caused...
by very uncommon gram-negative bacillus, *E. coli*. The unexpected causative bacteria of *E. coli* and sudden clinical changes without a premonitory severe symptom were peculiarity of this case.

This odd clinical process might be caused by malignant lymphoma and the infection of atypical causative bacteria, *E. coli*.

The first process of chronic pericardial effusion was suspected to the invasion of malignant lymphoma to pericardial space. Although more than half of the patients with underlying cancer have no-malignant pericardial effusion,7) he had direct neoplastic pericardial involvement. Before admission, the pericardial effusion probably increased slowly because he did not suffer from cardiac tamponade on admission in spite of a large amount of pericardial effusion. Malignant lymphoma infiltration to pericardium weakened its local autoimmune system and compromised pericardium had made possible bacterial pericarditis. This patient lacked an apparent primary focus of infection by *E. coli*. We suspected the infection sauce was bloodstream bacterial translocation from the gastrointestinal tract to pericardium. However, bloodstream infection by indigenous bacteria such as *E. coli* is hardly caused unless deficiencies in host immune defenses or disruption of the ecological gastrointestinal equilibrium to allow intestinal bacterial overgrowth.8) We could not assess certain lymphoma staging using imaging modality such as gallium scintigraphy and positron emission tomography with CT. He might have inapparent lymphoma invasion to infradiaphragmatic organs, and the lymphoma staging might be more advanced. Advanced lymphoma could suppress systemic immune competence.9) Furthermore, ab-
Figure 4. Chest computed tomography. A large amount of pericardial effusion existed without cardiac chamber collapse. Enhanced lymph node swelling and posterior mediastinum mass (15 mm) were observed.

Figure 5. Pericardial fluid. External appearance of pericardial was bloody pus, and Gram stain showed large number of gram-negative rod (Escherichia coli) and leukocytes phagocytizing these bacteria.
dominal invasion of lymphoma such as gut-associated lymphatic tissue might allow bacterial translocation from gastrointestinal tract to pericardium. Hematogenous translocation of *E. coli* to pericardium must be occurred before admission, because progression of bacterial pericarditis to purulent pericarditis probably needs several days. On admission, he had no fever, no shock vital signs, and tachycardia; these findings showed he had not sepsis at that time. We speculated he had clinically stable purulent pericarditis on admission and complicated with sepsis on the day 3. Finally, sepsis and cardiac tamponade by *E. coli* made him shocked and lethal.

The findings of CT scan on admission suggested that he had malignancy in the lung hilar lymph nodes and posterior mediastinum with pericardial invasion. Pericardiocentesis is an invasive procedure, but pericardial fluid drainage and analysis should be performed for a rapid diagnosis and cure. This case did not show enough pleural effusion for pleurocentesis. Moreover, swollen lymph nodes and mediastinum mass existed in difficult location for simple tissue biopsy. We had no choice but to do pericardial effusion analysis, and pericardiocentesis should be done soon on admission day. We should not hesitate pericardiocentesis and pericardial fluid evaluation even if the patient showed stable vital sign without cardiac tamponade. The pericardial fluid findings of bloody pus and gram-negative bacilli must have made us to start immediate antibiotic therapy, and it could change fatal clinical course of this patient. If early evaluation of pericardial fluid was performed, we might manage to treat purulent pericarditis and sepsis.

Reports of purulent pericarditis by *E. coli* in an adult are very few. We confirmed only two reports of polymicrobial purulent pericarditis by *E. coli* and other bacilli in adult patients. In both cases, pericardial fluid culture revealed the polymicrobial pathogens. One case was induced by *E. coli* and *Proteus* species, and another case was induced by *E. coli* and *Enterococcus faecalis*. This case is the first reported of purulent pericarditis by individual *E. coli* infection and massive *E. coli* infiltration of pericardium confirmed by only smear examination in an adult.

The clinical onset and course of purulent pericarditis of *E. coli* may be subacute and differ from purulent pericarditis of *Staphylococcus*, which shows acute clinical manifestation. The reports of *E. coli* purulent pericarditis in an adult were extremely few, and our consideration about the onset and clinical course of *E. coli* purulent pericarditis is speculation. We could not perform autopsy because of familial decision and confirm the primary infectious focus.

Disclosures

Conflicts of interest: None.

References