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Case Report

Gas-Containing Pericardial Abscess in a Type 2 Diabetic Patient

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Abstract A 63-year-old male with type 2 diabetes mellitus was admitted to our hospital with fever and chest pain. An echocardiogram, chest CT and MRI showed the gas-containing pericardial abscess located posterior to the right atrium. He was initially treated by thoracoscopic pericardial fenestration to set a drainage tube in the pericardial abscess. However, the surgical treatment was discontinued because of a large amount of bleeding from the abscess wall. The patient was then treated by continued administration of antibiotics and γ -globulin. The inflammatory reactions improved and shrinkage of the abscess was confirmed.

Key words : Pericardial abscess, Diabetes mellitus, Drainage

Introduction

Acute pericardial infection in the form of an abscess is quite rare and is a life-threatening condition¹⁾²⁾. It usually occurs after severe vomiting, esophageal instrumentation or mediastinal surgery. A detailed evaluation and pertinent treatment should be initiated promptly. Diabetic patients are known to be more susceptible to bacterial infection³⁾⁴⁾. There are various situations that can cause infections in diabetic patients. We report the clinical and radiological findings of an unusual case of a gas-containing pericardial abscess in a diabetes patient.

Case Report (Fig. 1)

In March 2007, a 63-year-old male with a 10-year history of type 2 diabetes mellitus who had been treated with glimepiride 1 mg daily was

admitted to our hospital with a 2–3 week history of fever and chest pain. He described dull pain across his chest with radiation to both shoulders accompanied by heartburn. On examination, he was sweaty with a temperature of 39.9°C. Blood tests showed an increased concentration of C-reactive protein (CRP : 35.86 mg/dl) and a raised white blood cell count ($15.3 \times 10^3 / \mu\text{l}$, 85% neutrophils). Other blood tests showed a mild elevation of HbA1c (6.9%). An electrocardiogram revealed atrial fibrillation. Lateral chest radiography showed the presence of free air in the middle mediastinum. A transthoracic echocardiogram confirmed the presence of a loculated fluid collection in the mass adjacent to the right atrium with no evidence of any valvular vegetation. Chest computed tomography (CT) showed gas bubbles within a mass, which was approximately 5 x 5 cm with homogeneous density fluid collection, located posterior to the right atrium (Fig. 2A). Furthermore, magnetic resonance imaging (MRI) confirmed the chest CT findings, demonstrating that the abscess was formed in the pericardial cavity (Fig. 2D).

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No aerobic or anaerobic bacteria could be found in the cultures of the urine and sputum. Furthermore, sputum and gastric juice were negative for acid-fast stain (AFS), tubercle bacillus (TB) polymerase chain reaction and TB culture. Repeated blood cultures were also negative during the clinical course.

We initially considered administration of broad-spectrum parenteral antibiotic therapy designed to eradicate aerobes and anaerobes; a 1.0 g/day regimen of panipenem/betamipron (PAPM/BP) and a 1200 mg/day regimen of clindamycin (CLDM), as well as surgical excision of the pericardial abscess. However, the operative method of median sternotomy is very invasive and would certainly have caused osteomyelitis, so we decided to perform thoracoscopic pericardial fenestration in order to set a drainage tube in the pericardial abscess. On the day after admission, we performed thoracoscopic pericardial fenestration and confirmed the presence of the pericardial abscess (Fig. 3A). Incision of the abscess wall with an electrical knife caused a large amount of bleeding (Fig. 3B), so we judged that any further surgery was dangerous and discontinued it.

We decided to perform a surgical operation

only if continued medical treatment yielded no improvement. Because the pus in the pericardial abscess was not able to be collected by the sudden hemorrhage, we could not identify the infecting organism. Then we continued administration of PAPM/BP and CLDM, the inflammatory reactions improved in comparison to those at admission, but there was no improvement from around day 10 of administration of PAPM/BP and CLDM (CRP : 10.0 mg/dl). We considered the participation of methicillin resistant *Staphylococcus aureus* (MRSA) or resistant *Pseudomonas aeruginosa*, we changed the antibiotics regimen to 1.0 g/day of vancomycin hydrochloride (VCM) and newquinolone considering the effect of preventing vancomycin resistant MRSA; 1.0 g/day of pazufloxacin mesilate (PZFX) and also administered of a γ -globulin preparation (5 g/day of venoglobulin IH for 3 days).

The patient's diabetes was adequately controlled during admission. He was treated with short-acting insulin before each meal and intermediate-acting insulin at bedtime. The maximum total daily dose of insulin was 44 units.

The inflammatory reactions improved steadily, with CRP declining to 2.0 mg/dl on day 14 after the start of administration, and both a shrinkage

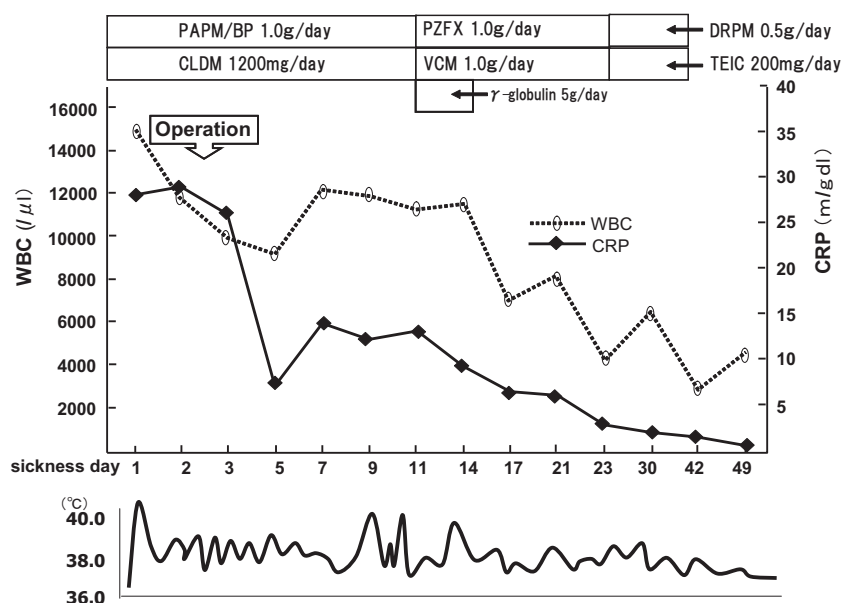


Fig. 1 Clinical course

*The admission day was assumed to be the first sickness day.

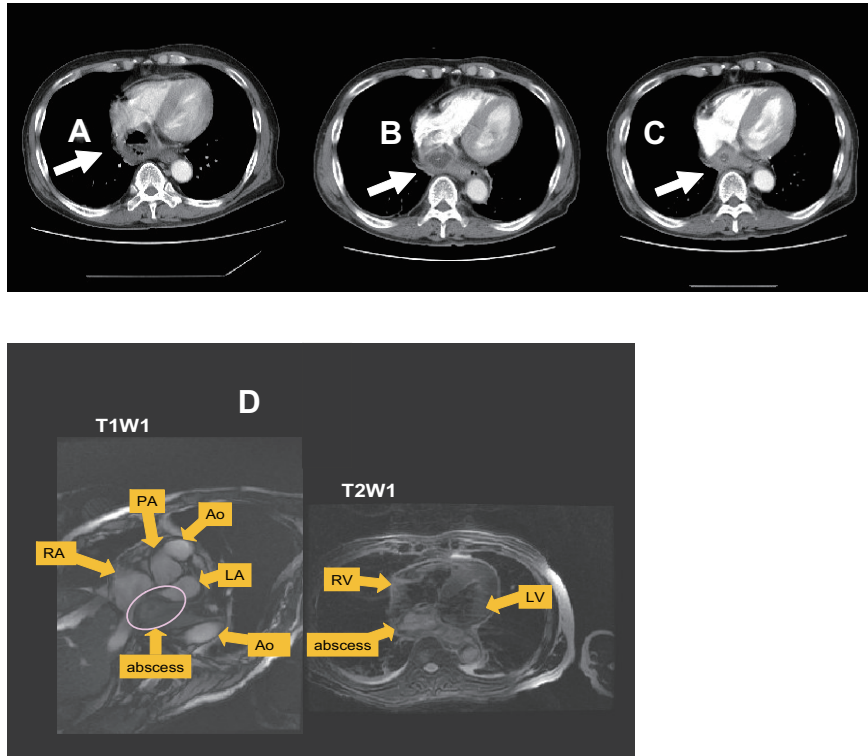


Fig. 2 A, Gas-containing pericardial abscess (arrow) on admission.
 B, Shrinkage of the abscess and the disappearance of gas (CRP : 2 mg/dl)
 C, Further shrinkage of the abscess (CRP : 0.5 mg/dl)
 D, MRI reveals a gas-containing abscess (circle) in the pericardial cavity.

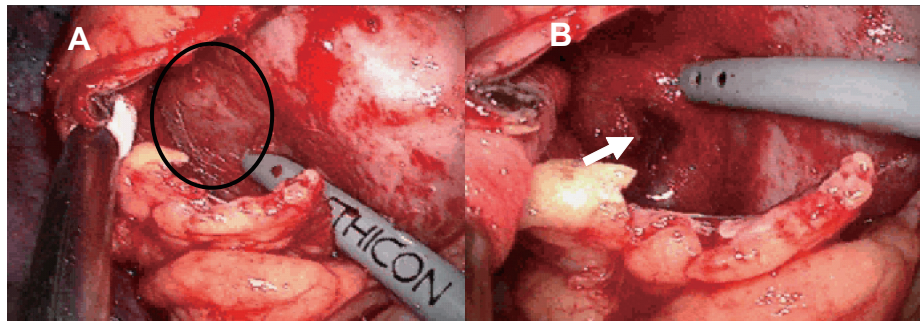


Fig. 3 Photographs of the abscess under the operation
 A, Thoracoscopy showing the presence of the pericardial abscess (circle).
 B, A large amount of bleeding (arrow) caused by incision of the abscess wall.

of the abscess (from 50 mm to 25 mm) and the disappearance of gas were confirmed on a chest CT (Fig. 2B). Parenteral antibiotic therapy should be continued until the patient is afebrile, asymptomatic and without inflammatory reaction. However, because the administration period of VCM and PZFX was prolonged, we changed the regimen to new-generation carbapenem and other anti-MRSA antibiotics ; 0.5 g/day of doripenem hydrate (DRPM) and 200 mg/day of teicoplanin (TEIC) because we were concerned

that microbial substitution might occur, and we once again administered the γ -globulin preparation (5 g/day of venoglobulin IH for 3 days). From day 10 after the start of administration of TEIC and DRPM, there was no fever observed and the inflammatory reactions had further improved (CRP : 0.5 mg/dl). Because further shrinkage of the abscess (10 mm) was confirmed on a chest CT (Fig. 2C), the administration of antibiotics was discontinued. Side effects of antibiotics used in this case such as pseudo-

membranous–colitis and liver dysfunction did not appear during the clinical course. The patient was asymptomatic at the 16–month follow-up.

Discussion

Acute pericardial infection is rare, and it is more often secondarily infected by hematogenous spread, directly spread from adjacent structures, or after cardiac surgery. Furthermore, the loculation of pericardial infection in the form of an abscess is quite rare, but it is a life-threatening condition¹⁾²⁾. It is still difficult to diagnose the nature of a pericardial abscess, although imaging techniques such as CT and MRI have been improved recently⁵⁾. We cannot explain the pathogenesis of our patient's infection, because the infection of the oral cavity, deep neck infection, surgical wound or subphrenic abscess were excluded by the clinical history, physical examination and radiological findings, and there was no evidence for esophageal fistula (In this case, esophagography was performed, but it showed a smooth outline of the esophagus without any apparent contrast medium leakage).

While the common etiologic agents of infective pericarditis have changed from predominantly pneumococcus and streptococcal species to staphylococcus, anaerobes¹⁾, and there have been reports of tubercle bacillus and amebic abscess from liver abscess⁶⁾, especially in an immunocompromised host. Recently gas-forming liver abscess was reported to occur predominantly in diabetic patient and induced mostly by *Klebsiella pneumoniae*⁷⁾⁸⁾. Though the primary cause of the abscess could not be identified in this case because the pus cannot be extracted from the abscess, it is considered that the primary infecting organism with the highest possibility is *Klebsiella pneumoniae*. As another gas production organism with the possibility in this case, it is Enterofacultative anaerobe such as *Escherichia coli* or obligatory anaerobe such as *Prevotella* and *Peptostreptococcus*.

Our case illustrates three important points.

First, although thoracoscopic drainage of a pericardial abscess is a treatment that is relatively non-invasive⁹⁾, there is a possibility of bleeding because of weakness in the cyst wall caused by inflammatory changes, as was seen in our case. Secondly, there are cases in which a pericardial abscess can only show an improvement after conservative medical treatment using antibiotics with an appropriately broad spectrum and immunoglobulin preparations¹⁰⁾¹¹⁾. Thirdly, this case was thought to be a gas-producing bacterial abscess; however, the route of infection was unclear. Diabetic patients are known to be more susceptible to bacterial infection because of an impairment of both the chemotaxis and phagocytosis of leukocytes³⁾⁴⁾⁹⁾. There are various situations that cause infections in diabetic patients, and the pericardial abscess in this case was one of those causal situations.

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(和文抄録)

ガス産生を伴った心嚢内膿瘍を発症した2型糖尿病の一例

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症例は63歳, 男性の2型糖尿病患者. 発熱および胸痛が出現し精査加療目的で当科に入院となった. 心エコー, 胸部CTおよびMRI検査にて右房後方の心嚢内にガス産生を伴った膿瘍を認めた. 入院翌日, 排膿目的で胸腔鏡下で心嚢内膿瘍へのドレナージ挿入術を施行したが, 嚢胞壁に小切開を加えたところ大量の出血が認められ挿入術を断念した. そのため抗菌剤およびγグロブリン製剤の投与による保存的治療に変更した. その効果により次第に炎症反応も改善し, CT上で心嚢内膿瘍は著明に縮小していった.