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Case report

Constrictive pericarditis caused by Cutibacterium (Propionibacterium) acnes: A case report and review of literature

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ABSTRACT

Constrictive and effusive-constrictive pericarditis are rare cardiac disorders. Only rarely are the conditions caused by purulent infection, and even more infrequently by anaerobe bacteria. We describe a case of constrictive – and effusive-constrictive pericarditis due to *Cutibacterium (*formerly *Propionibacterium) acnes* in a 75-year old, immunocompetent and previously healthy patient without any predisposition. The patient was successfully treated with subtotal pericardiectomy and beta-lactam antibacterials. *C. acnes* was the only infectious agent recovered from samples of cultured pericardial tissue.

C. acnes is a microaerophilic, Gram-positive anaerobic bacillus that is a part of the normal flora. In symptomatic patients, however, positive samples should be considered as clinically relevant and not dismissed as contamination. Due to the low virulence, the capability of adherence and biofilm formation of *C. acnes,* diagnosing *C. acnes* constrictive pericarditis may be difficult. In the context of compatible symptoms, the incubation time of clinical samples should be prolonged or supplemented by polymerase chain reaction techniques. Parenteral beta-lactam antibacterials are considered the drugs of choice.

Severe constrictive and effusive-constrictive pericarditis caused by *C. acnes* is rare, but can be seen even in otherwise healthy patients. Prolonged incubation time and polymerase chain reaction techniques may be required in order to confirm diagnosis.

Introduction

Constrictive pericarditis is a rare cardiac disorder characterized by encasement of the heart in a thickened, adhesive, fibrotic, non-pliable pericardium, resulting in impaired diastolic filling of all cardiac chambers leading to heart failure [1]. Constrictive pericarditis can virtually occur after any pericardial disease process. The most common etiologies are idiopathic, viral, post-cardiac surgery, and prior mediastinal irradiation [2,3]. Only three to six percent of cases are due to purulent pericarditis. Anaerobe bacteria are identified as the etiological agents in only a small fraction of these purulent pericarditis cases [2–4].

We describe a rare case of constrictive pericarditis caused by *Cutibacterium acnes (C. acnes)*, formerly *Propionibacterium acnes (P. acnes)* [5], in an immunocompetent patient without predisposition to *C. acnes* infection.

C. acnes has a low virulence, is a skin colonizer, and when isolated from clinical samples, it is often considered a contaminant [6]. We imply that these two factors result in the underestimation of its clinical significance and hence delay in diagnosis and appropriate treatment. In this case report we describe the clinical symptoms, characteristics, major findings and treatments of *C. acnes* associated pericarditis, and review the clinical literature on previously reported cases.

Case

A 75-year old man with hypertension, but otherwise healthy, was referred to our Cardiology department for a tentative diagnosis of congestive heart failure. Symptoms included fatigue, progressive dyspnea, position-dependent chest pain and pitting edema of the lower extremities. The symptoms had developed over a two-month period with worsening of symptoms seven days prior to admission. On admission his blood pressure was 136/65 mmHg, heart rate 90/min and a core temperature of 37.8 ° Celsius. Electrocardiography showed low voltage in the extremity leads, but no signs of arrhythmia or ischemia. Chest radiograph revealed ectasia cordis and left sided pleural effusion. Blood chemistry showed C-reactive protein 51 mg/L with a normal white blood cell count. Transthoracic echocardiography revealed concentric pericardial effusion measuring 16 mm, elevated central venous

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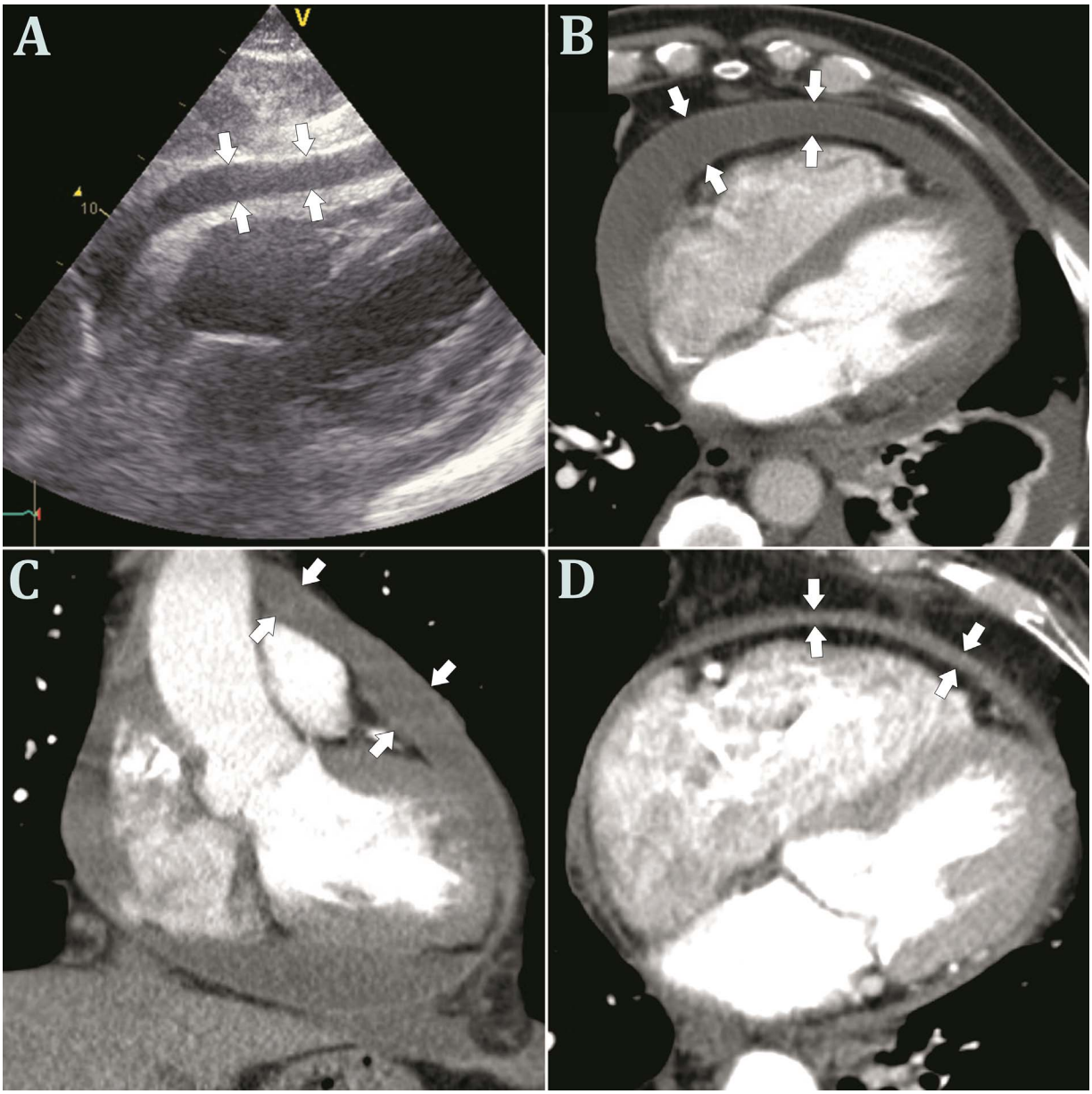

Fig. 1. A: Initial echocardiogram, subcostal view; Pericardial effusion due to effusive pericarditis. B, C: Early CT-scan, axial and coronal views; Severe pericardial effusion, slight thickening of the pericardium. D: Preoperative CT-scan, axial view; Minimal pericardial effusion with gross thickening of the pericardium.

pressure, normal chamber dimensions and wall thickness, no valve disease and normal left ventricular systolic and diastolic function (Fig. 1A).

The patient was tentatively diagnosed with viral pericarditis and parapneumonic effusion and received oral amoxicillin/clavulanic acid, furosemide and ibuprofen as an outpatient. However the patient's condition deteriorated over the following months. Throughout the clinical course, the predominant symptoms remained: asthenia, intermittent chest pain and symptoms of heart failure with dyspnea and pitting edema of the lower limbs.

Diagnostic approach for evaluation of pericarditis included testing for tuberculosis as well as other infectious causes and rheumatic and autoimmune diseases.

Intensified diuretic therapy and pleurocentesis resulted in only transient alleviation of the symptoms. The patient showed signs of progressive heart failure with increasing dyspnoea, excessive edema of the lower extremities and he developed drug resistant atrial fibrillation. Repeat echocardiography showed thickening of the pericardium at four millimeters, pericardial effusion, and signs of the heart being incased in a non-pliable pericardium [7]. Computed tomography (CT) scan revealed thickening of the pericardium, pericardial – and pleural effusions, but no other abnormalities (Fig. 1B, C). Invasive evaluation with simultaneous right – and left-heart catheterization showed an equalization of the end-diastolic pressures at 18 mmHg and pressure tracings showed the classic “Dip-plateau” or “Square-root” filling pattern. Both measurements are distinctive hallmarks of constrictive pericarditis [1,7].

Subtotal pericardiectomy through a median sternotomy was performed. Both the parietal and visceral pericardium showed significant thickening and fibrosis, and the visceral pericardium adhered completely to the myocardium. Postoperatively, the patient recovered rapidly with relief of symptoms. Bilateral cardiac catheterization revealed normalization of end-diastolic pressures and a marked improvement in cardiac output and cardiac index.

Gross examination of the pericardium showed severe fibrosis and thickening, approximately nine millimeters. Microscopic examination

showed chronic inflammation and intense pericardial fibrosis. Tissue samples from both the parietal and visceral pericardium were cultured; *C. acnes* was recovered from two samples. No other infectious agents were recovered. Polymerase chain reaction (PCR) for *Mycobacterium tuberculosis* was negative. Antimicrobial susceptibility testing revealed that is was susceptible to penicillin.

The patient was treated with intravenous penicillin G for four weeks, followed by 12 weeks with oral amoxicillin/clavulanic acid. Six months after surgery the patient remained free of infection, showing continued improvement in overall cardiac function, and a complete remission of both pericardial − and pleural effusions as well as atrial fibrillation.

Discussion

C. acnes is a slow-growing microaerophilic, Gram-positive anaerobic bacillus that is a part of the normal flora of the oral cavity, large intestine, conjunctiva and skin in humans [6]. Prolonged aerobic and anaerobic culture for up to two weeks may be required to isolate the organism from clinical samples [6,8,9]. Most laboratories routinely incubate blood cultures for five days, which may be insufficient for the growth of *C. acnes*. When isolated from clinical samples it is often considered as a contaminant. An increasing number of studies have described *C. acnes* as the cause of serious infections such as endocarditis, prosthetic joint infection, endophthalmitis, osteomyelitis and central nervous system infections. Due to the low virulence, the capability of adherence and biofilm formation of *C. acnes*, these infections, like constrictive pericarditis, are often associated with minimal clinical signs of infection at initial presentation and a diagnostic delay [8–12].

Like other cases described in the literature [4,13,14], our patient's acute effusive pericarditis developed into effusive-constrictive pericarditis, and, subsequently, into constrictive pericarditis requiring pericardiectomy (Fig. 1A–D).

Our case highlights the potential severity of *C. acnes* constrictive pericarditis, but also the difficulty in isolating *Cutibacterium* spp.

We conducted a search for similar cases described in PubMed and Medline databases. Two case reports described *C. acnes* as the cause of constrictive – or effusive-constrictive pericarditis in six patients [15,16]; five were in men and one in a woman. The clinical course of all patients were characterized by minimal signs of infection at initial presentation and a long diagnostic delay, mean 30 weeks, compared to 17 weeks for our case. The predominant symptoms were asthenia, chest pain, palpitations, and symptoms of heart failure. A combination of culture results, echocardiograms, magnetic resonance imaging and cardiac catheterization was used to ascertain the diagnosis. *C. acnes* was penicillin-sensitive in all patients. All patients needed a combination of surgery and prolonged treatment with antibacterials to alleviate symptoms. Three patients experienced relapses requiring further medical treatment including anti-inflammatory drugs, and corticosteroids.

The etiology of constrictive and effusive-constrictive pericarditis is identical. The most common causes of both conditions are: idiopathic and viral infections followed by postsurgical, postradiation, tuberculosis and miscellaneous [3,17]. Only three to six percent of cases are thought to be caused by purulent pericarditis and only a small number of these due to anaerobe bacteria [2–4]. However, a recent study in which samples from 138 patients with infectious pericarditis, 20 of whom had constrictive pericarditis, were examined using prolonged aerobic and anaerobic culture for up to two weeks, revealed *C. acnes* as the etiologic agent in 49 patients. The study included a high volume of cardio-vascular surgeries, prosthetic valve implantations, dental procedures and a high prevalence of immunocompromised patients [13]. *C. acnes* as the causative agent of constrictive and effusive-constrictive pericarditis is probably underestimated.

Untreated, the mortality of bacterial pericarditis approaches 100% [4]. Our patient was treated successfully with subtotal pericardiectomy and beta-lactam antimicrobials. He remained free of relapse after 6 months. Long-term survival for constrictive pericarditis patients with infectious or idiopathic disease is favorable (88%), compared to post-surgical (66%), or postradiation disease (27%) [2,3].

The current expert opinion suggests parenteral penicillin G for initial management of serious *C. acnes* infections, until the antibacterial therapy can be guided by antimicrobial susceptibility [18]. This is substantiated by a study of 304 isolates of *C. acnes* from invasive infections in Europe that found 100% susceptibility to penicillin and vancomycin [19].

We consider *C. acnes* to be an important and underestimated cause of constrictive pericarditis and effusive-constrictive syndrome.

This case demonstrates that invasive infection can occur even in an immunocompetent patient without predisposing factors. Diagnosis may be difficult, but in the context of compatible symptoms, the incubation time of clinical samples should be prolonged or supplemented by polymerase chain reaction techniques. In symptomatic patients, positive samples should be considered as clinically relevant and not dismissed as contamination.

Parenteral beta-lactam antimicrobials are considered the drugs of choice. However, the optimal duration of antibacterial therapy is unknown and should be individualized depending on the clinical circumstances.

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Conflicts of interest

None.

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