Fatal Systemic Nocardia Infection Revealed by Cardiac Tamponade

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Abstract

We report the case of a 50-year-old man admitted for cardiac tamponade. He was diagnosed with acute pneumonia. He had no previous medical history, but exhibited a body mass index of 41. Two days before admission, he complained of chest pain irradiating to the neck lateral side. Massive cardiac tamponade developed over 48 hours. There was no obvious cause for immunodepression. Pericardial puncture was ineffective, due to obesity and fluid high viscosity. Surgery was undertaken (Marfan intervention). Pericardial fluid was found to be purulent; direct examination revealed nocardia as bacteria with typical filamentous, branching rods. Despite adapted antibiotic treatment the patient died within a few hours. Acute pericarditis due to Nocardia is discussed.

Key words: cardiac tamponade, nocardia, pericarditis

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Introduction

Several bacterial infections could lead to pericardial disease. Acute pericarditis due to Nocardia are rare.

Clinical Case

We report the case of a 50-year-old man who was admitted for cardiac tamponade. He had been diagnosed as acute pneumonia and treated with oral amoxicillin for 15 days before admission. His pneumonia was considered to be cured. He had no previous medical history, but exhibited a body mass index of 41. No diabetes mellitus, Cushing syndrome or other disorders which could have led to obesity was noted. Two days before admission, he complained of chest pain irradiating to the neck lateral side. Massive cardiac tamponade developed over 48 hours leading to admission in the intensive care unit.

On admission, dyspnea had worsened; clinical examination showed mild right and left heart failure and tachycardia (97 bpm). Blood pressure was normal (145 mmHg/61 mmHg) without a paradoxical pulse. ECG showed sinus tachycardia and inferior and lateral ST segment elevation.

Echocardiography was performed and revealed large pericardial effusion (6 mm to 22 mm) (Fig. 1B) with evidence of right ventricular compression. Chest CT scan (Fig. 1A) showed a large pericardial effusion and hilar nodular image of 15 mm diameter. The troponin level remained normal and the inflammatory marker, C-reactive protein (CRP) peaked at 246 mg/L (n<5 mg/L). There was no obvious cause for immunodepression: HIV serology was negative, plasma electrophoresis was normal, white blood cell count showed non specific leucocytosis (35,000/mm³) with monocytosis (9,000/mm³) and polynucleosis (24,000/mm³). As a pericardial puncture was ineffective, due to obesity and fluid high viscosity, surgery was undertaken (Marfan intervention), which facilitated complete fluid evacuation. Macroscopic examination was not suggestive of any specific etiology, at first glance. Two days later, pericardial effusion reappeared associated with worsening of clinical symptoms with a high fever and severe sepsis, leading to a second surgical inter-
Pericardial fluid was found to be purulent, and direct examination (Fig. 1C and D) revealed inflammation and nocardia as bacteria with typical filamentous branching rods. Despite adapted antibiotic treatment (sulfamethoxazole + trimethoprim and tigecycline), the patient died within a few hours.

**Discussion**

Here, we report a rare case of severe nocardia infection revealed by initial cardiac tamponade, in a non-HIV patient. Acute pericarditis due to *Nocardia* is scarcely reported (1); the few reports are mainly in immunodeficient patients, such as AIDS (2), or in patients with other predisposing factors like alcoholism (3), or with immunologic disorders such as mixed connective tissue disease (4). Cardiac tamponade is rarely described (5-7). On the other hand, patients with nocardia infection in a context of therapeutic immunodepression can present with pericarditis leading to cardiac tamponades (6). In the present case, surgery showed dense adhesions and adherent pericardium associated with loculations. These pericardial abnormalities surgical necessitate exploration in such patients, and the findings have revealed that transthoracic puncture is scarcely feasible, due to the dense adhesions.

In the present case, cardiac tamponade revealed nocardia infection in a non-immunocompromised patient, without any other specific signs. Surgery was then mandatory in order to determine the etiology, yet the life-threatening clinical situation resumed. The poor evolution underlines the interest of etiological findings in this pathology, particularly in cases of potentially curable diseases, such as infectious or hematological diseases. Most of the time, culture of pericardial fluid or even pericardial tissues leads to a diagnosis. In the present patient, the first surgery did not reveal sufficient evidence to lead to a diagnosis. This first intervention might have enabled bacterial growth, although this was not confirmed, we can assume on the contrary that this reflected merely the natural evolution of the infection. Direct examination of the second intervention immediately revealed nocardia, illustrating evidence of massive inoculum. This point deserves to be underlined, as it could explain the deterioration and very rapid lethal outcome.

**References**


