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Correspondence

An oil refinery worker at Kaohsiung, with *Pseudoxanthomonas kaohsiungensis* bloodstream infection presenting as chronic pericarditis and masquerading as tuberculosis pericarditis

Dear Editor,

Pseudoxanthomonas kaohsiungensis was first reported in 2005, and was recovered from an oil-polluted site near Kaohsiung city in southern Taiwan, where it seems to an environmental organism.[1]

A 28-year-old man with 3 months of chest heaviness presented with one week of fever. This oil refinery worker at Kaohsiung, Taiwan, had received 3 years of renal replacement therapy for vesicoureteral reflux. Chest radiography showed bilateral costophrenic angle blunting (Fig. 1A). Echocardiography showed a thickened pericardium with an effusion, severe right ventricular hypokinesis, and preserved left ventricle systolic function. Straw-colored fluid with 950 leukocytes/μL (polymorphonuclear leukocytes [PMNs] 22%, lymphocytes 45%, and monocytes 33%) was drained via thoracotomy. Acid-fast bacillus staining of sputum and pericardial fluid was negative. Blood culture yielded no growth. Polymerase chain reaction testing was negative for *Mycobacterium tuberculosis*. Pericardial biopsy revealed acute and chronic inflammatory cell infiltration. Anti-tuberculous chemotherapy plus adjuvant corticosteroid therapy were prescribed with the impression of tuberculous pericarditis. However, fever and dyspnea on exertion developed after the steroid dose was reduced. Echocardiography revealed limited pericardial motion with a small effusion. Pericardiectomy was performed for effusive-constrictive pericarditis. Pericardial fluid revealed 612 leukocytes/μL (PMNs 2%, lymphocytes 59%, and monocytes 39%). Aerobic and anaerobic cultures were still negative. Blood cultures became positive for a Gram-negative bacillus after 4 days of incubation (BACTEC™ FX; BD Diagnostics, Sparks, MD, USA). Subcultures on blood agar yielded circular, convex, smooth, golden-yellow, non-hemolytic colonies but poor growth on EMB agar after 24 h of incubation at 35 °C with 5% CO_2. These colonies increased in size to about 1.5–2.5 mm in diameter after 72 h of incubation. *P. kaohsiungensis* was identified by matrix-assisted laser desorption ionization-time of flight mass spectrometry (Bruker Daltonik, Bremen, Germany) using Bruker Biotyper 3.1 System. The identification score value was 2.36 and its identification was subsequently confirmed by 16S rRNA (99.0% identical; accession number NR043070). Susceptibility (Fig. 1C) was assessed using the Phoenix automated microbiology system (BD Diagnostics). Categorical interpretations for minimal inhibitory concentration tests used the Clinical and Laboratory Standards Institute (CLSI) criteria for other non-Enterobacteriaceae.[2] Fever subsided on the third day of using ceftazidime. Echocardiography after 2 weeks of ceftazidime revealed slight pericardial sliding, little pericardial effusion, and inferior vena cava collapse with inspiration. The patient was discharged with oral ciprofloxacin for 2 weeks. He remained well, and the pleura-pericardial effusion resolved after 6 months (Fig. 1B), with no constrictive findings.

Purulent pericarditis can be caused by bacterial, viral, mycobacterial, and fungal infection.[3] This patient had more than 3 months of symptoms, suggesting chronic infection by an agent with low virulence. A common etiology of chronic pericarditis in immunocompromised hosts is mycobacterial infection.[4] Non-resolution of symptoms and the presence of effusive-constrictive pericarditis after treatment suggested an alternative etiology. *P. kaohsiungensis* was probably not

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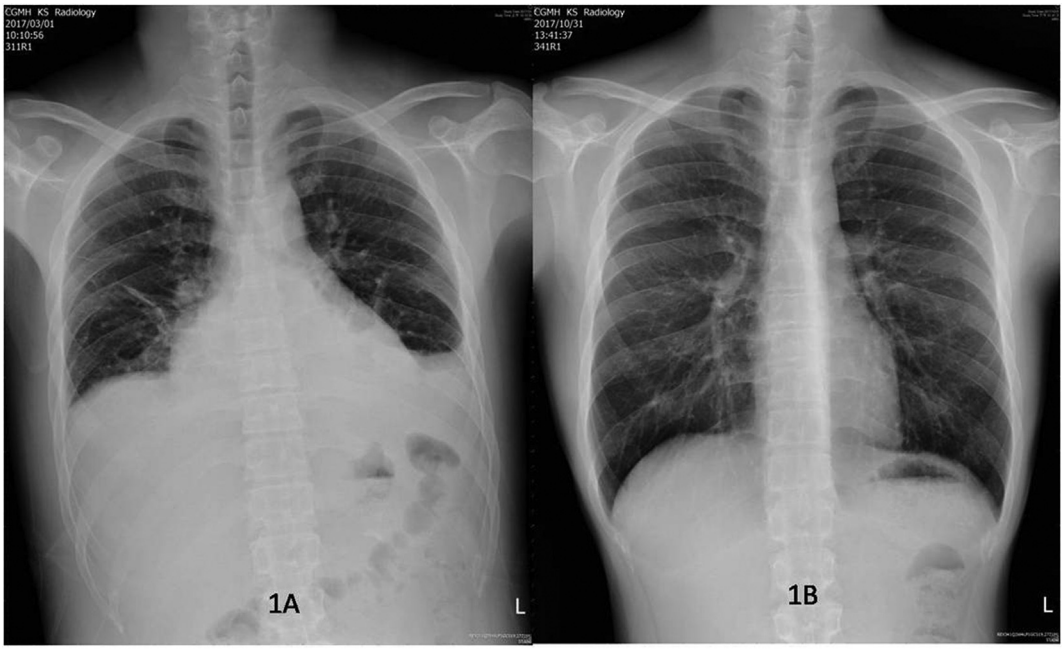

1C. Antibiotic susceptibility of *Pseudoxanthomonas kaohsiungensis*

Antibiotic	MIC (mg/L)	interpretation[b]
Amikacin	32	I
Aztreonam	16	I
Cefepime	<=2	S
Ceftazidime	<=4	S
Ciprofloxacin	<=0.5	S
Gentamicin	8	I
Imipenem	<=1	S
Levofloxacin	<=1	S
Meropenem	<=1	S
Piperacillin-tazobactam	<=4/4	S
SXT[a]	1/19	S

[a]SXT, Trimethoprim-sulfamethoxazole; S, susceptible; I, intermediate.

[b]Categorical interpretations for minimal inhibitory concentration (MIC) tests were the CLSI interpretative criteria for other non-Enterobacteriaceae including *Pseudomonas spp*.

Figure 1. (A) Chest X-ray showed cardiomegaly with a water bottle-sign and bilateral costophrenic angle blunting. (B) Follow-up chest X-ray 6 months later showed resolution of the pleura-pericardial effusion. (C) Antibiotic susceptibility of *Pseudoxanthomonas kaohsiungensis*. Categorical interpretations for minimal inhibitory concentration (MIC) tests used the CLSI criteria for other non-Enterobacteriaceae including *Pseudomonas spp*.

cultured from pericardial fluid because agar plates are often discarded after 3 days of incubation. The presence of pericardial effusion coincided with bacteremia in this oil refinery worker that resolved on antibiotics, making *P. kaohsiungensis* the likely cause of pericarditis. The evidence suggests that this organism is a potential human pathogen in an immunocompromised host, rather than simply a biosurfactant-producing bacterium in the environment.[5]

Ethics approval

Ethics approval was not required for this study.

Conflicts of interest

The authors have no conflicts of interest to declare.

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