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CASE REPORT

# An unusual case of pericarditis caused by *Cardiobacterium hominis*

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*Cardiobacterium hominis* is a Gram-negative coccobacillus, which is part of the normal human oropharyngeal flora. *C. hominis* is recognized as a rare cause of human infection leading to endocarditis and focal infections outside the vascular system.[1,2] To our knowledge, there is no reported case of *C. hominis* pericarditis without endocarditis.

A 10-year-old girl was admitted to our hospital with the complaints of fever, sweating, palpitations and chest pain lasting for 1-week. Her physical examination revealed a body temperature of 39 °C, decreased breath sounds in the left lower lobe of the lung and decreased heart sounds. Blood pressure was 90/60 mmHg and pulse was 88/min. There was no pulsus paradoxus. The liver edge was 2 cm palpable. Other systems' examination were normal. Laboratory analysis revealed white blood cell count: 10 000/mm,[3] antistreptolysin O: 415 IU/ml, CRP >201 mg/dl and erythrocyte sedimentation rate: 92 mm/h. There was marked enlargement of the cardiac silhouette on telecardiogram. Echocardiographic examination demonstrated massive pericardial effusion. There were no findings of echocardiographic pericardial tamponade. Left ventricular diameters and ejection fraction were also normal. There were no valvular dysfunction and no demonstrable vegetation in any of the heart valves on transthoracic and transesophageal echocardiography. Computed tomography of the thorax showed pericardial effusion and left sided pleural effusion, therefore 1000 cc of serous pericardial fluid was drained by partial pericardiectomy and 250 cc of serous pleural fluid was drained by thoracentesis. Histopathologically, the surgical specimen revealed purulent exudative membranes. Pleural and pericardial fluids were cultured. Before identification of the etiologic agent, broad spectrum antibiotic was started empirically (vancomycin 40 mg/kg/day parenterally and cefotaxime 150/mg/day parenterally) against the most common micro-organisms possibly causing, bacterial pericarditis. Although the pleural fluid and blood cultures were negative, culture of the pericardial fluid revealed *C. hominis*. Once culture and susceptibility testing were available antibiotic therapy was continued with cefotaxim alone for two more weeks. At the end of 4 weeks of antibiotic treatment, all laboratory measurements were in the normal range and there was no pericardial fluid collection on echocardiographic examination.

Pericardial and pleural fluid were incubated on trypticase soy agar with 5% sheep blood, chocolate agar, Eosin methylene blue agar (EMB) and Sabouraud agar at 35 °C in an atmosphere of 5% carbon dioxide. On the 3rd day of incubation partially

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sunken small gray colonies were observed on the blood and chocolate agar, however definite identification and result of susceptible testing was revealed on the 12th day. Rods swollen on one or both ends were seen after Gram-staining of the colony. The isolate was oxidase-positive, catalase-negative, nitrate-negative, indole-positive, urease-negative and non-motile. The organisms fermented glucose, sucrose and lactose. The biochemical profiles (BBL Neisseria/Haemophillus ID System BioMerieux) of the isolate were consistent with the identification of *C. hominis*. Three blood samples were taken from the patient and inoculated into BACTEC 9120 (Becton Dickinson and Company, USA) automatic blood culture bottles. All blood cultures were negative for organisms after 5 days of routine incubation. Since there is no standard antibiotic susceptibility testing for *C. hominis*, antibiotic susceptibility testing used for *Streptococcus pneumoniae* was performed on 5% sheep blood Muller-Hinton agar with Kirby Bauer Disk Diffusion Method as recommended by 'National Committee for Clinical Laboratory Standards'.[3] *C. hominis* was susceptible to all antibiotics used in the test (vancomycin, tetracycline, cefotaxime, trimethoprim-sulfamethoxazole, penicillin and erythromycin) except chloramphenicol.

By nature, *C. hominis* grows slowly in culture media and is an organism of relatively low virulence. The organism is facultative anaerobic and requires $CO_2$ and humidity for growth on solid microbiologic media. Tiny, glistening, opaque colonies are observed on 5% sheep blood agar for more than 2 days. In Gram-stain, *C. hominis* is seen singly or in pairs and short chains, showing bulbous ends.[4] The main characteristics of *C. hominis*, distinguishing it from other closely related organisms, are the absence of catalase activity, positive oxidase reaction, production of indole and the absence of nitrate production.[4]

*C. hominis* was first isolated from four patients with infective endocarditis.[1] The infection manifests itself either as infective endocarditis or associated complications, such as acute mitral insufficiency,[5] septic arthritis,[6] heart failure[7] or rupture of mycotic aneurysm.[8] Most of the cases have had pre-existing cardiac disease involving prosthetic valves and permanent pacemaker implantation.[9] Few cases of *C. hominis* infection occurring outside the vascular system have been reported, including bacterial meningitis,[2] abdominal abcess[9] and osteomyelitis.[10]

According to our knowledge, this is the first report a case of isolated pericarditis due to *C. hominis* in the absence of infective endocarditis and detectable vegetation. This case underlines that *C. hominis* can cause pericarditis and pericardial effusion without affecting the cardiac valves. *C. hominis* should be kept in mind in the presence of findings suggesting bacterial infection and pericardial effusion.

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