

Case Report

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Pseudomonas mendocina as an Agent of Bacteremia, Case Study and Literature Review

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Abstract

Pseudomonas mendocina is a Gram-negative, non-fermenting rod bacterium. It is generally isolated from soil and water samples. So far, it has been isolated from several human clinical samples. We report the seventh case of infection in human caused by this uncommon organism.

Keywords: Pseudomonas mendocina; Bacteremia; Antibiotic

Introduction

Pseudomonas mendocina is a Gram-negative, non-fermenting rod bacterium. The Pseudomonas family is known to comprise 191 species characterized by great variation with regard to their metabolic capabilities. The bacteria are most commonly found in humid areas such as soil, water sewage and vegetation. Their ability to fix nitrogen obtains iron from compounds, which are unavailable for other microorganisms via their capability to produce siderophores, allow them to survive in nutrieny-poor substances. The microorganisms have the capability to feed on organic substances which are harmful to the environment such as diesel oil and other organic hydrocarbons. In addition, the bacteria have the ability to produce exopolysaccharides such as alginic acid, which are involved in the production of biofilms. Some of these species may also be responsible for opportunistic and nosocomial infections.

Pseudomonas mendocina is an example of a non-fastidious organism. As the one representative of this group, it produces carotenoid pigments rather than florescent pigments, as other species do. It produces extracellular lipases characterised by a low substrate specificity, which makes it possible to hydrolyse fats with a range of compositions. These enzymes are resistant to the action of surfactants [1-5].

The name of the organism derives from its first isolation from water and soil samples in Mendoza, Argentina by Palleroni, in 1970 [6]. It is rarely found to be a human pathogen – it was not isolated from human blood until about 21 years later. Until now, only 6 cases of infection by *Pseudomonas mendocina* have been reported in humans; the example described in this report is only the 7th infection to be described [7-12].

Case Report

Pseudomonas mendocina was isolated from a blood sample taken from a 58-year-old man diagnosed with respiratory failure, cardiomyopathy and suspected septic process. A chest X-ray identified fluid in the pleural cavity extending to the second rib. A dignostic Ultrasonography (USG) examination of the heart revealed Ejection Fraction (EF) 25-28% and pericardial effusion. Other tests revealed the presence of elevated levels of inflammation biomarkers: 13.2 ng/ml procalcitonin, 554.65 ml/l C-reactive protein (CRP) (Figure 1). Biochemical parameters: K+, urea, creatinine show respectively (Figures 2-4). Because of fever, dyspnea, cough with expectoration of purulent secretions and haemoptysis, the patient was prescribed Rovamycine 112.500 IU/kg each 8 hours. The patient was monitored throughout his stay in intensive care, and was breathing on his own with passive oxygen insufflation. A route to the central vein was opened and blood was drawn directly to BD (Becton Dickinson) medium for aerobic and anaerobic blood culture. The medium was placed in a





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Bactec automatic Blood Culture System (BD).

A combination of 600 mg twice a day Linezolid and 400 mg each 8 hours Ciprofloxacine was administered. All drugs were administrated intravenously. An X-ray, an ultrasound and computed tomography scans (CT) of the chest were performed, as well as an ultrasound examination of the abdominal cavity. Based on the presence of fluid identified in the pleural cavity and the clinical condition of the patient, a drain was inserted in the right pleural cavity, which was removed after a few days. FFP was transfused to stabilize coagulation parameters. In response to such emerging sings of renal failure as elevated creatine, urea and oliguria levels, diuretic treatment was intensified.

Gram-negative rods were found in all blood samples taken from the patient. Yellow pigmented colonies were found in the agar medium inoculated with the blood samples. Gram staining revealed the presence of Gram-negative bacilli. The identification of the bacteria was performed with the use of a API 20E (Biomerieux) and Phoenix kits (BD). The cultivated bacterium was found to be *Pseudomonas mendocina*. Drug sensitivity was evaluated by disc-diffusion on Mueller-Hinton Agar (BD) with the use of the E-test (Biomerieux). The results revealed *P. mendocina* to have the following MIC values for the following drugs: ceftazidime (MIC=0.75), piperacillin (MIC=1), piperacillin/tazobactam (MIC=1), ticarcillin (MIC=1), ticarcillin / clavulanic acid (MIC=1), imipenem (MIC=0.19), meropenem (MIC=0.19), gentamicin (MIC=0.5), amikacin (MIC=1), ciprofloxacin (MIC=0.125), levofloxacin (MIC=0.094).

After receiving the results of the drug sensitivity tests, the empirical antibiotic therapy was changed to the targeted one using Levofloxacin 1 x 125 mg. It resulted in a significant clinical improvement. The patient remained in a moderately severe state, but with a stable circulatory system breathing and correct diuresis. After the test results normalised, the patient was transferred to the Department of Internal diseases, asthma and allergy for further treatment.

Discussion

This study documents the 7th recorded infection of *Pseudomonas mendocina* of the previous cases, three involved patients with endocarditis, one with spondylodiscitis and two who had not suspected any such disease. The bacterium was isolated from blood samples in 5 cases, and from mucus in one case, where the blood was not tested.

In the present study, *Pseudomonas mendocina* was isolated from a blood sample. The case is the third example of sepsis as a result of *P. mendocina*, and the fourth example of it being found in a patient with cardiomyopathy. The treatment was based on antibiotic therapy with penicillins, cephalosporins, aminoglycosides and fluoroquinolones. The therapy lasted from 2 to 7 weeks. The present case affected a 58-year-old patient with breathing problems and cardiomyopathy, who was administered linezolid and ciprofloxacin. After receiving the result of the susceptibility testing, linezolid was replaced with levofloxacin, which was then applied for 6 days as oral therapy.

The considerable pathogenic potential of *Pseudomonas* is believed to be associated with the synthesis of a range of virulence factors such as proteolytic enzymes, lipolytic enzymes and exotoxins. The bacteria also have the ability to produce an alginate, which is a component of the biofilm. Bacteria which produce a biofilm are more resistant to temperature and such antibacterial agents as disinfectants, surfactants and antibiotics.

Pseudomonas bacteria produce a number of pigments: bluegreen pyocyanin, fluorescent green pyoverdine, red piorubrin and brown melanin, as well as carotenoids. These pigments facilitate the pathogenicity of the bacterium and allow it to effectively counter the action of the host defence system [13-15]. Carotenoid pigments, which are also produced by *P. mendocina*, are powerful antioxidants which are readily oxidized and thus absorb the energy of Reactive Oxygen Species (ROS). The effectiveness of these pigments makes the cells more resistant to the damaging effects of hydrogen peroxide, hypochlorite and free radicals, and allows more bacteria to survive in neutrophils, which represent the first line of defence against pathogens (Table 1).

A limited amount of ROS are formed in eukaryotic cells as natural products of metabolism and these fulfil important functions in mitochondrial oxidative phosphorylation, apoptosis and blood clotting. In addition, increased ROS concentration is a typical response to bacterial infection, known as oxidative stress, and is an important part of the defence against infections. However, massive post-infection oxidative stress has many destructive effects: considerable increases in ROS represent a major threat to health and can lead to the apoptosis of neutrophils. Cytokine secretion resulting in suppression of the immune system has also been noted.

Carotenoid pigments, which neutralise free radicals, protect *Pseudomonas mendocina* against the immune system. It has been shown that pigmented strains demonstrate greater virulence than

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References	Age/ gender	Underlying	Symptoms	Isolation disease	Diagnosis and signs	Therapy site	outcome
[7]	63 M	poliomyelitis, diabetes mellitus, aortic valve replacement	fever, chills	blood	infective endocarditis	ceftriaxone + gentamicin (6wks) followed by oral ciprofloxacin (2wks)	survival
[8]	28 F	situs inversus, ventricular septal defect, dacron	abdominal pain, dyspnea, flu-like syndrome	blood	infective endocarditis	ampicillin + gentamicin followed by oral ciprofloxacin (7wks)	survival
[9]	65 M	renal disease, alcoholism	lower back pain	deep tissue pus	spondylodiscitis	cefepime followed by ciprofloxacin (7wks)	survival
[10]	36 M	mentally retarded	fever, weight loss	blood	infective endocarditis	ceftazidime + amikacin (6wks)	survival
[11]	31 M	none	fever, chills	blood	bacteremia	ceftazidime + oral ofloxacin (2wks)	survival
[12]	79 F	geriatric patient	fever	blood	bacteremia	piperacillin + gentamicin (6wks)	not specified
This case	58 M	respiratory failure, cardiomyopathy	fever, shortness of breath	blood	bacteremia	linezolid + ciprofloxacin followed by levofloxacin	survival

Table 1: Clinical manifestations and outcome of reported cases of Pseudomonas mendocina infection.

non-pigmented strains. However, despite the presence of such a large number of pathogenic factors, this kind of infection agent rarely infects healthy people due to their poor ability to colonise and destroy epithelial cells. Nevertheless, serious infections such as endocarditis or bacteremia can develop in immune compromised individuals. These infections are of a hidden character, and although their mode of entry to the blood is unknown, a causative factor may be the presence of carotenoid pigments which allow insensitivity to immune defence mechanisms. Hence, *Pseudomonas mendocina* is a classic example of an opportunistic pathogen which is a threat to people with weakened immune systems [13-15].

None of the cases of human *Pseudomonas mendocina* infection given above provide any information regarding the source of infection of their mode of transmission. This study highlights the significant role played by microbiological diagnostic tests in the rationalization of the therapy.

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