

### ***Corynebacterium xerosis* as a Cause of Community-Acquired Endocarditis**

Corynebacteria are gram-positive, nonmotile, nonsporulating, catalase-positive rod-shaped bacteria that often appear pleomorphic on Gram stain. The clinical significance of the nondiphtheria corynebacteria and their pathogenicity for man has only been recognized in the last decades (1, 2, 3). *Corynebacterium xerosis* is a normal commensal of the conjunctivae, nasopharynx and skin (1, 2). We report a case of community-acquired endocarditis caused by this bacterium in a man with no history of heart disease who was admitted to hospital presenting with cerebral infarct.

The patient, a 51-year old man, had twice undergone abdominal surgery 20 years previously for gastric ulcers and had since been healthy. Despite a history of alcohol abuse, in recent years the patient's consumption of alcohol had been modest. A simple, non-infected bursitis at the right had been aspirated by his general practitioner three weeks prior to admission; no antibiotic was administered at that time. Shortly afterward and until admission the patient experienced low-grade fever and general malaise. He was referred to the neurological department with a one-day history of dysarthria and paresis of the right arm. Neurological examination revealed supranuclear paresis of the right arm and central paresis of the right facial nerve. Computerized tomography of the cerebrum showed an infarct in the left hemisphere.

During hospitalization the patient was febrile with a temperature ranging between 37.8 °C and 39.8 °C. The leukocyte count in peripheral blood was  $10.5 \times 10^9/l$  on the day of admission and rose to  $23.1 \times 10^9/l$  on day 12. The erythrocyte sedimentation rate was 107 mm/h on the day of admission. Aside from the neurological symptoms the patient had no complaints and physical examination revealed no cause of the pyrexia. No peripheral embolic manifestations could be found on the skin, buccal mucosa, palate, conjunctivae or retinae. The teeth, however, were in poor condition.

Infection was suspected and six blood cultures performed between days 6 and 12 all yielded *Corynebacterium xerosis* within 48–72 h. On day 14 a cardiac murmur was detected and the diagnosis of infective endocarditis was confirmed by echocardiography, which revealed a vegetation on the posterior mitral valve. Treatment was initiated with  $2 \times 10^6$  U of benzylpenicillin t.i.d. in combination with 160 mg of tobramycin b.i.d. The patient improved and his circulatory condi-

tion remained stable, but when an echocardiogram obtained eight days later showed marked enlargement of the vegetation, he was transferred to the department of cardio-thoracic surgery. Thirty-four days after admission, cardiac surgery was performed with insertion of a prosthetic mitral valve. At operation a vegetation  $1 \times 1 \times 1.5$  cm in size was found on the posterior mitral valve, cultures of which were negative.

The postoperative course was uncomplicated. Antibiotic therapy with  $5 \times 10^6$  U of benzylpenicillin t.i.d. and gentamicin (based on trough serum concentrations) was continued until the patient was discharged one month later. Four months after being discharged the patient was in good health, had experienced no febrile episodes and had no cardiac complaints. Neurological symptoms had disappeared.

Taxonomic confusion regarding the grouping and species classification of the nondiphtheria corynebacteria still prevails. This is in part due to the fact that many of these organisms lack activity in conventional biochemical tests. Our isolate was characterized as *Corynebacterium xerosis* on the basis of the following properties. The strain was a nonmotile, gram-positive, short coccobacillar rod. On blood agar the colonies were approximately 1 mm in size at 24 h, raised and greyish-white, with a smooth creamy appearance; there was no hemolysis. On tellurite agar the colonies appeared flat and dark greyish. The isolate was able to reduce nitrate but was unable to hydrolyse urea or liquefy gelatin; it fermented glucose, saccharose and galactose, but not trehalose, maltose or lactose. These properties are in accordance with those described previously (4). In vitro susceptibility tests using a disk-diffusion technique (Rosco tablets, Rosco A/S, Denmark) showed that the organism was highly sensitive to penicillin, ampicillin, dicloxacillin, piperacillin, cefuroxime, cefotaxime, tetracycline, erythromycin, vancomycin and tobramycin.

The most commonly reported human infection caused by the nondiphtheria corynebacteria is infective endocarditis (1). Penicillin in combination with an aminoglycoside seems to be a rational initial regimen, but therapy should be guided by antimicrobial susceptibility testing (2, 3, 5, 6). The few infections proven to have been caused by *Corynebacterium xerosis* have either been in patients with serious underlying disease (7) or endocarditis occurring as a complication following aortic valve replacement (6). It is of note that our patient was a middle-aged man who, apart from a history of prior alcohol abuse, had no signs of underlying disease or compromised host defences. Furthermore, the

infection was community-acquired. As *Corynebacterium xerosis* is a normal commensal of the skin, it is possible that the aspiration of the the bursitis, which preceded the hospital admission, was followed by a transient bacteremia with subsequent dissemination to the mitral valves. Another possible source of the infection was the teeth.

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## *Proteus mirabilis* as a Cause of Recurrent Lung Infection in a Cystic Fibrosis Patient

*Proteus mirabilis* is rarely isolated from the bronchial secretions of cystic fibrosis patients. It is considered a transient colonizer of the bronchial secretions, generally being far outnumbered by *Pseudomonas aeruginosa*. To our knowledge there are no previous reports of the isolation of *Proteus mirabilis* as a single or overwhelmingly predominant organism during

very long periods of time in this type of patient, particularly in recurrent episodes of pneumonia (1, 2, 3).

An 18-year-old woman suffering from chronic respiratory disease since early childhood was referred to our hospital with a diagnosis of cystic fibrosis and acute respiratory tract infection. Two sweat tests revealed high chloride and sodium concentrations of over 120 meq/l. Steatorrhea and hyperamylasemia (1,084 IU/l) were also documented. During the previous two years she had been treated with tetracycline, amoxicillin and amoxicillin-clavulanate. When the patient presented to our outpatient department, *Proteus mirabilis* was isolated from a homogenized sputum sample (95 % of the cells were polymorphonuclear neutrophils) in pure culture on CLED medium (Oxoid, UK), with a count of  $3 \times 10^5$  CFU/ml; treatment with cotrimoxazole was started.

The patients's condition worsened and she was admitted to hospital. At this time she was suffering from a productive cough, dyspnea and fever (39 °C). The leukocyte count was 24,000/mm with neutrophilia and 13 % band forms. Chest X-ray revealed disseminated cystic bronchiectasis with multifocal pneumonia. *Proteus mirabilis* was again isolated from sputum, with a count of  $6 \times 10^6$  CFU/ml. The strain exhibited resistance to amoxicillin, tetracycline and cotrimoxazole. A variant of the same strain exhibited resistance to ticarcillin, cephalothin and cefazolin, and reduced susceptibility to amoxicillin-clavulanate.

*Aspergillus fumigatus* was also isolated from the sputum. Specific anti-*Aspergillus fumigatus* IgE and IgG levels were significantly increased, suggesting the presence of allergic bronchopulmonary aspergillosis, a disease present in about 10 % of cystic fibrosis patients. The serological response to the homologous *Proteus mirabilis* strain was tested: anti-H and anti-O agglutinating antibodies were detected at titres of 1:80 and 1:320, respectively. The acute exacerbation was treated with oral ciprofloxacin (750 mg b.i.d.) for ten days. One month later there was a significant increase in anti-H antibodies (1:1,280) and maintenance of anti-O antibodies (1:320).

The overall development of *Proteus mirabilis* counts in sputum is presented in Table 1. In June a small number ( $2 \times 10^2$  CFU/ml) of an arginine dehydrolase-positive, lysine and ornithine decarboxylase-negative *Pseudomonas* strain was isolated. This was presumptively identified as *Pseudomonas pseudomallei*, a species that has not previously been reported in cystic fibrosis patients. In the follow-up examination carried out at the end of June, *Proteus mirabilis* was again prevalent and the patient's