Ochrobactrum anthropi is an opportunistic pathogen in immunocompromised patients, it is increasingly being recognized to be as a causative agent in healthy hosts. The manifestations of *O. anthropi* infections are central catheter-related infections, osteochondritis, noma, necrotizing fasciitis, endophthalmitis, meningitis, osteomyelitis, pancreatic abscess, urinary tract infection, infection of retained pacemaker leads, and endocarditis (2-5). *O. anthropi* infection-related factors include the presence of indwelling medical devices, impaired host immunity, previous antibiotic therapy, a prior surgical procedure with allografts, an accidental wound and coinfection with another bacterium. This organism is usually resistant to β-lactam antibiotics (other than carbapenems), but is susceptible to gentamicin, ciprofloxacin, trimethoprim/sulfamethoxazole and carbapenems (1,3).

A 42-year-old man was admitted to our hospital with a 2-day history of fever, chills, rigors, abdominal pain, lethargy and retention of urine. His past medical history was significant; the patient had been operated on 6 months earlier for rupture of the terminal ileum and bladder, fracture of the pelvis and pneumothorax, and he had also been treated for a few days after the patient died, all three blood cultures yielded a Gram-negative bacillus. The organism was identified as *Ochrobactrum anthropi*.

The isolate was susceptible to β-lactam antibiotics (other than carbapenems), but is susceptible to gentamicin, ciprofloxacin, trimethoprim/sulfamethoxazole and carbapenems (1,3).

The patient showed bibasilar rales, and a pansystolic murmur was auscultable on cardiac examination. There was minimal splenomegaly. The patient’s Glasgow coma score was 11. He had a bilateral petechial rash on the extremities. Abnormal laboratory findings included a white blood cell count of 21,400/mm³, with 92.4% neutrophils, a hemoglobin of 10.5 g/dl, a thrombocyte count of 52,900/mm³, a blood urea nitrogen level of 96 mg/dl, a creatinine level of 3.23 mg/dl, a C-reactive protein level of 147 mg/l, a total bilirubin level of 4.33 mg/dl, a conjugated bilirubin level of 2.19 mg/dl, a total protein level of 4.67 g/dl, an albumin level of 1.58 g/dl, a gamma-glutamyl transpeptidase level of 76 U/l, an alkaline phosphatase level of 347 U/l, and a lactate dehydrogenase level of 917 U/l. His prothrombin time was 18.5 sec, his activated partial thromboplastin time was 39.8 sec, and he had an international normalized ratio of 1.85. His pH was 7.40, pCO₂ was 23.6 mmHg, bicarbonate was 14.4 mmol/l, pO₂ was 49.5 mmHg, and O₂ saturation was 81%. On a urine slide examined under high power (× 400) magnification, we observed ≥25 leucocytes and ≥25 erythrocytes in each field. A chest radiograph showed bilateral middle zone opacities. Abdominal ultrasonography showed splenomegaly, and minimal intra-peritoneal and bilateral intrapleural fluid. The transthoracic echocardiogram demonstrated vegetation on the posterior mitral valve 2.1 × 2.5 cm in diameter, moderate mitral regurgitation and rupture of the corda tendina. Cranial computed tomography (CT) revealed a hyperdense lesion with peripheral edema, 2 × 1 cm in diameter, at the right basal ganglia adjacent to the external capsule. A urine culture and two blood cultures were drawn upon admission, and 1 h later another blood culture was drawn. Intravenous infusion of vancomycin (500 mg twice a day) and meropenem (500 mg twice a day) were started for presumed sepsis and infective endocarditis empirically (6). Two days after starting the treatment, the patient died from sepsis and intracranial and gastrointestinal hemorrhage. A few days after the patient died, all three blood cultures yielded a Gram-negative bacillus. The organism was identified as *O. anthropi* by an API 20NE (BioMérieux, Marcy l’Etoile, France) and antimicrobial susceptibility testing was performed using a disk diffusion test following the guidelines of the National Committee for Clinical Laboratory Standards (NCCLS) (7).
to gentamicin, amikacin, trimethoprim/sulfamethoxazole, imipenem and ciprofloxacin, but was resistant to β-lactams (other than carbapenems), erythromycin and chloramphenicol. Another pathogen that may be a potential cause of endocarditis was not yielded in any blood cultures. The urine culture remained negative for bacterial growth.

To the best of our knowledge, only three cases of *O. anthropi* endocarditis have been reported to date in the medical literature (2,8,9). Although *O. anthropi* is an emerging pathogen in immunocompromised patients, none of the three patients reported in the literature were immunocompromised. However, two of these patients were fitted with prosthetic valves (2,9) and one had rheumatic heart disease with mild mitral stenosis (8). In contrast to these previously reported cases, the present case had neither a prosthetic valve nor rheumatic heart disease. The multiple invasive procedures carried out during the operations that were necessary because of his workplace accident and the urethral catheter that was indwelled 20 days before admission may have been the predisposing factors for *O. anthropi* endocarditis in this case.

In the case reported by McKinley et al. (9), the bacillus was isolated from blood but not from cardiac vegetation, and was classsified as *Achromobacter* Group B, but it was not genetically identified. In the case reported by Mahmood et al. (8), the bacillus was isolated from cardiac vegetation and embolus tissue but not from blood, and was identified only on the basis of biochemical procedures. However, in the case reported by Romero Gómez et al. (2), *O. anthropi* was repeatedly isolated from the blood and cardiac vegetation of a patient with endocarditis. Also, the organism was confirmed to be *O. anthropi* by a Biolog Gram-Negative (GN) Panel (Biolog, Inc., Hayward, Calif., USA) and the 16S ribosomal DNA sequence analysis method. In our patient, the bacillus was isolated only from blood cultures, but not from cardiac vegetation, from which no specimens were taken. We suggest that the opacities in the lungs may have been due to embolus tissue but we can not confirm this hypothesis because no specimens were obtained from the opacities.

In conclusion, clinicians should be aware of the possibility of infective endocarditis and septic shock with *O. anthropi* in patients on whom multiple surgical procedures have been performed and medical devices indwelled, even in patients without prosthetic valves, or rheumatic heart disease, immuno-suppression or underlying illness.

**REFERENCES**