Septic Shock Caused by *Ochrobactrum anthropi* in an Otherwise Healthy Host

Adrien Kettaneh,1 François-Xavier Weill,2 Isabelle Poilane,3 Olivier Fain,1 Michel Thomas,1 Jean-Louis Herrmann,2 and Laurent Hocqueloux4*

Service de Médecine Interne1 and Laboratoire de Bactériologie,1 Hôpital Jean Verdier, Bondy, Laboratoire de Bactériologie, Hôpital Saint-Louis, Paris,2 and Service des Maladies Infectieuses et Tropicales, Centre Hospitalier Régional, Orléans,4 France

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Reported is a case of life-threatening septic shock that occurred in an otherwise healthy host after administration of a peripheral venous infusion of a solution contaminated with *Ochrobactrum anthropi*, an unusual human pathogen. The rapid onset of shock may have been due to a large inoculum caused by nonsterile practices at the time of reconstitution.

**CASE REPORT**

A 30-year-old woman was hospitalized through the emergency room with a history of severe weakness with fevers, chills, abdominal pain, and diarrhea that had started suddenly 5 h earlier, approximately 30 min after a home health care nurse began a peripheral venous infusion of magnesium pidolate (MP). Her past medical history included bipolar mental disorder (controlled with carbamazepine 200 mg/day) and muscular spasms treated, when symptomatic, by intravenous infusions of MP. On arrival at the emergency room, clinical examination confirmed features of severe septic shock with hypotension (blood pressure, 50/30 mmHg), tachycardia (110 beats/min), and extreme weakness. Her temperature was 38.5°C. Abdominal examination revealed mild generalized tenderness with no signs of guarding. Laboratory findings showed neutrophilia (12,200/mm³ with 98% neutrophils), anemia (hemoglobin concentration, 9.1 g/dl), thrombocytopenia (91,000/mm³), an elevated serum C-reactive protein level (56 mg/liter), a twofold elevated ALT level (159 µmol/liter), and muscular spasms treated, when symptomatic, by intravenous infusions of MP. On arrival at the emergency room, clinical examination confirmed features of severe septic shock with hypotension (blood pressure, 50/30 mmHg), tachycardia (110 beats/min), and extreme weakness. Her temperature was 38.5°C. Abdominal examination revealed mild generalized tenderness with no signs of guarding. Laboratory findings showed neutrophilia (12,200/mm³ with 98% neutrophils), anemia (hemoglobin concentration, 9.1 g/dl), thrombocytopenia (91,000/mm³), an elevated serum C-reactive protein level (56 mg/liter), a twofold elevated ALT level (159 µmol/liter; n = 60 to 110). The CD4 cell count, serum immunoglobulins, and serum complement were normal. Chest and abdominal X-rays were normal. After blood and urine specimens were obtained for bacterial examination, intravenous infusion of gentamicin (240 mg once), ofloxacin (200 mg twice a day), and amoxicillin-clavulanate (2 g three times a day) was started. Over the ensuing hours, the patient experienced intense abdominal pain with right upper quadrant tenderness. An ultrasound scan showed gallbladder wall thickening (8 mm) with a sonographic Murphy’s sign. A diagnosis of acute acalculous cholecystitis was made, and a laparoscopic cholecystectomy was performed, demonstrating a distended noninflammatory gallbladder. Histopathological examination was consistent with nonspecific subacute cholecystitis and turbid pelvic fluid collection.

Postoperatively, the patient made a dramatic recovery. On the next day, all three blood cultures incubated in aerobic and anaerobic bottles (Vital; bioMérieux, Marcy l’Etoile, France) yielded a gram-negative bacillus. Colonies were obtained on Drigalski agar medium after 24 h of aerobic incubation. Drigalski lactose agar is a selective medium used for the isolation of all enterobacteria and several nonfermenting gram-negative bacteria. Gram-positive bacteria are inhibited by crystal violet and sodium deoxycholate. The color of gram-negative colonies depends on their ability to ferment lactose. Organisms were identified as *Ochrobactrum anthropi* by use of the API 20 NE system (bioMérieux). The organism was susceptible only to imipenem, tobramycin, amikacin, gentamicin, netilmicin, trimethoprim-sulfamethoxazole, and pefloxacin, a typical susceptibility pattern for *O. anthropi* (13, 16). Cultures of urine and peritoneal fluid remained sterile. Subsequently, amoxicillin-clavulanate was discontinued. Transthoracic echocardiography excluded a valvular abnormality. The patient made a complete recovery and was discharged from the hospital on day 9. Ofloxacin administration was stopped on day 11.

Because of this unusual presentation, we interviewed the nurse who had initiated the infusion. She revealed to us that, in order to save time, the patient had reconstituted the solution herself by adding six MP vials to 100 ml of a 5% glucose solution. Unfortunately, her last infusion had been delayed and the solution had been left to stand at room temperature. The solution was then not administered for 24 h after its reconstitution. We obtained the empty 10-ml vials of MP that had been used for the infused preparation. Cultures of washings from the vials yielded *O. anthropi* with the same antibiotic susceptibility profile as the strain isolated from the patient’s blood. The strains of *O. anthropi* isolated both from the patient and from the vials were analyzed and compared to four reference strains from the collection of the Pasteur Institute (Paris, France) by pulsed-field gel electrophoresis (PFGE). Genomic DNA was digested with the restriction endonuclease SpeI, and PFGE was done by using the PSU program of the GenePath System (Bio-Rad, Iivy sur Seine, France). Analysis of the restriction pattern showed that the strains isolated from the patient’s...
O. anthropi, formerly known as Centers for Disease Control and Prevention group Vd or Achromobacter Vd, was definitively named in 1988 (10). It is a nonfastidious, gram-negative, motile, nonfermenting bacillus with strict oxidative metabolism that possesses a very active urease. O. anthropi is widely distributed in the environment, in soil, plants, water sources including normal saline and antiseptic solutions, dialysis liquids, swimming pools, etc. (2, 6, 7, 10, 14). It may thus share the same microbial niche as Pseudomonas species (6). Like the latter, O. anthropi is characterized by a broad spectrum of antibiotic resistance. However, O. anthropi has rarely been reported as a human pathogen. It is thought to have low pathogenic potential, with most of the published cases being nosocomially acquired infections in debilitated hosts with indwelling medical devices (6–9, 12, 14, 17).

In a Medline search of the English literature from 1988 through 2001 with the keywords Ochrobactrum anthropi, we identified 57 citations. These confirm that the pathogenic potential of O. anthropi is strongly associated with the presence of indwelling medical devices, such as central venous catheters (6, 7, 9), drainage tubes, and intraperitoneal catheters (14). This is likely to be due to its ability to adhere to various synthetic materials (1). Other factors have also been involved, such as impaired host immunity (8), previous antibiotic therapy (17), a prior surgical procedure with allografts (5), an accidental wound (2), and coinfection with another bacterium (17).

Additionally, nearly all of the reported cases were indwelling-material-related infections. Almost half were sporadic nosocomially acquired bacteremias complicating central venous catheters in immunocompromised hosts or patients with severe underlying diseases (in particular, those with hematological malignancies or solid tumors) (17). Another quarter of the reports described pyogenic infections complicating indwelling medical devices or surgical procedures, such as intraocular lenses (3) or catheters for peritoneal dialysis (14). Rarely, O. anthropi has been described as a community-acquired pathogen (2, 4) occurring in patients with no previous history of an indwelling medical device, as in a report by Yu et al. (17). Nevertheless, all of these patients had debilitating underlying diseases.

It is noteworthy that, despite a high level of resistance to antibiotics and considering that the majority of cases have been described in debilitated hosts, O. anthropi is thought to have low intrinsic pathogenic power and virulence (17).

Our observations differ radically from previously reported cases. Our patient was an otherwise healthy host with no debilitating medical disease. By contrast, she presented with life-threatening septic shock, a situation not previously reported. In this setting, we believe that her cholecystitis was neither the source nor a septic metastatic manifestation of the infection. The pathologic findings suggested that it was more likely to be a complication of the shock itself (11). Furthermore, the only indwelling catheter our patient had was a transient peripheral venous cannula. We believe that the infusion itself, and not the catheter, was contaminated with O. anthropi. This hypothesis is supported by the fact that PFGE results revealed the presence of a strain identical to the patient’s O. anthropi strain in the vials used to prepare the solution of MP. PFGE has been shown to be the most discriminatory method for epidemiological analysis of O. anthropi (7, 15).

Ezzedine et al. (8) reported five cases of O. anthropi bacteremia in allograft recipients receiving rabbit anti-thymocyte globulin vials from a contaminated lot. Ineffectiveness of the industrial sterilization process was suspected. Unlike our case, these patients were severely immunocompromised, receiving treatment with a combination of cyclosporine A, azathioprine, and steroids. We believe that in our case, the solution was contaminated by the patient herself at the time of reconstitution, as a consequence of nonsterile practices. We cannot exclude the possibility of contamination of the MP vials at the source. However, a malfunction in the vial-manufacturing process is unlikely because the manufacturer was not informed of any similar cases that occurred after infusion of the same lot of vials. We hypothesize that a large inoculum was responsible for the rapid onset of septic shock in our patient. The delayed time between the reconstitution and infusion of the contaminated solution, with storage at ambient temperature for 24 h, may have allowed massive growth of this environmental bacterium.

Physicians must be aware that a normal host may be infected by O. anthropi under some exceptional circumstances. Intravenous infusion of a heavily contaminated solution may cause life-threatening septic shock in this setting.

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REFERENCES


