CASE REPORTS

Roseomonas gilardii Infection: Case Report and Review

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Roseomonas gilardii is a bacterium that has been indicated as a rare cause of human infections. The case of a patient presenting with cellulitis and bacteremia secondary to *R. gilardii* is described together with the clinical characteristics of infection with this organism obtained from a review of cases previously reported.

CASE REPORT

A 42-year-old Hispanic female presented to her physician with a 2-day history of chills, fever to 103°F, and diaphoresis associated with swelling and redness of the left foot and pain when walking. She did not remember being bitten, but she did remember finding a spider on her pillow upon awakening. Past medical history was significant for hypertension, hepatitis C, and hypothyroidism. Her medication consisted of benazepril, estrogen, and levothyroxine sodium. Examination at that time revealed that her temperature was 99.6°F, and the dorsum of her foot was swollen to her toes, with an area of purplish discoloration 2 inches in diameter, with two dark spots. This was diagnosed as a spider bite, and she was prescribed an oral antibiotic, dapsone (50 mg by mouth twice a day), and was sent home with the recommendation to apply ice to the affected area and elevate the foot.

The patient was reexamined the next day; she was still having chills and reported a fever of 104°F with an increase in pain and swelling of her left foot. The examination in clinic revealed her to be afebrile with palpable pulses in the lower extremities and with a nontender calf. Her foot, however, had more-extensive erythema and increased swelling. She was therefore admitted to the hospital with a diagnosis of cellulitis secondary to a spider bite.

On admission, her temperature was 99°F, and it rose to a maximum of 100.3°F during the hospitalization. At the time of admission, her white blood cell count was 8.6×10^9 /liter, with 71% neutrophils, and it remained normal. Her liver function tests revealed a mildly elevated bilirubin and alkaline phosphatase but were otherwise normal. Two separate blood cultures were drawn on the day of admission, when her temperature was 100.3°F.

She was initially placed on intravenous nafcillin, oral dapsone, and topical silver sulfadiazine ointment. During the next 2 days, however, her foot appeared clinically worse, with increasing erythema and swelling. The silver sulfadiazine was stopped, and levofloxacin was added empirically. On the third day of hospitalization, two new physical findings were noted: first, the foot had developed blistering on the lateral aspect and continued to look worse; second, a soft systolic murmur was now evident. At this point, an echocardiogram was ordered to exclude valvular vegetations and one of the blisters was aspirated and sent for culture. In the meantime, one of the two blood culture sets obtained at admission showed growth of a gram-negative bacillus from both anaerobic and aerobic bottles. Based upon these results, we replaced levofloxacin with cefepime to widen gram-negative bacterium coverage and specifically to ensure adequate coverage against Pseudomonas aeruginosa. Over the ensuing few days, her foot began to improve, becoming less edematous and erythematous, and this continued. The hospital laboratory was now reporting the organism to be gram variable and was having difficulty identifying the organism, so on day 5 it was sent on to the Texas State Department of Health laboratory for final identification. Since the patient seemed to have improved after the institution of cefepime therapy, at this point the nafcillin and dapsone were stopped and cefepime was continued. By day 6, the culture by aspiration of the blisters returned negative, as did the echocardiogram performed to exclude valvular vegetations.

In the meantime, the patient continued her improvement and was discharged home on day 7, after central line placement, and she continued on home intravenous cefepime (2 g every 12 h for a total of 14 days). The patient went on to make a complete recovery. The State Laboratory identified the organism as *Roseomonas gilardii*, using the Elizabeth King Protocol from the Centers for Disease Control and Prevention to make its final determination. Antibiotic susceptibility testing was not performed by either laboratory.

Our case demonstrates some features in common with case

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R. gilardii is a pink gram-negative coccobacillus belonging to the genus *Roseomonas*. These bacteria have been isolated from clinical specimens for the last 30 years, but prior to the current classification, the group had been referred to as Pink Coccoid groups 1 to 4 (1, 2, 5, 9). In 1993, Rihs divided the genus into six *Roseomonas* species based on biochemical and DNA hybridization techniques (6). Of these six, *R. gilardii* is most frequently related to human infections.

reports and case series of patients found to have infections with R. gilardii. There are two case reports and two case series, making a total of 25 cases that have sufficient clinical information to document infection by R. gilardii (3, 7, 8; L. Alcala, F. J. Vasallo, E. Cercenado, F. Garrote, M. Rodrigo-Creixems, and E. Bouza, Letter, J. Clin. Microbiol. 35:2712, 1997). Lewis et al. reported the clinical characteristics of seven patients, from whose blood cultures pink coccoid organisms were isolated, retrospectively analyzed, and found to be R. gilardii (3). Struthers has also reported clinical information obtained from a retrospective analysis of previously described pink pigmented coccobacilli that were the suspected cause of infection (8). Unfortunately, they were able to obtain clinical information in only some of these cases and often it was incomplete. However, some common themes with regard to human R. gilardii infection emerge when the information from these cases is pooled together.

The natural reservoir for this infection is not known. Although it has been isolated from water supplies (6, 7), it may also exist as a commensal in humans. This view is supported by the fact that, of those cases with sufficient information reported by Struthers, the bacterium was felt not to be a primary or secondary pathogen in 15% of them (8).

In a number of the cases of *R. gilardii* infections reported in the literature (14 of 25 [56%]), the initial symptoms were indicative of infection and bacteremia. It seems also that this outcome is often associated with the presence of a central line (9 of 25 cases reporting clinical information [3, 7, 8; Alcala et al., letter]). Other sites of infection have also been reported; respiratory problems occurred in 16% of cases, wound infection or bone disease occurred in 8%, peritonitis in 4%, enteritis and/or abdominal pain in 8%, kidney transplant infection in 4%, and eye infection in 4% (3, 6–8; Alcala et al., letter).

A common finding with this infection is that it seems to be a feature of patients with underlying debility: 80% of patients reported had underlying disease, most commonly malignancy, followed by renal disease, inflammatory bowel disease, diabetes, and others (alcohol abuse, osteoarthritis, cystic fibrosis, and circulatory insufficiency) (3, 7, 8; Alcala et al., letter).

A consistent feature of this organism is its slow-growth properties on culture; it often takes 4 to 5 days before any growth is seen. Further analysis reveals that in 88% of these cases, the organism was isolated from the blood (3, 7, 8; Alcala et al., letter). Often it was isolated from only one of several blood cultures or from a central venous catheter line and not the peripheral blood sample. Other sites of isolation were more rare: wounds, respiratory tract, and peritoneum (4% each).

Another common characteristic of this gram-negative organism is its antibiotic susceptibility pattern, especially with regard to its behavior with cephalosporins, which appear ineffective against any of the *Roseomonas* species. Rihs found that all six species exhibited >96% resistance to cephalosporins including expanded-spectrum generation cephalosporins: cefotaxime, ceftriaxone, and ceftazidime (6). Alcala et al. (letter) and Nahass et al. (4) confirmed this resistance in their isolates. In contrast, Lewis reports that all of her isolates were susceptible to cefotaxime (3), with at least two of her patients making a complete recovery on cefotaxime. There are no data on susceptibility to "fourth-generation" cephalosporins, such as cefepime and cefpirome. The species has universal susceptibility to imipenem, amikacin, gentamicin, tobramycin, and tetracycline (3, 4, 6, 7). It is largely resistant to penicillins, including the extended-spectrum penicillins such as piperacillin and mezlocillin. Addition of a beta-lactam can increase susceptibility (3, 4, 7); Rihs et al. reported only 13% resistance to ticarcillin-clavulanate (6).

We do not have data on susceptibility to newer quinolones, but 65% of the isolates studied by Rihs et al. were susceptible to ciprofloxacin and all other isolates that were tested were susceptible to this antibiotic (3, 4, 7; Alcala et al., letter).

Despite the fact that this infection seems to occur in debilitated patients, mortality from it seems to be relatively low and patients do usually recover completely. There are two deaths reported in the literature. In both of these cases the death was felt to be due to underlying problems including human immunodeficiency virus (3) and chronic lung disease (4). However, the *Roseomonas* species was not identified for the latter patient.

We believe that R. gilardii was the causative organism in the case described here for several reasons. First, the patient's systemic symptoms were at their height on the day that the positive blood culture set was drawn and it grew from both aerobic and anaerobic bottles. Although only one blood culture set became positive, this is consistent with other cases reported in the literature, all of which report that only one of several blood cultures grow the organism (3, 7, 8; Alcala et al., letter). Bacterial growth also took 3 days, which is consistent with the slow-growth properties reported elsewhere. The clinical presentation that we observed is also almost identical to that of other cases: namely, the presence of underlying medical problems and presentation with systemic symptoms of infection with bacteremia. Unlike other patients, though, our patient did not have a central line until after the diagnosis was made. Although the source of infection in our case was felt to be the skin, we were not able to isolate the infecting organism from there. However, this is also consistent with previous findings, in that only one case has been reported in which this organism has been successfully cultured from the skin (8). As in the majority of other cases, our patient made a complete recovery with treatment. Unfortunately, we did not receive antibiotic susceptibility data to guide our therapy. We chose cefepime because the organism had originally been reported as gram negative and we wished to ensure adequate coverage for P. aeruginosa. Clinically, the patient's foot was worsening until this point, and it was only after the institution of cefepime treatment that it started to improve. As already discussed, data on susceptibility of R. gilardii to fourth-generation cephalosporins are not available. However, we are convinced that our organism was sensitive to this antibiotic because of the clinical improvement; and although most isolates have been resistant to expanded-spectrum cephalosporins, our findings are consistent with those of Lewis et al. (3) who did observe a susceptibility to cefotaxime.

Physicians should familiarize themselves with the characteristics of infection with R. gilardii because of diagnostic and management implications. This organism should be considered in the differential diagnosis when a patient presents with underlying debility and sepsis in the presence of a central line, especially if there is no other focus of infection and no growth on blood culture for 3 to 4 days. In addition, contamination should not be immediately assumed if only one blood culture is positive. It is important to pursue final identification of the organism because of its unique susceptibilities to antibiotics. Consideration of gram-negative coverage with an aminoglycoside rather than a cephalosporin would be reasonable.

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