## A Case of Peritonitis Caused by *Roseomonas gilardii* in a Patient Undergoing Continuous Ambulatory Peritoneal Dialysis

J. A. T. SANDOE,<sup>1\*</sup> H. MALNICK,<sup>2</sup> AND K. W. LOUDON<sup>1</sup>

Department of Medical Microbiology, Clinical Sciences Building, Manchester Royal Infirmary, Manchester M13 9WL,<sup>1</sup> and Laboratory of Hospital Infection, PHLS Central Public Health Laboratory, London NW9 5HT,<sup>2</sup> United Kingdom

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## A case of peritonitis caused by *Roseomonas gilardii* in a patient receiving continuous ambulatory peritoneal dialysis is presented. The patient's domestic water supply was implicated as the probable source of infection. This is the first report of *R. gilardii* causing such an infection.

Continuous ambulatory peritoneal dialysis (CAPD) is an established treatment for end-stage renal failure. Peritonitis is the main complication of CAPD and is most commonly caused by skin commensal organisms (3, 5, 14). Environmental organisms are a rare cause of peritonitis in these patients, and this is the first time to our knowledge that the pink-pigmented organism *Roseomonas gilardii* has been implicated.

A 62-year-old retired widow was placed on CAPD for chronic renal failure of unknown etiology in August 1995. She dialyzed at home by using a two bag exchange system four times a day. In July 1996 she presented with vague abdominal pain and cloudy peritoneal dialysate but was apyrexial and not systemically unwell. A clinical diagnosis of peritonitis was made, and samples of dialysate fluid were taken for culture and microscopy. Treatment with 2 g of intraperitoneal vancomycin and 50 mg of intraperitoneal netilmicin was commenced. Netilmicin at a dosage of 50 mg once daily was continued on an outpatient basis for 2 weeks, and the patient made a full recovery. The patient had noticed a persistent clouding of her domestic water supply around the time of the peritonitis episode. The tap water cleared following repair of a local water main leak. She managed her exchanges well and had only experienced one previous episode of peritonitis caused by Escherichia coli. No further episodes of peritonitis have occurred.

Samples of dialysis effluent were obtained by specialist CAPD nurses using an aseptic technique and were inoculated into a sterile 50-ml plastic container, a Hémoline performance diphasic culture bottle (BioMerieux, Marcy-L'Etoile, France), and a 150-ml sterile plastic container. A leukocyte count, differential, and Gram's stain were performed on the dialysate from the 50-ml container. Dialysate in the 150-ml container was enriched with 20 ml of quadruple-strength brain heart infusion broth (Oxoid, Basingstoke, United Kingdom), then incubated at 37°C and examined daily for turbidity. The diphasic culture bottle was incubated at 37°C and subcultured daily by inversion. Antimicrobial sensitivity testing of the isolate was carried out by a disk diffusion method and E test (AB Biodisk, Solna, Sweden). Swabs (Technical Service Consultants Ltd., Heywood, United Kingdom) were taken from the hot taps, cold taps, the shower head, and the sink overflow in the pa-

\* Corresponding author. Mailing address: Department of Medical Microbiology, Clinical Sciences Building, Manchester Royal Infirmary, Oxford Road, Manchester M13 9WL, United Kingdom. Phone: 0161 276 6333. Fax: 0161 276 8826. E-mail: jsandoe@labmed.cmht.nwest .nhs.uk.

tient's bathroom and cultured on 7% horse blood agar (Oxoid) at 37°C for 5 days. Samples of water from the patient's hotwater and cold-water supplies were collected in sterile plastic 250-ml containers and filtered through 0.1-µm-pore-size membranes (Millipore, Bedford, Mass.), and the filters were cultured as described above.

Microscopic examination of the CAPD fluid revealed a leukocyte count of  $210 \times 10^6$ /liter, with 95% polymorphonuclear leukocytes. No organisms were seen on a Gram's stain of centrifuged sediment. After 48 h of incubation the diphasic bottle and the 200-ml bottle grew a short, nonvacuolated, gram-negative rod. Colonies were 0.5 to 1.0 mm in diameter, pale pink, shiny, raised, entire, and mucoid after 2 days of incubation on 7% horse blood agar, chocolate agar, and Mac-Conkey agar (Oxoid) under aerobic conditions at 37°C. There was no growth anaerobically. The organism was oxidase negative, nonmotile, and catalase and urease positive. It was susceptible to gentamicin, ciprofloxacin, and erythromycin but resistant to penicillin and cephalosporins (Table 1). The isolate was identified as R. gilardii by using the tests described by Holmes et al. (6) and by the lack of absorption of long-wave UV light (11) (Table 2). The environmental swabs and water samples did not grow R. gilardii.

The type strain of *R. gilardii* (ATCC 49956) was isolated from potable water around 1980 but was not named *R. gilardii* until 1993 by Rihs et al. (11). It is considered to be pathogenic for humans. In 1984 Gilardi and Faur (4) designated seven strains of pink gram-negative rods, isolated from blood, cerebrospinal fluid, and sputum, "an unnamed taxon" distinguished from the pink organism *Methylobacterium mesophili* 

 TABLE 1. MICs for R. gilardii 96/SID/1295 isolated from peritoneal dialysate

Antibiotic	MIC (µg/ml)
Penicillin	. >32
Ampicillin	.>256
Piperacillin/Tazobactam	.>256
Augmentin	. 32.00
Imipenem	. 1.00
Cefuroxime	.>256
Ceftazidime	.>256
Gentamicin	. 0.50
Netilmicin	. 0.38
Ciprofloxacin	. 0.75
Erythromycin	. 1.0

 TABLE 2. Properties of R. gilardii 96/SID/1295 from peritoneal dialysate and 40 R. gilardii strains identified by the Central Public Health Laboratory, Colindale, London, United Kingdom

Characteristic or test	<i>R. gilardii</i> 96/SID/1295	<i>R. gilardii</i> archive strains <sup>a</sup>
Acid from:		
Adonitol	—	n/a
Arabinose	+	V
Cellobiose	—	n/a
Dulcitol	-	_
Ethanol	+	n/a
Fructose	+	+
Glucose	+	V
Glycerol	+	+
Inositol	-	n/a
Lactose	-	-
Maltose	—	_
Mannitol	+	V
Raffinose	—	_
Rhamnose	—	_
Salicin	—	_
Sucrose	—	_
Starch	-	n/a
Trehalose	—	n/a
Xylose	+	V
Absorption of UV light	_	_
Arginine dihydrolyse	-	-
Lysine decarboxylase	-	-
Ornithine decarboxylase	-	-
Citrate	+	+
DNase	_	—
Growth on cetrimide	_	—
Gelatin liquefaction	_	—
Hydrogen sulfide production	-	-
Hydrolysis of:		
Tween 20	_	V
Tween 80	_	-
Esculin	—	—
Indole production	_	_
Nitrate reduction	-	-
ONPG <sup>b</sup>	-	—
PHBA <sup>c</sup> growth	+	+
PHBA inclusions	+	+
Tyrosine growth	+	+
Tyrosine hydrolysis	+	V
Tyrosine pigment	_	-
Urease	+	+
Vacuolated cells	-	—

 $^{a}$  +, ≥95% of strains positive; -, ≤5% of strains positive; n/a, not available; v, variable.

<sup>b</sup> ONPG, o-nitrophenyl-β-D-galactopyranoside.

<sup>c</sup> PHBA, polyhydroxybutyrate agar.

*cum* (formerly named *Pseudomonas mesophilica*) by their Gram-stain morphology, inability to produce acid from methanol, growth on MacConkey agar, growth at 42°C, and acetate utilization. In 1990 Wallace et al. (15) examined 156 clinical isolates of pink gram-negative rods which they divided into "pink coccoid groups" I through IV on the basis of the oxidation of D-xylose and D-mannitol and the hydrolysis of esculin. Subsequently, Rihs et al. (11) studied strains of the Centers for Disease Control and Prevention pink coccoid groups I through IV, Gilardi's unnamed taxon, and various other clinical isolates, identifying six DNA-relatedness groups. *R. gilardii* corresponded to "genomospecies" 1; none of the genomospecies 1 strains had been isolated from peritoneal fluid.

The genus *Roseomonas* is characterized by pink-pigmented, gram-negative, nonfermentative, plump coccoid rods or cocci, appearing in pairs or short chains. They grow on 5% sheep blood agar, chocolate agar, buffered charcoal-yeast extract agar, and almost always (91%) on MacConkey agar but do not grow on media containing greater than 6% NaCl. *Roseomonas* spp. grow at 25, 30, 35, and usually 42°C. Motility, nitrate reduction, and oxidation of L-arabinose, D-galactose, D-glucose, D-mannose, D-mannitol, and D-xylose are variable characteristics (11).

Most cases of CAPD peritonitis are caused by skin commensals. Pathogens may contaminate the peritoneum from exit site and tunnel infections, from transient bacteremia, and through contamination of the dialysate delivery system during bag changes (13). Environmental organisms are rarely implicated in CAPD peritonitis; however, there are reports of waterassociated organisms causing peritonitis in these patients. Rutherford et al. (12) reported a case of peritonitis caused by P. mesophilica (M. mesophilicum) originating from the patient's water supply and bathroom hand rail. Pseudomonas aeruginosa, Pseudomonas paucimobilis, and Acinetobacter spp. have all caused peritonitis, and in each case a water bath used to preheat dialysis bags was incriminated as the source (1, 7, 9). Episodes of peritonitis caused by Mycobacterium chelonei have also been attributed to tap water entering the peritoneal cavity (2, 10).

The source of infection in this case was not found, but the domestic water supply would be a likely source of the organism. The patient's water supply cleared after water main repairs, which may explain why the organism was not found by environmental screening. The risk of peritonitis caused by *Pseudomonas* spp. and other water-borne gram-negative rods can be decreased by minimizing the patient's contact with household water (8).

This case emphasizes the potential for organisms in domestic water supplies to cause opportunistic infection in patients undergoing CAPD and confirms *R. gilardii* as a potential pathogen in these patients.

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