Comamonas testosteroni-associated peritonitis in a pediatric peritoneal dialysis patient

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Abstract

Comamonas testosteroni (C. testosteroni) has been rarely observed as an infectious agent in clinical practice. Few reports described its potential pathogenicity in bloodstream and abdominal infections. Here, we report our experience in the treatment of a C. testosteroni-associated peritonitis in a four-year-old girl receiving chronic peritoneal dialysis (PD). The organism was shown to be highly susceptible to appropriate antibiotic therapy. Infection responded promptly and the patient was managed conservatively without withdrawal from PD.

Key words: Comamonas; Peritonitis; Peritoneal dialysis; Comorbidity; Children

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INTRODUCTION

Comamonas testosteroni (C. testosteroni) is an aerobic Gram-negative organism with a widespread
environmental distribution. Infection by *C. testosteroni* is infrequent, however there are some reports describing its potential causative role in bacteremia, meningitis, urinary tract infections, endocarditis, cellulitis, and pneumonia.[1,2] Isolation of *C. testosteroni* has also emerged in localized peritonitis as a complication of perforated appendicitis[3,4].

Here, we present our experience in the treatment of a *C. testosteroni*-associated peritonitis in a four-year-old girl receiving chronic peritoneal dialysis (PD).

## CASE REPORT

The girl was previously diagnosed with end-stage renal disease due to atypical haemolytic-uraemic syndrome, and she had been treated with automated PD for 10 mo. The girl was also affected by severe motor-cognitive impairment and idiopathic epilepsy.

She was admitted to our Department with a 24-h history of high-grade fever and complaining of abdominal pain. Physical examination revealed abdominal tenderness, along with cloudy peritoneal effluent. The patient’s white blood cell count was normal (6130/mm$^3$), whereas the C-reactive protein was significantly increased (290 mg/L). The leukocyte in peritoneal effluent showed a count of 6600/mm$^3$ (90% polymorphonucleated). One month before this event, the patient had experienced a *S. aureus* peritonitis, for which she had completed a 3-wk course of intraperitoneal therapy with glycopeptide.

After admission, empiric antibiotic therapy was started with both intravenous ceftazidime and teicoplanin. The fever subsided within 48 h and the leukocyte count in effluent resulted normal (< 100/mm$^3$) within 72 h from the start of antibiotic treatment. Signs and symptoms of peritonitis regressed within 48 h. On hospital day 3, cultures from peritoneal effluent resulted positive for *C. testosteroni*. Antibiotic treatment was then simplified, with single-agent intraperitoneal ciprofloxacin in order to complete a 3-wk course of therapy. The patient was discharged and a follow-up after 14 and 30 d showed persistence of antibiotics, including aminoglycosides, cephalosporins, fluoroquinolones, carbapenems, piperacillin-tazobactam, cephalosporins, and trimethoprim-sulfamethoxazole[5]. According to the survey by Farshad et al,[1] 32 out of 35 reported cases of human infection by this bacterium were less than 5% of episodes[6]. The vast majority of patients are treated successfully with antibiotics administered intraperitoneally and continue PD. The poorest outcomes are observed in patients with Gram-negative organisms or fungi peritonitis and in those with a relapsing infection. In this case, early PD catheter removal with transient switch to haemodialysis is sometimes required[7]. Despite the lower prevalence, fungal infections are associated with the highest mortality rate.

*C. testosteroni* is a gram-negative aerobic bacillus that is found in various environments, including soil, water, plants, and animals. In spite of its wide environmental distribution, there are few reports on its involvement in human infections.[8] Most of the reported infections by this organism are community-acquired, however some authors suggested that it can also survive for a long time in the hospital setting. Indeed, it can colonize several devices, such as intravenous lines, respiratory equipment, and humidifiers[9,10]. This seems mostly due to an extraordinary capability of this organism in both environmental adaptation and biofilm formation[10]. Nevertheless, few molecular biological investigations were taken on the pathogenicity and virulence of *C. testosteroni*. Very recently, Liu et al[11] conducted a comprehensive genomic analysis among 10 *C. testosteroni* strains. They identified 24 types of virulence factors that were involved in several functions such as adherence, anti-phagocytosis, invasion, and secretion system. Moreover, the authors found that most of the virulence factors were owned by all of the strains and were highly conserved. These results supported the molecular biological basis of the potential pathogenicity of this bacterium.

Along with its own virulence factors, pathogenicity of *C. testosteroni* seems to be emphasized in patients with some degree of immunosuppression such as malignancy, prematurity, primary or secondary immunodeficiency induced by chronic liver disease and end-stage renal disease.[11,12] Moreover, bacterial translocation from the gastrointestinal tract seems to play an important role in the pathogenesis of infections[13].

Very recently, Altun et al[14] published the first continuous ambulatory PD patient treated for a *C. testosteroni*-associated peritonitis. The authors described a 29-year-old woman with end-stage renal failure secondary to hypertensive nephrosclerosis who had been treated with CAPD for 10 mo. In this case, along with the chronic dialysis status, the predisposing factor for peritonitis with this pathogen was probably a previous laparoscopic intervention because of incidental dislocation of an intrauterine device to the space between the peritoneum and the anterior abdominal wall. Signs and symptoms of peritonitis regressed rapidly during a 14-d period of oral ciprofloxacin.

*C. testosteroni* is usually sensitive to a broad range of antibiotics, including aminoglycosides, cephalosporins, fluoroquinolones, carbapenems, piperacillin-tazobactam, cephalosporins, and trimethoprim-sulfamethoxazole[15]. The patient was discharged on oral ciprofloxacin, with fungi responsible for
promptly responsive to antibiotic treatment. Outcome was fatal in three cases, including a 64-year-old woman on hemodialysis with a central venous catheter-related bacteremia.[12]

To our knowledge, the present case is the second report of a *C. testosteroni*-associated peritonitis in a PD patient, but is the first description in pediatric age. Information regarding immune function in children with chronic kidney disease or receiving dialysis are sparse. The incidence of infectious episodes in children on dialysis is higher than that found in adults; moreover, immaturity of the immune system may also contribute to its dysfunction especially in children with chronic diseases and several co-morbidities. In our patient, both dialysis status and severe motor-cognitive impairment may have increased the pathogenicity of *C. testosteroni*, similarly to previous adult case reports. Moreover, the previous and recent episode of *S. aureus* peritonitis might have represented a further predisposing factor to *C. testosteroni* infection. In fact, in PD patients, bacterial peritonitis induces a subsequent breakdown of intestinal barrier function and a transient impairment of host mucosal immune defense.[16] This may have allowed further enteric low-virulence organisms to enter the peritoneal cavity by transmural migration and to cause peritonitis.[17]

*C. testosteroni* should be kept in mind as a rare cause of bacterial peritonitis in children receiving chronic PD. With the improvement in care of end-stage renal disease patients and given the potential for favorable outcomes, a higher number of children with severe co-morbidities is now started to PD. Clinical management of these children is demanding; in a report of the International Pediatric Peritoneal Dialysis Network, Neu et al.[18] showed that children on PD with comorbidity had a higher hospitalization rate than did patients without a comorbidity. Infections from both common and unusual pathogens were the most frequent reasons for hospitalization and mortality. Our experience confirms that *C. testosteroni* peritonitis responds promptly to adequate antibiotic therapy. A conservative management can be adopted without loss of the PD catheter and withdrawal from the PD.

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**Treatment**

Intraperitoneal ciprofloxacin in order to complete a 3-wk course of therapy.

**Related reports**

Infection by *C. testosteroni* is infrequent, however there are some reports describing its potential causative role in bacteraemia, meningitis, urinary tract infections, endocarditis, cellulitis, and pneumonia. Isolation of *C. testosteroni* has also emerged in localized peritonitis as a complication of perforated appendicitis.

**Term explanation**

*C. testosteroni*-associated peritonitis represents a rare complication of chronic PD. The pathogenicity of this bacteria might be increased in immunodeficient patients, in children with severe chronic diseases, and affected by co-morbidities.

**Experience and lessons**

*C. testosteroni* should be kept in mind as a rare cause of bacterial peritonitis in children receiving chronic PD. The pathogenicity of this bacteria might be increased in immunodeficient patients, in children with severe chronic diseases, and affected by co-morbidities.

**Peer-review**

*C. testosteroni*-associated peritonitis in PD patients is a rare condition. This case-report can add some new information to clinical practitioners.
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