First report of *Comamonas kerstersii* causing urinary tract infection

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Abstract

The association of *Comamonas kerstersii* with peritonitis resulting from perforated appendix and its isolation from a psoas abscess and pelvic peritonitis have previously been described by us. We present the first case of *C. kerstersii* urinary tract infection, broadening the spectrum of infections caused by this species.

Keywords: *Comamonas kerstersii*, emerging pathogen, psoas abscess, secondary peritonitis, urinary tract infection
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Comamonas spp. are nonfermenting Gram-negative bacilli of the *Comamonadaceae* family. The genus includes 22 species. Mainly *Comamonas testosteroni and Delftia* (*Comamonas*) *acidovorans* have been implicated in human infections [1]. However, an increase in the number of reports of human infections by another species, *Comamonas kerstersii,* described in 2003 by Wauters et al. [2], has recently been observed [3,4]. In this sense, the use of new and revolutionary methodologies for bacterial identification in routine laboratory practice, such as matrix-assisted desorption ionization–time of flight mass spectrometry (MALDI-TOF MS) [5,6], could explain the increase in the frequency of isolation of this species reported in the literature in recent years [3,4,7,8].

A previously healthy 5-year-old girl with no history of urinary tract infection but with abdominal and low back pain, fever and vomiting was hospitalized to rule out an acute surgical abdomen. The physical examination and the abdominal ultrasound revealed nothing abnormal, but the patient had persistent fever, abdominal pain and positive fist percussion. Therefore, pyelonephritis was diagnosed. A peripheral venous blood sample collected at admission showed white blood cell count 24 900/mm^3 (with 83% neutrophils), haematocrit 38%, haemoglobin count 12.4 g/dL, platelet count 286 000/mm^3, erythrocyte sedimentation rate 86 mm, blood urea nitrogen level 21 mg/dL and creatinine level 0.5 mg/dL. In addition, a kidney ultrasound revealed both kidneys had preserved shape, size and structure, although an 8.1 mm left pyelocalyceal dilatation was observed.

Microscopic analysis of urine revealed 20 leukocytes per high-power field, four erythrocytes per high-power field and two epithelial cells per high-power field. After 24 hours of incubation at 35°C, a pure growth of a nonfermenting Gram-negative bacillus of more than 10^5 CFU/mL on cystine lactose electrolyte deficient agar, incubated at ambient air temperature, was observed. The patient was treated empirically with ceftriaxone 50 mg/kg per day for 3 days.

The microorganism was identified as *Comamonas kerstersii* by MALDI-TOF MS using a Microflex LT instrument with Biotyper 3.1 software (Bruker Daltonics, Bremen, Germany), with a spectral score of 2.134. Phenotypic identification was confirmed by molecular identification (amplification and subsequent sequencing of the *gyrB* gene) using the primers described by Tayeb et al. [9]. *gyrB* gene sequence analysis revealed 99% identity with the *gyrB* sequence of *Comamonas kerstersii* (GenBank accession no. KC714047).

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TABLE 1. Clinical and microbiologic characteristics of patients with *Comamonas kerstersii* infections

Case No.	Age (years)/Sex	Site of infection	Clinical presentation	Underlying disease	Predisposing conditions	Identified pathogens	Antibiotic treatment	Reference
1	43, F	Peritoneal fluid	Febrile syndrome, abdominal pain	Ovarian tumour with peritoneal metastases	Sigmoid perforation by foreign body (biliary stent), rectovaginal fistula and colostomy	*Escherichia coli, Bacteroides fragilis, Comamonas kerstersii*	Ampicillin/sulbactam followed by piperacillin/tazobactam and then ertapenem	[7]
2	48, M	Peritoneal fluid	Febrile syndrome, abdominal pain for 3 days	No underlying disease	Perforated appendix	*Streptococcus anginosus group, Aeromonas hydrophila group, Escherichia coli, Comamonas kerstersii*	Ampicillin/sulbactam, ciprofloxacin and then amoxicillin/clavulanic acid	[7]
3	10, F	Peritoneal fluid	Abdominal pain for 3 days, bilious vomiting and febrile events	No underlying disease	Perforated gangrenous appendix	*Streptococcus anginosus group, Escherichia coli, Comamonas kerstersii*	Ampicillin/metronidazole/gentamicin and then amoxicillin/clavulanic acid	[7]
4	21, F	Peritoneal fluid	Abdominal pain for 3days associated with vomiting	No underlying disease	Perforated gangrenous appendix	*Citrobacter amalonaticus, Comamonas kerstersii*	Ampicillin/metronidazole/gentamicin	[7]
5	65, M	Blood	Fever, chills, vomiting, diarrhea	Diabetic	None	*Comamonas kerstersii, Bacteroides fragilis*	Ciprofloxacin, imipenem	[4]
6	12, M	Peritoneal fluid	Abdominal pain	None	Appendicitis	*Comamonas kerstersii, Escherichia coli, Streptococcus sp. (group anginosus/ milleri)*	Coamoxicillin, metronidazole and amikacin followed by coamoxicillin alone	[4]
7	10, M	Peritoneal fluid	Abdominal pain	None	Perforated appendicitis	*Comamonas kerstersii Streptococcus constellatus*	Piperacillin/tazobactam and then amoxicillin clavulanic acid and ciprofloxacin	[3]
8	9, M	Peritoneal fluid	Abdominal pain, pyrexia	None	Perforated appendicitis	*Comamonas kerstersii Streptococcus constellatus, Bacteroides fragilis*	Amoxicillin/clavulanic acid, gentamicin and metronidazole and then oral amoxicillin/clavulanic acid	[3]
9	54, F	Psoas abscess	Septic shock. Diabetic ketoacidosis	Obesity, hypertension and diabetes	Left psoas abscess	*Comamonas kerstersii, Escherichia coli, Bacteroides fragilis*	Piperacillin/ tazobactam + vancomycin and then trimethoprim/ sulfamethoxazole + metronidazole	[8]
10	15, F	Peritoneal fluid	Abdominal pain, vomiting, febrile syndrome	No underlying disease	Purulent peritonitis, salpingitis	*Comamonas kerstersii, Escherichia coli, Streptococcus anginosus, Bacteroides fragilis*	Ceftriaxone, metronidazole, doxycycline, oral amoxicillin/ clavulanic acid	[8]
11	36, F	Peritoneal fluid	Abdominal pain, nausea, vomiting	No underlying disease	Gangrenous appendicitis, purulent peritonitis	*Bacteroides fragilis, Comamonas kerstersii*	Ampicillin, ampicillin/sulbactam, piperacillin/tazobactam	[8]
12	61, M	Peritoneal fluid	Abdominal pain, febrile syndrome	No underlying disease	Gangrenous acute appendicitis, acute peritonitis	*Escherichia coli, Comamonas kerstersii*	Piperacillin/tazobactam	[8]
13	40, M	Peritoneal fluid	Abdominal pain, febrile syndrome, vomiting	No underlying disease	Gangrenous acute appendicitis, acute generalized peritonitis	*Escherichia coli, Comamonas kerstersii*	Ceftriaxone, ornidazole	[8]
14	38, F	Peritoneal fluid	Abdominal pain, febrile syndrome	No underlying disease	Acute appendicitis, pelvic abscess	*Escherichia coli Comamonas kerstersii*	Ciprofloxacin, metronidazole	[8]
15	18, F	Peritoneal fluid	Abdominal pain, fever, nausea, vomiting	No underlying disease	Gangrenous acute appendicitis with perforated base, generalized peritonitis	*Streptococcus viridans group Comamonas kerstersii*	Piperacillin/tazobactam, ampicillin/ sulbactam	[8]
16	21, F	Peritoneal fluid	Abdominal pain, febrile syndrome	No underlying disease	Gangrenous appendicitis, purulent peritonitis	*Citrobacter amalonaticus, Comamonas kerstersii*	Ceftriaxone, ornidazole	[8]
17	84, M	Peritoneal fluid	Abdominal pain, febrile syndrome	No underlying disease	Perforated appendicitis	*Escherichia coli, Comamonas kerstersii*	Ceftriaxone, ornidazole	[8]
18	32, M	Peritoneal fluid	Fever, retroperitoneal haematoma	smoking, inhalational drugs	Firearm wound; colon perforation, faecal peritonitis, interloop abscesses	*Streptococcus anginosus, Comamonas kerstersii*	Piperacillin/tazobactam, vancomycin, colistin + drainage	[8]
19	19, M	Peritoneal fluid	Acute abdomen	No underlying disease	Perforated appendicitis, peritonitis appendicular	*Escherichia coli, Comamonas kerstersii*	Ampicillin, gentamicin, metronidazole	[8]
20	35, M	Peritoneal fluid	Abdominal pain	No underlying disease	Peritonitis appendicular	*Escherichia coli, Comamonas kerstersii*	Ciprofloxacin, metronidazole	[8]
21	67, M	Peritoneal fluid	Acute abdomen	Glioblastoma multiforme, hypertension, mitral valve surgery	Purulent peritonitis resulting from perforated sigmoid	*Escherichia coli, Streptococcus viridans group, Bacteroides fragilis, Comamonas kerstersii*	Ampicillin/sulbactam, amoxicillin/ clavulanate after oral + colostomy	[8]
22	63, M	Peritoneal fluid	Abdominal pain	Diabetes, dyslipidaemia, obesity	Diverticulum, appendicular purulent peritonitis	*Escherichia coli, Comamonas kerstersii*	Ciprofloxacin, metronidazole	[8]
23	5, F	Urine	Abdominal and low back pain, fever and vomiting	No underlying disease	None	*Comamonas kerstersii*	Ceftriaxone, then piperacillin/ tazobactam followed by oral amoxicillin/clavulanic acid.	Present case

The minimal inhibitory concentration (μg/mL) using the VITEK 2 System (AST-082 panel; bioMérieux, Marcy l'Étoile, France) was as follows: ampicillin 16; Ampicillin/sulbactam (AMS) $\leq$ 2; cephalothin $\leq$ 2; piperacillin-tazobactam $\leq$ 4; cefotaxime 2; ceftazidime 2; cefepime 8; imipenem $\leq$ 0.25; meropenem $\leq$ 0.25; amikacin 16; gentamycin 8; ciprofloxacin $\leq$ 0.25; Trimethoprim/sulfamethoxazole (TMS) $\leq$ 2; colistin $\leq$ 0.5. The minimal inhibitory concentration results were interpreted using Clinical and Laboratory Standards Institute categories [10].

With the report of growth of a nonfermenting Gram-negative bacillus, antibiotic therapy was changed to piperacillin/tazobactam 200 mg/kg per day provided intravenously every 8 hours for a 10-day period. The patient had a favourable progress and was therefore discharged, completing a 14-day treatment with oral amoxicillin/clavulanic 50 mg/kg per day.

The association of *Comamonas kerstersii* with peritonitis resulting from the presence of perforated appendix has previously been described by us [7]. Recently, other authors have also reported new cases of intra-abdominal infections due to perforated appendix [3], as well as the first case of bacteraemia by *C. kerstersii* in a 65-year-old patient with signs of diverticulosis [4]. In addition, we have recently pointed out the isolation of this microorganism, not previously described in the literature, from two forms of unusual infection presentations: psoas abscess and pelvic peritonitis [8]. The increase in *C. kerstersii* isolation frequency from human infections in the literature points at it as an emerging pathogen (Table 1). These findings suggest that infections caused by *C. kerstersii* could be underestimated because identification of isolates using only conventional phenotypic methods does not allow accurate determination of the genus. In the pre–MALDI-TOF MS era, the identification of *Comamonas* isolates was only achieved by phenotypic methods, which do not allow differentiation among species of genus [7].

The use of mass spectrometry in routine bacterial identification has revolutionized microbiology. The potential for identification at the species level within minutes makes MALDI-TOF MS an ongoing revolution in the clinical microbiology laboratory [5]. MALDI-TOF MS is a powerful tool not only for routine bacterial identification but also for identification of rare bacterial species implicated in human infectious diseases [6]. In this regard, we have recently demonstrated the ability of MALDI-TOF MS to identify 29 genera of nonfermenting Gram-negative bacilli, including uncommon species. Specifically, *C. kerstersii* isolates (n = 10) included in our study were correctly identified at the species level [11]. In agreement with Opota et al. [4], we consider that the use of this revolutionary methodology could help establish the epidemiology and clinical impact of this species.

Here we describe the first case of urinary tract infection due to *C. kerstersii*. In view of the finding of this unusual pathogen as a potential cause of urinary tract infection, we looked for this microorganism in the patient's faeces, but only a few colonies of *C. kerstersii* were found in a culture mainly containing *Escherichia coli*. *Comamonas kerstersii* growth in pure culture of more than 10^5 CFU/mL in urine culture, the presence of leukocyturia and the intestinal colonization associated with clear clinical and radiologic signs of pyelonephritis in this patient pointed to *C. kerstersii* as the aetiologic agent of this infection; the ascending path was the most likely route of infection.

Comamonas kerstersii isolation from the stool of patients with gastroenteritis has recently been reported by us [8] and by other authors [3], indicating a potential intestinal carriage resulting from environmental exposure.

We highlight the possibility of *C. kerstersii* isolation from extraintestinal sites. Therefore, the isolation of *C. kerstersii* from urinary tract infections broadens the spectrum of infections caused by this microorganism.

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Conflict of interest

None declared.

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