

Unusual presentations of *Comamonas kerstersii* infection

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Abstract

The association of *Comamonas kerstersii* with peritonitis resulting from the presence of perforated appendix has previously been described by our research team. In the present study, we describe the isolation of this microorganism from two forms of unusual presentations of *C. kerstersii* infection not previously described in the literature: localized intra-abdominal infection (psoas abscess) and pelvic peritonitis.

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Introduction

Comamonas spp. are nonfermenting Gram-negative rods originally described in 1985 including a single species, *Comamonas terrigena* [1]. In 1987, *Pseudomonas acidovorans* and *Pseudomonas testosteroni* were later reclassified as members of the genus *Comamonas*. Subsequently *Comamonas acidovorans* was reclassified as *Delftia acidovorans* [2]. The previous single species *C. terrigena* actually comprises three distinct DNA hybridization groups: *Comamonas terrigena*, *Comamonas aquatica* and *Comamonas kerstersii* [3]. Phenotypic and genotypic differences among these three DNA hybridization groups indicate they are different species: *Comamonas terrigena* (DNA group 1), *Comamonas aquatica* (*C. terrigena* DNA group 2), and *Comamonas kerstersii* (*C. terrigena* DNA group 3) [3].

C. kerstersii is a recognized phytopathogen, widely distributed in water, soil and plants, with the ability to survive in aquatic

environments, making it an opportunistic pathogen [3]. We have previously described the ability of *C. kerstersii* to cause perforated-appendix related intra-abdominal infections [4], which had never previously been reported.

Here we describe two cases of *C. kerstersii* infections with unusual clinical presentations: first, a case of a psoas abscess and absence of free fluid in the abdominal cavity in a diabetic patient, and second, a case of pelvic peritonitis (peritonitis resulting from genital tract infection) in a patient with grade IV pelvic inflammatory disease. In addition, we report another 12 clinical cases of intra-abdominal infections by this organism from patients admitted to our hospital.

Material and methods

Clinical specimens were properly collected and processed according to the *Manual of Clinical Microbiology* [5]. After 48 hours of incubation at 35°C at ambient air temperature, growth of a nonfermenting Gram-negative bacillus was observed in all abdominal cavity cultures. White, smooth colonies with entire edges grew to a diameter of 1.5 mm on blood agar at ambient air temperature.

Phenotypic identification of all isolates ($n = 14$) was performed by conventional biochemical tests [6] and by the scheme

proposed by Wauters and Vaneechoutte [7] on the basis of three enzymatic tests, i.e. oxidase, trypsin (benzyl–arginine arylamidase or benzyl–arginine aminopeptidase) and pyrrolidonylaminopeptidase. Additionally, other biochemical tests, such as determination of acid production from glucose, colistin susceptibility, desferrioxamine susceptibility, urease production, motility, nitrate reduction, growth at 42°C and tyrosine hydrolysis, were required to reach final identification. *C. kerstersii* strain 25 (GenBank accession no. KC714047) was used as a control strain for phenotypic identification studies. In addition, identification by matrix-assisted desorption ionization–time of flight mass spectrometry (MALDI-TOF MS; Bruker Daltonics, Bremen, Germany) was carried out. Phenotypic identification was confirmed with molecular identification (amplification and subsequent sequencing of the *gyrB* gene (coding for subunit beta of DNA gyrase) using the primers described by Tayeb *et al.* [8].

Antibiotic susceptibility test was performed using the VITEK 2 System using the AST-082 panel (gram negative susceptibility card). The minimum inhibitory concentration results were interpreted using Clinical and Laboratory Standards Institute categories [9].

Case 1

A 54-year-old woman with a history of obesity, hypertension and diabetes was admitted to the intensive care unit with septic shock. She developed diabetic ketoacidosis with polyphagia, polydipsia and polyuria and progressive sensory deterioration until 2 days before hospital admission. A peripheral venous blood sample taken at admission had the following laboratory findings: white blood cell count, 32 300/mm³ (normal range, 3500/mm³ to 10 500/mm³); haematocrit, 42% (normal range, 38–46%); haemoglobin count, 11.5 g/dL (normal range, 12.3–15.3 g/dL); and platelet count, 268 000/mm³ (normal range, 150 000/mm³ to 450 000/mm³).

An arterial blood gas test revealed pH 7.09, partial CO₂ pressure 20.1 mm Hg, partial O₂ pressure 71.0 mm Hg, HCO₃ concentration 6 mmol/L, 82.8% O₂ saturation and a –22 mEq/L base excess—parameters consistent with metabolic acidosis.

The serum biochemistry results were as follows: [Na], 127 mEq/L; [K], 2.4 mEq/L; blood urea nitrogen level, 60 mg/dL; creatinine level, 8.5 mg/dL; and glycemia, 627 mg/dL.

Her husband told us that 15 days before admission the patient had experienced lumbosacral pain, for which she consulted different medical institutions and received different analgesic treatments. A 14 by 89 mm heterogeneous collection was found in the left parietocolic space by abdominal ultrasound. However, the abdominal cavity was free of fluid.

An abdominal computed tomographic scan revealed a left psoas abscess. Percutaneous drainage was performed, which

resulted in our obtaining abundant purulent material, which was sent for culture. Empirical treatment with piperacillin/tazobactam 4.5 g iv every 8 hours and vancomycin 1 g iv every 12 hours was started for a 14-day period. *C. kerstersii* with *Escherichia coli* and *Bacteroides fragilis* growth was observed in the culture. The antibiotic treatment scheme was changed to trimethoprim/sulfamethoxazole 15 mg/kg (based on the trimethoprim component) iv/12 hours and metronidazole 500 mg iv/8 hours for 30 days. The patient's response was favourable, and she was therefore discharged.

Case 2

A 15-year-old female patient was assisted at a healthcare centre in the suburbs with an abdominal pain episode of 72 hours of progress associated with vomiting and fever in the last 12 hours. Symptomatic treatment was indicated.

As a result of the persistence and worsening of the signs and symptoms, she consulted the Hospital de Clínicas Emergency Room, where she was found to be seriously ill and febrile, with abdominal splinting. The admission diagnosis was acute surgical abdomen.

The peripheral venous blood sample taken at admission had the following laboratory findings: white blood cell count, 14 000/mm³; haematocrit, 42%; haemoglobin count, 14.2 g/dL; and platelet count, 330 000/mm³. Her serum biochemistry results were as follows: [Na], 137 mEq/L; [K], 3.5 mEq/L; blood urea nitrogen level, 16 mg/dL; creatinine level, 8.2 mg/dL; and glycemia 113 mg/dL.

Because perforated appendicitis was suspected, the consulting paediatric surgeon performed an emergency laparoscopy. During the exploratory laparoscopy examination, purulent four-quadrant peritonitis was observed, but appendicitis or bowel perforation could not be confirmed. The appendix was intact; therefore, no appendectomy was performed. Instead, an erythematous, congestive and enlarged (5 × 3 cm) right fallopian tube coated with fibrin as well as inflamed ovaries were observed, so right salpingectomy was performed. Peritoneal fluid and two salpingectomy samples were taken for culture. A drainage tube was placed in the Douglas pouch. *C. kerstersii*, *E. coli*, *Streptococcus anginosus* and *B. fragilis* grew in the peritoneal fluid sample and in both salpingectomy samples.

The patient received antibiotic treatment with ceftriaxone (2 g/day iv, 6 days), metronidazole (500 mg/12 hours by mouth, 8 days) and doxycycline (100 mg/12 hours by mouth, 8 days), completing a 14-day treatment with amoxicillin/clavulanic acid 500 mg/8 hours by mouth. After completing the antibiotic course, and as a result of a favourable clinical progress, she was discharged from hospital.

Other cases

In 12 other patients in the 2010–2015 period, *C. kerstersii* was isolated from the abdominal fluid of patients with diagnosed acute peritonitis. In most of them (n = 10), it was the result of a gangrenous appendix or perforated appendicitis (Table 1). In all cases, *C. kerstersii* was isolated with accompanying flora. Patients were 18 to 84 years old. The clinical progress of all patients was favourable (Table 1).

In all 14 cases, spectral scores between 2021 and 2145 for *C. kerstersii* were obtained by MALDI-TOF MS. Additionally, *gyrB* gene sequences analysis revealed 99% identity with the *gyrB* sequence of *Comamonas kerstersii* (GenBank accession no. KC714047).

The minimum inhibitory concentration ranges (expressed in µg/mL) of *C. kerstersii* isolates were as follows: ampicillin ≤2 to 32; Ampicillin/sulbactam (AMS) ≤2 to ≤2; cephalothin ≤2 to ≤2; piperacillin/tazobactam ≤4 to ≤4; cefotaxime ≤1 to 4; ceftazidime ≤1 to 8; cefepime ≤1 to 4; imipenem ≤0.25 to 0.5; meropenem ≤0.25 to 0.25; amikacin ≤2 to 16; gentamycin ≤1 to 4; ciprofloxacin ≤0.25 to 2; Trimethoprim-sulfamethoxazole (TMS) ≤2 to 16; colistin ≤0.5 to 16.

Discussion

In 2013 we described the association of *C. kerstersii* isolation from free liquid in the abdominal cavity with the presence of perforated appendix [4]. Other authors have reported new

cases of intra-abdominal infections due to perforated appendix [10]. These findings suggest that infections caused by *C. kerstersii* could be underestimated because the identification of isolates using only conventional phenotypic methods does not allow accurate determination of the genus. In previously published cases of *Comamonas* infection, the identification of isolates was only achieved by phenotypic methods, which do not allow differentiation among species of genera [11–17].

In this work, we highlight the isolation of this species from a localized intra-abdominal infection: a psoas abscess of potential renal origin in a diabetic patient (patient 1). In a series of 42 cases of psoas abscesses studied by Wong et al. [18], the most common causative organism for a primary psoas abscess was methicillin-susceptible *Staphylococcus aureus*, while for abscesses originating in the gastrointestinal or urinary tract it might be polymicrobial [18–21]. In this type of infections, *E. coli* was usually the most common organism [19,21]. Other organisms—including *Bacteroides* spp., *Peptostreptococcus* spp., *Streptococcus viridans* group and *Enterococcus faecalis*—were also reported [18–21]. To date *C. kerstersii* has not yet been described as being involved in the cause of psoas abscess.

In the second case we describe here, the peritonitis infection might have ascended from the vagina through the fallopian tubes because this patient had salpingitis [22,23]. This source of infection has been described in a previously healthy 31-year-old woman [24].

TABLE 1. Clinical and microbiologic characteristics of patients with secondary peritonitis due to *Comamonas kerstersii*

Case no.	Age (years)/Sex	Clinical presentation	Underlying disease	Predisposing condition	Identified pathogens	Antibiotic treatment	Progress
1	36, F	Abdominal pain, nausea, vomiting	No underlying disease	Gangrenous appendicitis, purulent peritonitis	<i>Bacteroides fragilis</i> , <i>Comamonas kerstersii</i>	Ampicillin, ampicillin/sulbactam, piperacillin-tazobactam	Favourable
2	61, M	Abdominal pain, febrile syndrome	No underlying disease	Gangrenous acute appendicitis, acute peritonitis	<i>Escherichia coli</i> , <i>C. kerstersii</i>	Piperacillin/tazobactam	Favourable
3	40, M	Abdominal pain, febrile syndrome, vomiting	No underlying disease	Gangrenous, acute appendicitis, acute generalized peritonitis	<i>E. coli</i> , <i>C. kerstersii</i>	Ceftriaxone, ornidazole	Favourable
4	38, F	Abdominal pain, febrile syndrome	No underlying disease	Acute appendicitis, pelvic abscess	<i>E. coli</i> , <i>C. kerstersii</i>	Ciprofloxacin, metronidazole	Favourable
5	18, F	Abdominal pain, fever, nausea, vomiting	No underlying disease	Gangrenous acute appendicitis with perforated base, generalized peritonitis	<i>Streptococcus viridans</i> group, <i>C. kerstersii</i>	Piperacillin/tazobactam, ampicillin/sulbactam	Favourable
6	21, F	Abdominal pain, febrile syndrome	No underlying disease	Gangrenous appendicitis, purulent peritonitis	<i>Citrobacter amalonaticus</i> , <i>C. kerstersii</i>	Ceftriaxone, ornidazole	Favourable
7	84, M	Abdominal pain, febrile syndrome	No underlying disease	Perforated appendicitis	<i>E. coli</i> , <i>C. kerstersii</i>	Ceftriaxone, ornidazole	Favourable
8	32, M	Fever, retroperitoneal haematoma	smoking, inhalational drugs	Firearm wound; colon perforation, faecal peritonitis, interloop abscesses	<i>Streptococcus anginosus</i> , <i>C. kerstersii</i>	Piperacillin/tazobactam, vancomycin, colistin + drainage	Favourable
9	19, M	Acute abdomen	No underlying disease	Perforated appendicitis, peritonitis appendicular	<i>E. coli</i> , <i>C. kerstersii</i>	Ampicillin, gentamicin, metronidazole	Favourable
10	35, M	Abdominal pain	No underlying disease	Peritonitis appendicular	<i>E. coli</i> , <i>C. kerstersii</i>	Ciprofloxacin, metronidazole	Favourable
11	67, M	Acute abdomen	Glioblastoma multiforme, hypertension, mitral valve surgery	Purulent peritonitis resulting from perforated sigmoid	<i>E. coli</i> , <i>S. viridans</i> group; <i>B. fragilis</i> , <i>C. kerstersii</i>	Ampicillin/sulbactam, amoxicillin/clavulanate acid after oral + colostomy	Favourable
12	63, M	Abdominal pain	Diabetes, dyslipidemia, obesity	Diverticulum, appendicular purulent peritonitis	<i>E. coli</i> , <i>C. kerstersii</i>	Ciprofloxacin, metronidazole	Favourable

Salpingitis involves inflammation of the fallopian tube. It usually presents as acute abdomen, and because appendicitis usually includes the same symptoms, salpingitis diagnosis may be delayed until the appendix is surgically explored [25]. Salpingitis, mainly reported in sexually active women, is usually caused by sexually transmitted microorganisms, such as *Neisseria gonorrhoeae* and *Chlamydia trachomatis* [26], although other microorganisms that colonize the lower genital tract can ascend to the endometrium, producing endometritis, salpingitis and peritonitis. The polymicrobial aetiology of acute salpingitis has been well documented [26,27], showing that anaerobes (*Peptostreptococcus* and *Bacteroides* spp.), *Enterobacteriaceae* (*E. coli*) and aerobic streptococci are the most frequently isolated microorganisms [26,27]. Moreover, infrequent microorganisms like *Edwardsiella tarda* [28] and *Plesiomonas shigelloides* [29] have also been implicated in salpingitis. However, to our knowledge, the isolation of *C. kerstersii* has not previously been reported in this type of infection.

In addition, the isolation of *C. kerstersii* from the stool of patients with gastroenteritis (seven cases; data not shown) indicates potential intestinal carriage resulting from environmental exposure (i.e. water or food). Other authors have also proposed water as the source of intestinal infection [30,31]. Biswas *et al.* [10] reported the isolation of *C. kerstersii* from the faecal samples of 27 patients with diarrhoea, suggesting that the environmental exposure leading to the intestinal carriage may be more common than most commonly supposed.

In our previous study, we pointed out the isolation of *C. kerstersii* from peritonitis resulting from perforated appendix; in this work, we highlight the isolation of this microorganism from peritonitis resulting from salpingitis and from a localized intra-abdominal infection (psoas abscess).

The presence of a nonfermenting Gram-negative rod in the abdominal cavity of a patient with intestinal perforation should raise suspicion of *C. kerstersii*. Also, *C. kerstersii* isolation from abscesses broadens the spectrum of infections caused by this microorganism.

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Conflict of interest

None declared.

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