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Comamonas testeroni Bacteremia in a Young Male with Pancreatitis: A Case Report

Lovell ARO[1]* and Forde CA[2]

[1]*Department of Internal Medicine, Queen Elizabeth Hospital, Barbados*
[2]*Department of Hospital of Infection Prevention and Control/Infectious Diseases, Queen Elizabeth Hospital, Barbados*

Abstract

Background: *Comamonas Testeroni* is a gram-negative bacillus which rarely causes infection in humans. This case represents the first case reported in the English-speaking Caribbean. As a pathogen it is most often implicated in intra-abdominal infections, most often appendicitis; this cause is the first report of pancreatitis/ pancreatic collection being the nidus of infection.

Case study: Informed consent was attained for the use of their clinical details and images. It was assured that identifying demographics were removed to ensure confidentiality. There were no vulnerable groups or individuals in this case.

Conclusion: *C. testeroni* rarely causes infection in humans but when it does, an intra-abdominal source is most often the source of infection.

Keywords: Infection; Pancreatitis; Treatment

Introduction

Comamonas is a rare low virulence organism seldom implicated in human disease. This ubiquitous organism can survive in most environments including the hospital environment. It is an aerobic, pink-pigmented, oxidase positive gram negative bacillus with four known species: *C. testeroni, C. terrigena, C. denitrificans and C. nitritivorans* [1-5]. *C. testeroni* is the most common of the species, and is given this name as it can grow in a media containing testosterone as its only source of carbon [2]. Since it was first discovered in 1987 [3-5] no case has previously been described in the English speaking Caribbean.

Infections have been documented most commonly intraperitoneally, most often in association with perforated appendicitis, but cerebrospinal fluid and blood, especially in immunocompromised host, have also been noted sites of infection [4]. No case has been published citing pancreatitis as the primary infection. Here we describe a case of *C. testeroni* bacteremia in a young male with pancreatitis.

Case Study

A 39-years-old male with a history of asthma and heavy alcohol misuse presented to a tertiary level institution with the complaints of abdominal pain and vomiting. He had been drinking heavily over the preceding days. He consumed approximately 475 ml of dark rum over the preceding 48 hours, which was in keeping with quantities he usually consumed during his weekend binges.

His vitals on the day of presentation were;

- **Temperature:** 36.0°C,
- **BP:** 140/85 mmHg,
- **HR:** 109 bpm,
- **RR:** 26 bpm,
- **O_2 saturation:** 100% on room air.

His cardiovascular, respiratory and neurological examinations were normal and his abdominal exam was significant for mild epigastric tenderness with no guarding or rebound tenderness. His arterial blood gas showed a metabolic acidosis, RBS was 6.3 mmol/dL. Urinalysis was normal and ECG showed sinus tachycardia (Table 1).

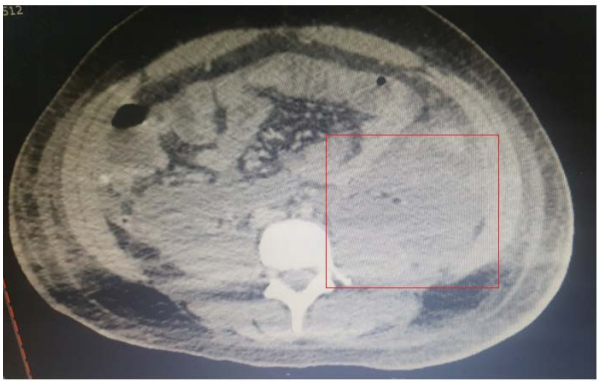

Figure 1: Showing intra-abdominal collection.

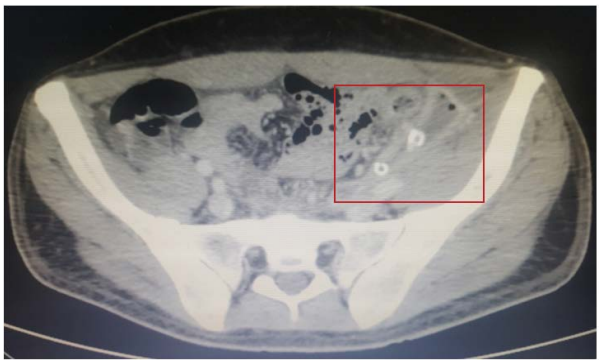

Figure 2: Showing collection with drain inserted.

***Corresponding author:** Lovell ARO, Department of Internal Medicine, Queen Elizabeth Hospital, Barbados, Tel: +12462495458; E-mail: andrelovellms@gmail.com

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Hb	17.4	g/dl
Wbc	8.8×10^9	Cells/ml
Plt	128×10^9	Cells/ml
Na	132	mmol/L
K	5.3	mmol/L
Cl	100	mmol/L
HCO_3	18	mmol/L
Urea	6.2	mmol/L
Creat	161	Umol/L
Bili T	25	mg/dL
AST	179	U/L
ALT	74	U/L
ALP	108	U/L
Amylase	430	U/L

Table 1: Showing patients laboratory results.

Antibiotic	Susceptibility	MIC
Amoxicillin/Clav	Intermediate	>16/8
Ampicillin	Intermediate	>16
Cefazolin	Resistant	>4
Cefepime	Sensitive	<4
Cefotaxime	Sensitive	<2
Ceftriaxone	Sensitive	<4
Ceftazidime	Intermediate	16
Ciprofloxacin	Sensitive	<1
Levofloxacin	Sensitive	<2
Ertapenem	Resistant	>2
Meropenem	Sensitive	<1
Gentamicin	Resistant	>8
Pip/Tazo	Sensitive	<16
Trim/Sulf	Sensitive	<2/38

Table 2: Showing sensitivity profile of the *C. testeronii* cultured.

The patient was diagnosed as acute alcohol induced pancreatitis and transferred to the intensive care unit. On day 2 of admission the patient was noted to be more ill appearing, more tachypneic in the setting of worsening metabolic acidosis and had a fall in his consciousness and was subsequently intubated. The patient became haemodynamically unstable requiring vasopressor support and became febrile with a temperature of 39.5°C and was started on meropenem 1g IV every 8 hours, after blood cultures were taken.

Initial blood cultures were no growth at after 72 hours of incubation, urine cultures were also no growth. Tracheal aspirates grew *C. parasilosis* and the patient, under the guidance of the Infectious Disease Team, was started on fluconazole 800 mg IV stat followed but 400 mg IV daily and all subsequent aspirates were sterile. Despite this the patient continued to worsen; he continued to be febrile, continued to require vasopressor support, developed acute kidney injury requiring haemodialysis and significant thrombocytopenia and persistently elevated WBC.

As such, an abdominal CT scan was done and showed a peripancreatic collection. This collection was drained percutaneously and fluid cultured we also no growth at 48 hours (Figures 1 and 2). The patient completed a 21 day course of meropenem and a 14 day course of fluconazole but showed no clinical improvement. Repeat blood cultures remained negative until one was done after the above antibiotic were completed and grew *C. testeroni* and the patient was started on trimethoprim/sulfamethazole. Following this all the patient's clinical parameter improved with him becoming afebrile, normalisation of his WBC and resolution of his thrombocytopenia and acute kidney injury, and him being successfully weaned from ventilatory and circulatory support Table 2 below shows the sensitivity profile of the organism.

Discussion

Comamonas species seldom causes human infection but when it does it is usually associated with intra-abdominal sepsis and is often seen in immunocompromised host. The organism has been isolated in soil and in water sources ranging from natural bodies of water to distilled water stores within hospital. It is also thought to be a part of the endogenous human microbiome but when it acts as a pathogen the abdominal cavity is the most common site of infection; with the majority of these cases being associated with ruptured appendicitis, suggesting that the organism has a predilection to the organ [4,5].

Conclusion

C. testeroni is usually sensitive to fluoroquinolones, cephalosporins, penicillins, aminoglycosides, and trimethoprim/sulfathoxazole. Many cases have been successfully treated with beta-lactams, however the organism isolated in this case showed some resistance to the penicillins and trimethoprim/Sulfathoxazole was used. The fact that the organism was sensitive to meropenem may explain why the initial series of blood cultures were negative and why the pus drained from the intra-abdominal collection was did not culture any organisms. Fortunately, treatment with appropriate antibiotics leads to resolution and death from infection is rare. *C. testeroni* rarely causes infection in humans but when it does, an intra-abdominal source is most often the source of infection.

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