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A rare case of comamonas testosteroni causing spontaneous bacterial peritonitis in a patient with alcoholic liver cirrhosis: A case report and review of literature

Debomallya Bhuyan[1], Kunal Bhowmik[2], Mesashebha Rupsi[3], Arijit Kumar Das[3]
[1] Head of Department, Department of Internal Medicine, Nazareth Hospital, Shillong, Meghalaya, India
[2] Research Scholar Department of Internal Medicine, Nazareth Hospital, Shillong, Meghalaya, India
[3] Consultant, Department of Internal Medicine, Nazareth Hospital, Shillong, Meghalaya, India

Abstract
Comamonas testosteroni is a rare human pathogen with only 52 documented cases till date. Among them, 13 cases reported the isolation of organism from peritoneal fluid, 12 of which were associated with an intra-abdominal source of infection. A definitive case of Spontaneous bacterial peritonitis caused by Comamonas testosteroni is yet to be reported.
We are reporting a case of a 69 years old male, with no prior comorbidities, who presented with abdominal distension and pain for 4 weeks along with swelling of his legs. He was started on empirical therapy for spontaneous bacterial peritonitis owing to a high degree of suspicion. His liver function tests and imaging studies were suggestive of decompensated chronic liver disease with no foci of infection. Peritoneal fluid analysis confirmed the diagnosis of spontaneous bacterial peritonitis. He did not respond to cefotaxime and his condition worsened, developing encephalopathy. Peritoneal fluid culture showed growth of Comamonas testosteroni, resistant to cefotaxime. Antibiotic was changed according to sensitivity report. After 3 days, repeat paracentesis confirmed the resolution of infection. Patient's condition improved and he was eventually discharged.
This case highlights the possibility of uncommon pathogens causing SBP and the resistance of such pathogens to commonly advocated empirical therapy.

Keywords: spontaneous bacterial peritonitis, comamonas testosteroni, SBP, case report

Introduction

Spontaneous Bacterial Peritonitis (SBP) is defined as an acute infection of ascitic fluid in patients with cirrhosis in the absence of an intraabdominal or surgically treatable source of infection. It is a frequent complication particularly in patients with decompensated cirrhosis accounting for approximately 10%-30% of all bacterial infections in cirrhosis. A combination of translocation of enteric bacteria across the intestinal lumen and impaired host defence mechanisms in cirrhotic patients have been implicated in its pathogenesis. [1, 2] SBP is usually suspected when a cirrhotic patient presents with abdominal pain, fever, encephalopathy but incidences of asymptomatic presentation has also been reported with a good minority of patients. The diagnosis of SBP is confirmed by an ascitic fluid polymorphonuclear (PMN) cell count of more than 250/mm3. Ascitic fluid culture in SBP typically reveals the growth of a single organism but are frequently negative. Escherichia coli, Klebsiella have been reported as the most common infecting organisms [3]. However there has been a recent increase in reported incidences of SBP caused by other organisms [4].
Comamonas testosteroni, formerly known as Pseudomonas testosteroni, is an aerobic, motile, non-spore forming, Gram-negative bacteria. It has widespread environmental distribution and is rarely encountered in clinical practise. [5] There have only been a handful of reported cases of Comamonas testosterone causing infections in humans. In 1986, Barbaro *et al*. reported 18 cases of Comamonas testosteroni infection prior to which there were only 2 reported cases. [6] In 2011, Tung-Lin Tsui *et al*. reported a case of chronic Hepatitis B with cirrhosis and hepatocellular carcinoma post trans arterial chemoembolization. [7] In 2015, the first documented case of Comamonas testosteroni in India was reported by B.Swain *et al*. following which 2 more reports were made by Shaika Farooq *et al*. and Tiwari et. al in 2017 and 2019 respectively. [8–10] None of the reports suggested Comamonas testosteroni causing spontaneous infection without a foci.
Geographically, no cases of SBP caused by Comamonas testosteroni have been reported in India among the only three documented cases. Herewith, we are reporting the first case of SBP caused by Comamonas testosteroni in India unresponsive to cefotaxime in a patient with alcoholic hepatic cirrhosis and pneumonia. Since cefotaxime is the go-to empirical antibiotic used in patients with SBP, the purpose of the study is to sensitize the clinicians regarding the possibility of unusual causative organisms which might not respond to conventional therapy.

Case report

A 69 years old male presented to the emergency department on 6th February, 2022, with complains of painful abdominal distention that has been gradually increasing for the last 4 weeks and swelling of legs along with intermittent episodes of vomiting.

He had no other complaints and no known comorbidities.

He is a chronic alcoholic averaging 350-500ml of country liquor every day for many years.

On examination, he was hypotensive, maintaining saturation in room air and had icterus with pitting pedal edema.

His abdomen was distended, tender with fluid thrill, GCS was 15/15 with no asterixis and his chest had fine crepitations bilaterally.

His initial blood reports showed 18900 WBC/cumm with 95% neutrophils, peripheral blood picture showed neutrophilia with presence of macrocytes. He had a deranged coagulation profile (PT-23.8 seconds, INR-1.9, APTT 92.9 seconds) and his liver function test showed a hepatocellular and cholestatic pattern of injury with conjugated hyperbilirubinemia and a reversed albumin globulin ratio of 1:1.6 (Total bilirubin 14.8 mg/dL, Conjugated bilirubin 13.1 mg/dL, SGOT 201 U/L, SPGT 58 U/L, Alkaline phosphatase 203 U/L, GGT 496 U/L, Albumin 2.5 g/dL, Globulin 3.9 g/dl). His renal function was within normal parameters, urine had no evidence of proteinuria, viral markers were negative and procalcitonin was 1.26. His chest radiography showed multifocal opacities in both lung fields (Figure 1). Peritoneal fluid analysis showed 1000 RBC/cumm, 9390 WBC/cumm with 95% neutrophils (corrected PMN count- 8916/cumm) and 2 g/dL protein. His Child Pugh Score on admission was 12 (Class C).

He was started on noradrenaline, octreotide, human albumin, vitamin K, diuretics, thiamine and 2g cefotaxime every 8 hourly empirically after performing diagnostic paracentesis and was given 6 units of fresh frozen plasma.

On day 2, his stool tested negative for occult blood and he was normotensive. Noradrenaline and octreotide were stopped and prednisolone 40mg/day was started in view of his Maddrey's Discriminant Function being 62.2. Abdominal Ultrasound and Transient elastography were done which showed coarse hepatic echotexture with ascites suggestive of chronic liver disease and a median stiffness of 75kPa respectively.

On Day 3, he developed hepatic encephalopathy and was requiring supplemental oxygen. Diuretics were stopped and he was started on rifaximin, lactulose and L-Ornithine L-Aspartate. Peritoneal fluid culture showed moderate growth of Comamonas testosterone sensitive to Cefoperazone/Sulbactam which warranted a change of antibiotics. Blood and urine culture showed no growth.

On day 4, repeat chest radiography showed increase in consolidations following which levofloxacin was added to his treatment regimen (Figure 2).

On day 6, his GCS was 15/15 and he was maintaining saturation in room air. His LFT and coagulation profile showed gradual improvement (Table 1). Repeat paracentesis showed 148/cumm WBCs (Neutrophils-72%). Cefoperazone/Sulbactam was continued for 5 days duration. Chest radiograph showed partial recession of opacities (Figure 3). He was discharged after 9 days of hospitalization on diuretics, branched chain amino acids, prednisolone, rifaximin and fluroquinolone.

Discussion

Spontaneous bacterial peritonitis is a very frequent complication observed in cirrhotic patients with decompensation. It is an ominous condition with an in-hospital mortality rate of 20%. Patients with cirrhosis, total serum bilirubin greater than 2.5 mg/dL, ascitic fluid total protein less than 1g/dL, variceal haemorrhage, chronic use of proton pump inhibitors and a prior episode of SBP are at an increased of developing SBP in the future. [2] Survivors of SBP usually have a poor prognosis with 1-month, 6-month, 1-year mortality rates of 33%, 50% and 58% respectively. 30-40% patients who develop renal failure are at an increased mortality risk. [11]

Despite E. coli and Klebsiella accounting for more than half of the cases of SBP, reports of uncommon bacteria like Listeria being implicated as the causative organism, has been growing in recent times. [12]

Comamonas testosteroni, a gram negative, aerobe, has a wide range of habitat including soil, water, plants as well as some hospital equipment. [9] Even though previously it had been rarely implicated in human infections, recently more frequent incidences have been reported. A review of literature revealed around 52 reported cases till date most of which had an intraabdominal source (Table 2). Comamonas species are usually susceptible to aminoglycosides, fluoroquinolones, carbapenems, piperacillin-tazobactam and cephalosporins, however reports about its varied resistance patterns has been made. [9, 13] Even though empirical therapy with cefotaxime is considered standard of care in SBP, reports of resistance to 3rd generation cephalosporins have been increasing in recent times. [4, 14]

In this case, we are reporting SBP caused by an unusual pathogen, Comamonas testosteroni, in a patient with alcoholic cirrhosis of liver. SBP was highly suspected based on his presenting symptoms which was later confirmed by paracentesis. Even though the patient was started on cefotaxime, he failed to respond. Owing to his deterioration, antibiotic was changed according to the culture report and the patient showed significant improvement within 3 days. Furthermore, his ultrasound did not reveal any intraabdominal source of infection and he did not have any surgical or catheter related procedure.

The purpose of this report is to raise clinical awareness and emphasize the need for more studies regarding uncommon pathogens causing SBP which might warrant a change in empirical therapy in cases of high suspicion.

Figure legend

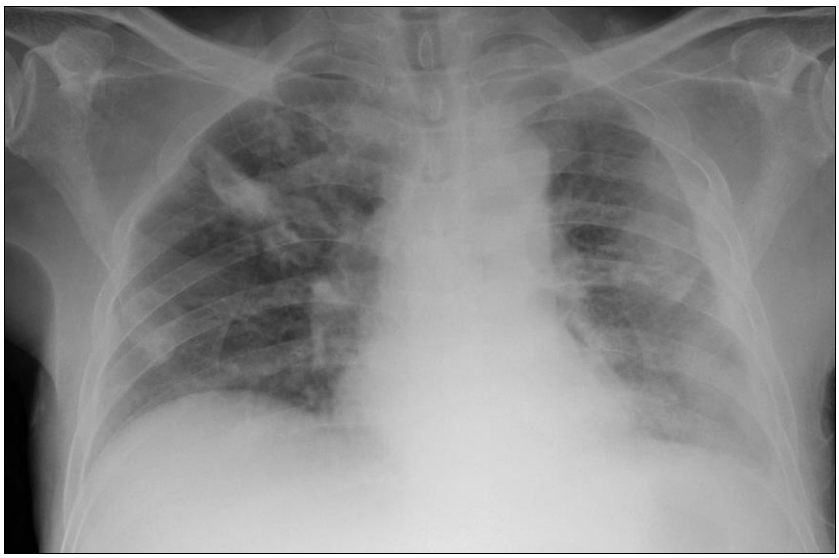

Fig 1: Chest radiograph on admission

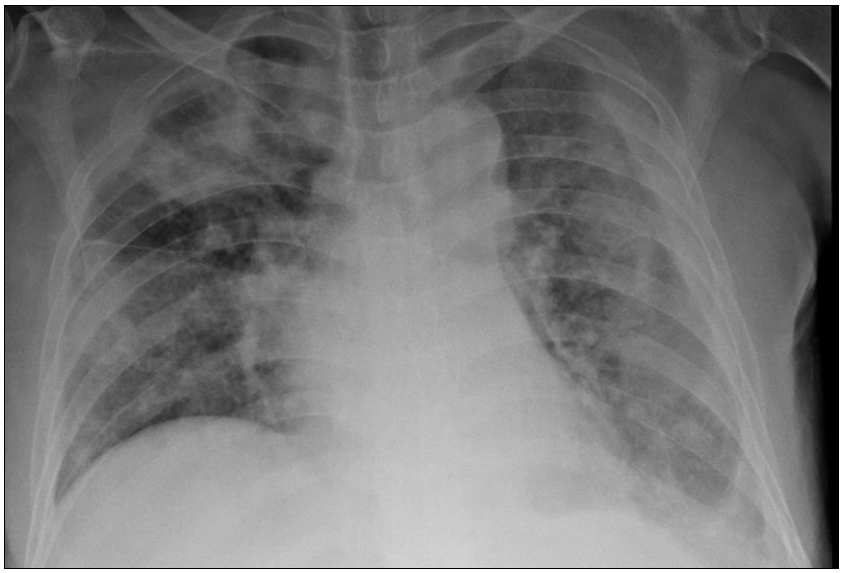

Fig 2: Chest radiograph showing increase in multifocal patchy opacities

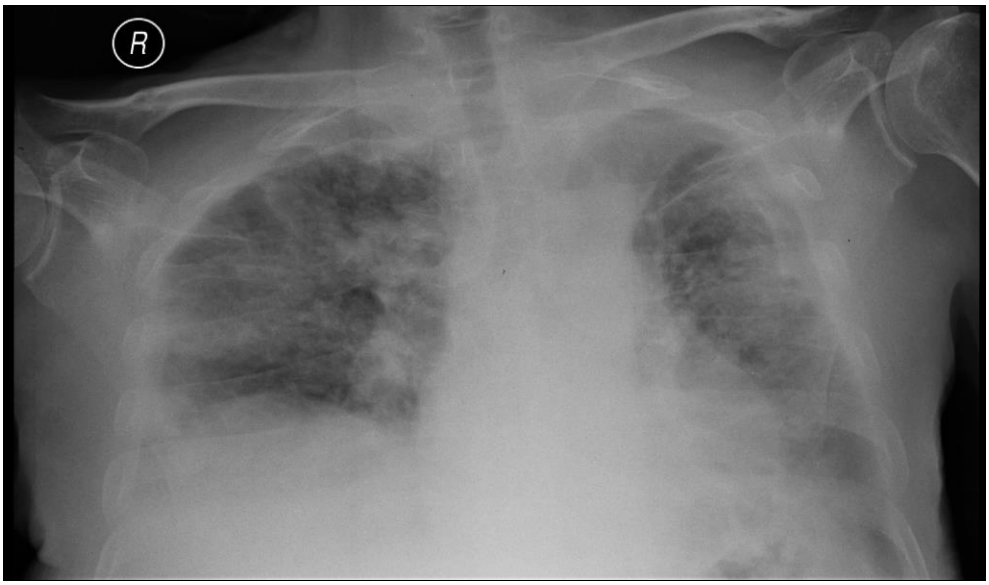

Fig 3: Chest radiograph showing comparative recession of opacities

Table 1: Laboratory parameters during hospital stay

Laboratory parameter	Day 1	Day 3	Day 4	Day 6	Day 9	Reference range
Total Bilirubin	14.8		13.7	13.3	11.1	0.2-1.3 mg/dL
Unconjugated Bilirubin	1.7		1.9	1.6	1.7	0.0-1.1 mg/dL
Conjugated Bilirubin	13.1		11.8	11.7	9.4	0.0-0.3 mg/dL
SGPT	58			41		<50 U/L
SGOT	201			113		17-59 U/L
Alkaline Phosphate	203			98		38-126 U/L
GGT	496			247		15-73U/L
PT	23.8		50.7	23.4		13.5 seconds
INR	1.9		4.3	1.8		1-2
APTT	92.9	79.6		56.5		20.1-30.1 seconds
Total WBC count	18900				14410	cells/cumm

SGPT-Serum glutamic pyruvic transaminase, SGPT- Serum glutamic-oxaloacetic transaminase, GGT-Gamma-glutamyl transferase, PT- Prothrombin time, INR- International normalized ratio, APTT- Activated partial thromboplastin time, WBC- White blood cell.

Table 2: Review of literature pertaining to Comamonas testosteroni

	Age/Sex	Isolated from	Underlying condition	Therapy	Outcome	Author
1	71/F	Blood	Rheumatic heart disease	Penicillin	Cured	Sonnenwirth (1970)
2	31/F	Bone marrow and blood	Sepsis	Kanamycin, tetracycline	Cured	Atkinson *et al*. (1975)
3	17/F	Peritoneal fluid	Appendicitis	No data	Cured	Barbaro *et al*. (1983)
4	59/M	No data	No data	No data	Cured	Barbaro *et al*. (1983)
5	No data	Abdomen	No data	No data	Cured	Barbaro *et al*. (1983)
6	66/M	Peritoneal fluid	No data	No data	Cured	Barbaro *et al*. (1984)
7	14/M	Appendix	Appendicitis	No data	Cured	Barbaro *et al*. (1984)
8	4/M	Blood	No data	No data	Cured	Barbaro *et al*. (1985)
9	28/F	Blood	No data	No data	Cured	Barbaro *et al*. (1985)
10	15/M	Peritoneal fluid	No data	No data	Cured	Barbaro *et al*. (1985)
11	31/M	Abscess	Perforated appendicitis	Cefoxitin; after drainage-ampicillin, clindamycin, gentamicin	Cured	Barbaro *et al*. (1987)
12	24/F	CSF	IVDA	Moxalactam and nafcillin	Cured	Barbaro *et al*. (1987)
13	59/M	Peritoneal fluid	Alcoholic cirrhosis	Cefoxitin	Cured	Barbaro *et al*. (1987)
14	11/M	Peritoneal fluid	Perforated appendicitis	Ampicillin, clindamycin, and Tobramycin	Cured	Barbaro *et al*. (1987)
15	12/F	Peritoneal fluid	Perforated appendicitis	Cefoxitin	Cured	Barbaro *et al*. (1987)
16	21/F	Peritoneal fluid	Perforated appendicitis, pregnancy	Cefoxitin	Cured	Barbaro *et al*. (1987)
17	Stillborn	Cord blood	Maternal IVDA	No data	Stillbirth	Barbaro *et al*. (1987)
18	84/F	Urine	CHF	Ampicillin	Cured	Barbaro *et al*. (1987)
19	24/M	Peritoneal fluid	Appendicitis	Cefoxitin	Cured	Barbaro *et al*. (1987)
20	Newborn	Blood	Maternal IVDA, prematurity	Ampicillin, amikacin	Died within 24 hours	Barbaro *et al*. (1987)
21	No data	Respiratory secretions	AIDS	Ceftazidime	Cured	Franzetti *et al*. (1992)
22	35/M	Bite tissue	Zoonotic infection	Ceftazidime, gentamicin	Cured	Isolato *et al*. (2000)
23	75/F	Blood and CVC	CRBSI in Breast	Ceftazidime, gentamicin	Cured	Le Moal *et al*.

			carcinoma			(2001)
24	89/M	Blood	Tropical fish exposure	Levofloxacin	Cured	Smith *et al*. (2003)
25	50/M	CSF	Cholesteatoma	Meropenem	Cured	Arda *et al*. (2003)
26	49/M	Blood and mitral valve	Infective endocarditis	Cefepime, gentamicin; then ampicillin; surgery	Cured	Cooper *et al*. (2005)
27	54/F	Blood	CRBSI in metastatic esophageal carcinoma	Cefepime, ciprofloxacin, drotrecogin alfa	Cured	Abraham *et al*. (2007)
28	22/M	Peritoneal fluid, blood	Perforated appendicitis	Cefazolin	Cured	Gul M *et al*. (2007)
29	54/M	CSF	Chronic alcoholic, car accident	No data	Died	Jin *et al*. (2008)
30	82/F	Vitreous biopsy	Endophthalmitis, diabetes	Ceftazidime (IV and topical)	Cured	Reddy *et al*. (2009)
31	83/M	Blood	Ischemic CVA	Amikacin, piperacillin/tazobactam	Cured	Katircioglu *et al*. (2010)
32	54/M	Blood	Cellulitis following foot injury	Oxacillin, ciprofloxacin	Cured	Tsui *et al*. (2011)
33	73/M	Blood	Post TACE in HCC	Gentamicin, levofloxacin	Cured	Tsui *et al*. (2011)
34	63/M	Blood	ESRD on hemodialysis	Vancomycin, ceftriaxone	Died	Nseir *et al*. (2011)
35	10/M	Tracheal aspirate	Cerebral palsy, tracheostomy	Ceftriaxone, clarithromycin	Cured	Ozden *et al*. (2011)
36	10/M	Blood	Medulloblastoma, chemotherapy	Ciprofloxacin, amikacin	Cured	Farshad *et al*. (2012)
37	19/F	Blood	Osteosarcoma	Ciprofloxacin, vancomycin, imipenem	Cured	Farshad *et al*. (2012)
38	16/M	Peritoneal fluid	Perforated appendicitis	Amikacin, ampicillin, clindamycin	Cured	Bayhan *et al*. (2013)
39	80/F	Blood	Diabetes mellitus	Cefazolin, doripenem	Cured	Orsini Jose *et al*. (2014)
40	51/M	Aortic valve	Endocarditis	Ciprofloxacin	Cured	Arzu Duran *et al*. (2015)
41	42/F	Blood and urine	Septic shock	Ceftazidime, levofloxacin	Cured	Hyun Jung Kim *et al*. (2015)
42	50F	Blood	CKD, gluteal abscess	Piperacillin-tazobactam then cefoperazone-sulbactam	Died	B Swain *et al*. (2015)
43	4/F	Peritoneal fluid	ESRD on peritoneal dialysis	Ciprofloxacin (intraperitoneal)	Cured	Parolin M. *et al*. (2016)
44	62/M	Blood	Diabetes, ischemic CVA	No data	Died	Pekinturk N. (2016)
45	68/M	Blood	Lung cancer, adrenal metastasis	Cefepime, teicoplanin	Died	Yasayancan N *et al*. (2017)
46	1/F	Blood	Acute gastroenteritis, sepsis	Ceftriaxone	Cured	Ruziaki W. (2017)
47	65/F	Stool	Gastroenteritis	Ciprofloxacin	Cured	Shaika Farooq *et al*. (2017)
48	30/F	Blood	Neutropenia	Moxifloxacin	Cured	Aktar *et al*. (2018)
49	46/F	Blood and urine	Possibility of food and water contamination	Gentamicin, imipenem	Cured	Tiwari *et al*. (2019)
50	4/F	Urine	Persistent cloaca	Ceftazidime, amikacin	Cured	S. Gayenur Buyukberber (2021)
51	No data	Burn swab	Burn wound infection	No data	No data	Ghafil *et al*. (2021)
52	16/M	Burn and CVC	CRBSI in burn	Colistin, amikacin	Cured	Sammoni *et al*. (2022)
53	69/M	Peritoneal fluid	SBP in alcoholic cirrhosis	Cefotaxime; then cefoperazone-sulbactam and levofloxacin	Cured	Present case

Conclusion

SBP is a commonly encountered condition in patients with decompensated cirrhosis, usually associated with a poor prognosis with a quick propensity for deterioration if inadequately treated. Increasing reports of uncommon causative organisms and their varied pattern of resistance is important to report so as to guide future care and careful reconsideration of empirical therapy.

Conflicts of interest and funding
The Authors who have taken part in this study declared that they do not have anything to disclose regarding funding or conflict of interest with respect to this manuscript.

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