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Case Report

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A Rare Pathogen Comamonas Testosteroni: A Case Report and Review of the Literature

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Abstract

Comamonas testosteroni is a Gram- negative, aerobic, motile, non-spore-forming bacillus. It has not been recognized as a component of the endogenous human microflora. Due to its ability to survive in liquid environments, it can survive for a long time in a hospital environment and cause opportunistic infections. Although rare, C. testosteroni has been reported as a cause of cellulitis, peritonitis, endocarditis, meningitis, endophthalmitis, tenosynovitis, pneumonia and bacteremia. Here, we present a case of a 4-year-old girl who was operated on for persistent cloaca with C. testosteroni isolated in her urine culture. Identification studies were performed by MALDI-TOF MS (bioMerieux, France) mass spectrophotometer method. Antibiotic susceptibility tests were performed with the automatic device VITEK-2 Compact (bioMérieux, France). Microorganism was found susceptible to ceftazidime and ciprofloxacin; intermediate susceptible to meropenem and piperacillin / tazobactam and resistant to gentamicin, amikacin, imipenem and trimethoprim-sulfamethoxazole. With this case report, C. testosteroni was reported as the first cause of urinary tract infection in our country and the third in the world.

Introduction

Comamonas testosteroni, formerly known as Pseudomonas testosteroni, is a gram-negative, aerobic, motile, non-fermentative and non-spore forming bacillus [7]. The name "Testosteroni" comes from the bacteria's ability to use the carbon in testosterone metabolism, as is observed in some types of Pseudomonas and fungi [10]. C. testosteroni is commonly found in soil, water, plants, animals and waste all over the world and is considered as an environmental microorganism which is not defined as a human endogenous microflora element [6,7]. Because of its ability to live in liquid environments, this organism is seen as an opportunistic nosocomial pathogen in hospitals and intensive care units. It grows well on routine bacteriologic media such as sheep blood agar and chocolate agar [7]. The need for minimal nutrients to grow even in distiled water and adaptation to different physical conditions leads to its important role as an opportunistic pathogen in hospitals. Since it became clinically important; it has been reported in publications as a causative agent of cellulitis, peritonitis, endocarditis, meningitis, endophthalmitis, tenosynovitis, pneumonia and bacteremia [10].

Case Report

A 4-year-old girl with frequent urinary incontinence and recurrent urinary tract infection despite taking prophylaxis has applied to our hospital for further examination. She was operated

two years ago due to persistant cloaca, a total urogenital mobilization was performed, and postoperative uterus, vagen and partial bladder necrosis occured. She has a colostomy in the left lower quadrant, and has frequent urinary incontinence and recurrent urinary tract infection despite taking prophylaxis.

Urinalysis report was; leukocyte 14 p/HPF, leukocyte esterase ++, bacteria were trace and others were found within normal limits. Colonies grown on blood and MacConkey agar, incubated at 35–37°C, were identified as 99% probability C. testosteroni by mass spectrometry method MALDI-TOF MS (bioMérieux, France). Antibiotic susceptibility tests were performed with the automatic device of VITEK-2 Compact (bioMérieux, France) and interpreted in accordance with EUCAST criteria and it was susceptible to ceftazidime, ciprofloxacin; intermediate susceptible to meropenem, piperacillin / tazobactam; resistant to gentamicin, amikacin, imipenem, trimethoprim-sulfamethoxazole. Ceftazidime was added to the treatment of the patient who was already treated with amikacin. There was no growth in the control urine culture performed after 5 days of treatment. And then the patient was discharged with cure. The increase in pathogenicity and antibiotic resistance of C. testosteroni once again demonstrates the importance of rational antibiotic use. With this case report, C. testosteroni was reported as the first cause of urinary tract infection in our country and the third in the world.

Discussion

C. testosteroni which was first reported in 1975, is an infection agent in samples such as cerebrospinal fluid, abdominal abscess and appendix tissue, most commonly from blood and peritoneal fluid [Table 1]. Most of the previously reported cases were immunodeficient due to malignancy, diabetes mellitus, chronic liver disease, and end-stage renal disease [10]. The agent is most frequently isolated from the blood (20 cases), followed by peritoneal fluid (9 cases), cerebrospinal fluid (3 cases) and urine (2 cases). There are rare cases in which the agent was isolated from cord fluid, respiratory secretions, deep tracheal aspirate, abdominal abscess, bite site tissue (animal bite), aortic valve, appendicitis, stool and vitreous fluid. Appendicitis is found to be the most common predisposing factor in cases detecting C. testosteroni in peritoneal fluid. Although bacteria is not an endogenous flora element, the isolation of a large number of patients with perforated appendicitis (7 out of 9 cases) made us think that this microorganism could create a unique location in the appendix [1].

Eight *C.testosteroni* cases have been reported in Turkey so far. The first case was purulent meningitis in a patient with recurrent cholesteatoma reported by Arda et al. in 2003. The microorganism was isolated from the cerebrospinal fluid and treated with meropenem [3].

The second case was *C. testosteroni* bacteremia in a patient with perforated acute appendicitis. It was isolated from the patient's peritoneal fluid and blood cultures by Gul et al. in 2007. It had been treated with cefazolin [8].

The third case was reported in 2010 by Katircioglu et al. It was a case of bacteremia in an intensive care patient. This case isolated from blood cultures was the first case that showed the development of multiple antibiotic resistance including imipenem [9].

The fourth case was the pneumonia case in an intensive care patient reported by Ozden et al. in 2011. Microorganism isolated from endotracheal aspirate culture; was found susceptible to amikacin, imipenem, levofloxacin, meropenem, netilmicin, piperacillin-tazobactam, ceftazidime, cefepime, tigecycline, trimethoprim-sulfamethoxazole and resistant to aztreonam, colistin, gentamicin, ciprofloxacin and tetracycline [11].

The fifth case was reported by Bayhan et al. in 2013. It was a case of 16-year-old perforated appendicitis with the complaints of acute abdominal pain, vomiting and constipation. The microorganism isolated from the peritoneal fluid was found susceptible to ampicillin, ampicillin-sulbactam, ceftazidime, cefazolin, gentamicin, amikacin, ciprofloxacin, imipenem, piperacillin and resistant to ceftriaxone, cefuroxime and trimethoprim-sulfamethoxazole. It was treated with amikacin, ampicillin and clindamycin [6].

The sixth case was an endocarditis case reported by Duran et al. in 2015. The microorganism isolated from the aortic valve was resistant to piperacillin-tazobactam, imipenem, meropenem, gentamicin and netilmicin and was treated with ciprofloxacin [7]. The seventh case was *C. testosteroni* bacteremia, isolated from the blood cultures of a patient with diabetes and hemiplegia reported by Pekinturk et al. in 2016. The microorganism was found resistant to aztreonam and colistin [12].

The eighth case was the bacteremia due to *C. testosteroni* grown in the blood culture of a patient with neutropenia who presented with high fever reported by Aktar et al. in 2018. Cefixime and ceftazidime resistant microorganism was treated with moxifloxacin [2].

Although *C. testosteroni* can survive for a long time in hospital environments, C. testosteroni infections are community acquired [7]. Gastrointestinal pathologies are often accompanied by intraabdominal infections, and these infections are the most commonly reported group of infections. Risk factors that may cause infections in other cases are the use of central venous catheters, drug injections, skin cuts and subcutaneous lacerations, and surgical procedures [8].

C. testosteroni infections rarely cause death and mostly respond well to antibiotic therapy. In the reported cases, it was susceptible to aminoglycosides, fluoroquinolones, carbapenems, piperecillin-tazobactam, most cephalosporins and trimethoprim-sulfamethoxazole [3,15]. However, it should be kept in mind that antibiotic resistance has increased over the years and the resistance profile may change. C. testosteroni was reported resistant to aminoglycosides in 2009, and resistant to aminoglycosides, carbapenems and piperacillin-tazobactam in 2015 [7,15].

The strain isolated in this case; was found susceptible to ceftazidime (2.0 mg/L), ciprofloxacin (≤ 0.25 mg/L), intermediate susceptible to meropenem (8.0 mg/L), piperacillin / tazobactam (64.0 mg/L), resistant to gentamicin (≥16.0 mg/L), amikacin (≥64.0 mg/L), imipenem (≥16.0 mg/L), trimethoprim- sulfamethaxole (80.0 mg/L). Our patient's urogenital disorder and multiple surgical interventions in this case were predisposing factors for infection. The isolate in our case was found susceptible to ceftazidime and ciprofloxacin, while it was resistant to preparations such as carbapenem and piperacline-tazobactam, suggesting that it was an infectious agent in the hospital environment due to the widespread use of broad-spectrum antibiotics. With this case report, a rare pathogen was drawn to attention; the importance of rational antibiotic use and the contribution of accurate and rapid diagnosis of microorganisms to treatment with new technologies have been emphasized.

Table 1: Comamonas testosterone-induced infection cases in the literature.

No	Age / Gender	Specimens iso- lated	Predisposing factor	Antibiotic therapy	Outcome	References
1	31/F	blood	rheumatic heart disease	kanamycin, tetracycline	recovered	Atkinson et al. (1975) [4]
2	31/M	abdominal abscess drainage fluid	perforated appendicitis	cefoxitn, ampicillin then drainage, gentamicin, clindamisn	recovered	Barbaro et al. (1987) [5]
3	24/F	cerebrospinal fluid	intravenous drug abuse	moxalactam, nafcillin	recovered	Barbaro et al. (1987) [5]
4	59/F	peritoneal fluid	alcoholic cirrhosis	cefoxitin	recovered	Barbaro et al. (1987) [5]
5	11/M	peritoneal fluid	perforated appendicitis	ampicillin, clindamycin, tobramycin	recovered	Barbaro et al. (1987) [5]
6	12/F	peritoneal fluid	perforated appendicitis	cefoxitin	recovered	Barbaro et al. (1987) [5]
7	21/F	peritoneal fluid	perforated appendicitis, pregnancy	cefoxitin	recovered	Barbaro et al. (1987) [5]
8	Stillborn	Cord fluid	maternal intrave- nous drug abuse	could not be cured due to death	dead	Barbaro et al. (1987) [5]
9	84/F	urine	congestive heart failure	ampicillin	recovered	Barbaro et al. (1987) [5]
10	Newborn	blood	maternal intrave- nous drug abuse, prematurity	ampicillin	dead	Barbaro et al. (1987) [5]
11	17/F	peritoneal fluid	appendicitis	no data	recovered	Barbaro et al. (1987) [5]
12	59/M	no data	no data	no data	recovered	Barbaro et al. (1987) [5]
13	66/M	peritoneal fluid	no data	no data	recovered	Barbaro et al. (1987) [5]
14	14/M	appendix	appendicitis	no data	recovered	Barbaro et al. (1987) [5]
15	15/M	peritoneal fluid	no data	no data	recovered	Barbaro et al. (1987) [5]
16	4/M	blood	no data	no data	recovered	Barbaro et al. (1987) [5]
17	28/M	blood	no data	no data	recovered	Barbaro et al. (1987) [5]
18	24/M	peritoneal fluid	perforated appendicitis	cefoxitin	recovered	Barbaro et al. (1987) [5]
19	No data	respiratory secretions	AIDS-related complex	ceftazidime	recovered	Franzetti et al. (1992)
20	35/M	animal bite tissue	zoonotic infection	ceftazidime, gentamicin	recovered	Isolato et al. (2000)
21	75/F	blood, central venous catheter	cancer central venous catheter	ceftazidime, gentamicin	recovered	Le Moal et al. (2001)
22	89/M	blood	advanced age	levofloxacin	recovered	Smith et al. (2003)
23	50/M	cerebrospinal fluid	cholesteatoma	meropenem	recovered	Arda et al. (2003) [3]

24	49/M	Blood, mitral valve	infective endocar- ditis	cefepime, gentamicin then ampicillin and surgery	recovered	Cooper et al. (2005)
25	22/M	blood, peritoneal fluid	perforated appendicitis	cefazolin	recovered	Gul et al. (2007) [8]
26	54/F	blood	chemotherapy, cen- tral venous catheter	cefepime, ciprofloxacin	recovered	Abraham et al. (2007) [1]
27	54/M	cerebrospinal fluid	chronic alcoholic car accident	no data	dead	Jin et al. (2008)
28	82/F	vitreous biopsy	advanced age, diabetes	ceftazidime, ciprofloxacin	recovered	Reddy et al. (2009) [13]
29	83/M	blood	advanced age, ischemic cerebrovascular accident	amikacin, piperacillin / tazobactam	recovered	Katircioglu et al (2010) [9]
30	64/F	blood	hemodialysis	no data	recovered	Nseir et al. (2011)
31	54/M	blood	foot injury	oxacillin, flomoxef then ciprofloxacin	recovered	Tsui et al. (2011) [15]
32	73/M	blood	hepatocellular cancer, chronic hep- atitis B	metasin, gentamicin fol- lowed by levofloxacin	recovered	Tsui et al. (2011) [15]
33	10/M	Endotracheal aspirate	Cerebral palsy, tracheostomy	ceftriaxone, clarithromycin	recovered	Ozden et al. (2011) [11]
34	10/M	blood	medullablastoma, chemotherapy	ciprofloxacin, amikacin	recovered	Farshad et al. (2012)
35	19/F	blood	osteosarcoma	imipenem, vancomycin, ciprofloxacin	recovered	Farshad et al. (2012)
36	16/M	peritoneal fluid	perforated appendicitis	amikacin, ampicillin, clin- damycin	recovered	Bayhan et al. (2013) [6]
37	80/F	blood	diabetes	cefazolin, doripenem	recovered	Orsini et al. (2014) [10]
38	51/M	aortic valve	no	ciprofloxacin	recovered	Duran et al. (2015) [7]
39	42/F	Blood	septic shock	ceftazidime and levoflox- acin	recovered	Who h. et al (2015)
40	62/M	blood	diabetes, ischemic serbrovascular accident	could not be cured due to death	dead	Pekinturk N., Akgunes A. (2016) [12]
41	1/F	blood	acute gastroenteritis, sepsis	ceftriaxone	recovered	Ruziaki W., Hashami H. (2017) [14]
42	65/F	stool	acute gastroenteritis , cholelithiasis	ciprofloxacin	recovered	Farooq S. et al. (2017)
43	30/F	Blood	neutropenia	moxifloxacin	recovered	Aktar et al. (2018) [2]
44	46/F	Blood and urine	sepsis	Gentamicin and imipenem	recovered	Tiwari S et al. (2019)

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Conflict of Interest:

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Ethics Approval

All procedures performed were under the institutional and/or national research committee's ethical standards and the 1964 Helsinki declaration and its later amendments or comparable ethical standards. This report was supervised and approved by the Ankara City Hospital No.1 clinical research Ethics Committee. (reference no:E1/1539/2021)

Contributions

All authors contributed to the development of the manuscript. Literature review and manuscript writing were done by Dr. S.Gayenur Büyükberber. All authors read and approved the final manuscript.

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