Community acquired recurrent urinary tract infection caused by *Pseudomonas aeruginosa* in a public health institute in eastern india: A case report

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ABSTRACT

Urinary tract infection (UTI) is common in childhood with no gender preponderance. Often the diagnosis is missed or cases are neglected, and improperly treated. Common pathogens in community-acquired UTI are Escherichia coli, *K. pneumoniae* and *Proteus* spp.*P. aeruginosa* is quite rare as auropathogen in the community. A case of recurrent UTI was encountered in our Urban Health Centre where an eight-year-old male child had presented with urgency and frequency of urination without fever for the last one year. His urine culture revealed infection with highly drug-resistant P. aeruginosa. A history of contact with a relative who died of urosepsis at home several months back was traced. This is a serious instance of the spread of deadly strains of the bacteria into the society posing a great threat to the community particularly the vulnerable group like children.

Keywords: *Child, Drug-resistant, Pseudomonas aeruginosa, Urinary tract infection.*

Urinary Tract Infection (UTI) is a common infection among children across the globe, and by 6 years of age, 3-4% of girls and 1% of boys will have had one episode of UTI at least [1]. Worldwide, about 150 million people are diagnosed with UTI each year, costing the global economy more than 6 billion US Dollars [2]. UTI is not very common in children though, with common predisposing factors being vesicoureteral reflux, neurogenic bladder, phimosis, anatomical abnormalities of the lower urinary tract, repeated improper antibiotic therapy and constipation [3].

Pseudomonas aeruginosa is a rare cause of UTI in children, more commonly incriminated agents being *E. coli*, *Klebsiella* spp. and *Enterococcus* species [3]. However, it becomes important in cases like structural anomalies of the urinary tract and multiple antibiotic uses. We here report such a case of recurrent community-acquired UTI by *P. aeruginosa*in a child as entailed below in a public health institute.

CASE REPORT

An 8-year-old boy, residing in a nearby slum, accompanied by his father presented at the school health OPD at Urban Health Unit and Training Centre of our institute in Kolkata with chief complaints of the urgency of micturition and burning while urinating. The child and his father were interviewed, and a thorough clinical evaluation of the child and necessary investigations regarding family and socioeconomic status were carried out. On further enquiry, it was revealed that his parents have been healthy all along except minor illnesses like headaches and low-back pain.

The father told us that the boy was doing fine one year ago when he first complained of burning sensation during micturition along with fever. He was treated by local physicians with antibiotics (Ciprofloxacin as recalled by father), Non-steroidal anti-inflammatory drugs (NSAIDs) and plenty of fluids. He was free from symptoms for a brief period of 5-6 days. Treatment was discontinued 2-3 days after initiation, as soon as symptoms subsided. He then developed symptoms like increased frequency and loss of bladder control with lower abdomen pain occasionally. He was again treated by antibiotics (Amoxyclav 250 mg thrice daily) only to be partially relieved from symptoms for a brief period. This time also, treatment was discontinued 2-3 days after the initiation, when symptoms subsided. Such episodes have been recurring repeatedly since the last one year.

On a general examination of the urogenital system of the child by inspection, no abnormality was detected, and flow and stream were observed to be normal. On palpation, prepuce was retractable, both testes were palpable normally. Impulse on cough was absent. There was no tenderness at renal angles.

After all these visits, investigations like urine routine examination and microscopy, ultrasonography (USG), and uroflowmetry were done. All were within normal limits except urine examination. Routine examination of the urine showed the following findings: appearance was straw coloured, acidic, urine protein (albumin) and 1-2 WBCs/hpf was present. Midstream urine culture on Cystine Lactose Electrolyte Deficient (CLED) agar grew pure colonies of *Pseudomonas aeruginosa* (>10^5 CFU/ml). The isolate was susceptible in vitro to Gentamicin and Ciprofloxacin, moderately susceptible to Cotrimoxazole and resistant to Amoxiclav, Ampicillin, Cefepime, Cefuroxime, Tetracycline and Cefotaxime, in susceptibility method done by Kirby-Bauer method on Mueller-Hinton agar [4]. Thus the isolate was highly drug-resistant.

However, in view of the urine report, a more detailed interview of the parents revealed that an aunt of the child came to stay with them for a few months. She had passed away 8 months back. She died at home 10 days after being discharged from the hospital where she had been treated for presumptive Urosepsis with uncontrolled diabetes with other complications including renal failure. She was catheterised for a long duration. Before death, the child's aunt stayed under the same roof at their house for about 6 months and was hospitalised multiple times. Based on this, a diagnosis of hospital-acquired UTI was made. The interview revealed that the parents were completely ignorant about the hospital-acquired infections and its hazards. They were never educated regarding personal hygiene practices like hand washing and disinfection of clothes and bed linens that are essential components of taking care of a sepsis patient at home.

The child was administered Nitrofurantoin 50 mg four times a day for 7 days. Plenty of fluid was advised with maintenance of strict personal hygiene. The child, on follow up, was found clinically cured after receiving treatment for the given period.

DISCUSSION

P. aeruginosa is an emerging cause of community-acquired recurrent UTI, with improper, repeated antibiotic use and underlying anatomical urinary tract abnormality as major predisposing factors [5]. In this case, *P. aeruginosa* caused recurrent community-acquired UTI in the male child. It was probably acquired from the aunt who was suffering from suspected urosepsis. The isolate emerged to be highly drug-resistant, especially to Beta-lactam antibiotics, possibly due to improper antibiotics used and for the inappropriate duration, or maybe it was a nosocomial beta-lactam resistant strain causing UTI and sepsis in the child's aunt and subsequently spreading to the child.

The bacterium is rarely reported from community-acquired UTI in India. There is one report that mentions *P. aeruginosa* causing UTI in Odisha in children, but its occurrence was quite rare among all uropathogens, to the tune of 9% [6]. In a study from Aligarh, India, *P. aeruginosa* isolates were rarely found from the community and were highly resistant to Tetracycline and Cotrimoxazole [7]. In a report from China, only 22 cases of *P. aeruginosa* UTI could be found in a community, and >30% of strains were resistant to penicillins and cephalosporins [8]. The bacterium is more common in cases of catheter-associated UTI in the hospital or in ICU [9]. Here, it caused recurrent UTI since the patient had only intermittent rounds of antibiotics for a few days and personal hygiene was poor.

As far as we know, this is the first case of Nitrofurantoin susceptible *P. aeruginosa* causing community-acquired recurrent UTI in this part of the country. The use of antibiotics either for prophylaxis or for treatment purposes within the preceding 2 months has been shown to be a single independent risk factor for *P. aeruginosa* UTI in children (odds ratio 21.6, p = 0.0001) [10]. In a report from Israel, UTI due to *P. aeruginosa* in the community was found to be more common in boys than girls [11]. Our patient was also a male child.

Usually, *P. aeruginosa* is resistant to Nitrofurantoin, but some reports mention it to be rarely susceptible to the drug [12]. Ours was such an isolate that was susceptible in-vivo to Nitrofurantoin. This is important because Nitrofurantoin is usually ineffective against *P. aeruginosa*.This was also possibly a residual infection because the child had received multiple antibiotics at different points of time and all in suboptimal doses. Further studies are needed in this regard so that more awareness is generated about this pathogen causing UTI in the community.

CONCLUSION

P. aeruginosa is not so uncommon as a causative agent of community-acquired UTI in our country as previously thought and should not be missed. Proper diagnosis by microscopy and culture and the full duration of correct antibiotic helps in complete cure in these cases, especially in children.

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