**Sphingomonas paucimobilis** Urinary Tract Infection in a Renal Transplant Recipient: a Rare Case

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**Abstract**

Infections in immunocompromised hosts warrant special attention. Normally existing as hospital contaminants, *Sphingomonas paucimobilis* can be a rare opportunistic human pathogen. We report one such case by this non-fermenting bacilli causing uncomplicated urinary tract infection in a renal transplant recipient patient with histoplasmosis of colon. Awareness about the possibility of the same is important. Infection control measures of the hospital needs to be stepped up with the isolation of such organisms to prevent cross-transmission and outbreaks.

**Key words:** *Sphingomonas paucimobilis*, urinary tract infection, renal transplant, infection control

**Introduction**

Infections in the immunocompromised host continue to be a special topic of interest with the isolation of uncommon etiological agents. *Sphingomonas paucimobilis* is regarded as a rare opportunistic human pathogen. This organism is widely distributed in the natural environment (especially water and soil). It has been implicated in nosocomial outbreak of bacteremia, catheter-related sepsis, meningitis, peritonitis, cutaneous infections, visceral abscesses, urinary tract infections, adenitis, and endophthalmitis.1-6 It has also been recovered from nebulizers, respirators, dialysis, IV fluids and other medical equipments and has been documented to cause infection in the immunocompromised host.7-9 They are thus known to exist as hospital environmental contaminants. Quinolones or the aminoglycosides (either alone or in combination with a b-lactam agent) are the antibiotics of choice in the treatment of infections caused by this organism. Reports exist with B-lactamase production too and treatment may safely be guided by the antibiotic susceptibility studies of the respective isolates. We report a case of *Sphingomonas paucimobilis* causing uncomplicated urinary tract infection in the renal transplant recipient patient.

**Case Report**

37 yr old man, a post renal transplant recipient three years ago and a case of Histoplasmosis of colon since two years, presented with the 15 day history of fever and burning micturition, mild pain and swelling of right testis. No associated history of Frequency, urgency and dysuria were noted. There was no history suggestive of renal calculus or any anatomical/functional urinary tract abnormality. There was history of persistent watery, non-hemorrhagic diarrhea from two years. Past records of the patient showed him to be a case of extrapulmonary neck node Tuberculosis having completed Category 1 DOTS treatment.* He was also diagnosed with oesophageal and duodenal candidiasis, while on first year of immunosupression. The same year, he had also developed cyclosporine toxicity (recovered) and herpetic abdominal lesions while on second year of immunosuppression for which he was treated. He was a diabetic and hypertensive from over two and half years and was on treatment. On examination, patient was pale, no palpable lymph nodes, non-icteric with normal vitals. Systemic examination of all the organ systems was normal. Normal looking right testis, on palpation was hard, tender with 3x2 cm swelling. Digital rectal examination revealed tender grade I prostate with enlarged seminal vesicle. S.creatinine was 2.5, S. sodium was 122mol/ml with a mildly elevated APTT. Rest of the laboratory parameters were within normal limits. Urine and stool microscopy were
normal. Urine was negative for AFB. Stool culture did not isolate any intestinal pathogen and staining for opportunistic stool pathogens were negative. Repeated urine culture on alternate days were sent, which grew oxidase positive yellow pigmented non-fermenting bacilli with colony forming units of $>10^8$ per ml. The isolate was resistant to the entire first, second and third line of antibiotics tested for routine gram negative bacilli including colistin. Blood culture was sterile for 7 days. Ultrasound scan of the abdomen revealed normal transplanted kidney of size 9x4cm with no hydronephrosis, prostate of 3x3cm with hypoechoic lesion in right lobe. A tentative diagnosis of prostatic abscess with urinary tract infection was made and patient was started on intravenous aztreonam, cefoperazone and oral quinolone in agreement with renal parameters. TURP with biopsy tissue of prostate and epididymis was sent for pathology. Prostatic abscess was drained and fluid sent for culture and sensitivity which was sterile after 72 hours.

* Category 1 DOTS treatment in India: 2 months of INH+ Rifampicin+ Pyrazinamide+ Ethambutol and 4 months of INH+ Rifampicin

**DISCUSSION**

*S. paucimobilis* (formerly called *Pseudomonas paucimobilis*) causing urinary tract infection is a rarely encountered entity, particularly from this part of the world. The other case of UTI reported was in a 40 year old lady with breast cancer.\(^\text{10}\) Our identification matched the description provided by Holmes and colleagues in 1977.\(^\text{11}\) Being catalase and oxidase positive, colonies were small, circular, convex, with entire edge, smooth and yellow. Biochemically, this gram negative non-fermenting bacilli did not grow on McConkey agar, was motile on wet mount and not in mannitol motility media, oxidized OE maltose, sucrose and xylose, hydrolyzed esculin and inert with aminoacids. Identification was
confirmed by API GN (bioMérieux, France). Biochemical identification generally includes differentiation from closely resembling CDC groups O 1,2,3. Also, other yellow pigmented bacteria like Flavobacterium Spp, Elizabethkingella, Sphingobacterium Spp and yellow pigmented Psuedomonas Spp needs to be ruled out.

Sphingomonas can survive in the moist hospital environment. The development of infection in the immunocompromised generally requires an exposure to a contaminated medical device, catheters, implants etc and colonization. The multiple outbreaks. The development of infection in the immunocompromised generally requires an exposure to a contaminated medical device, catheters, implants etc and colonization. The multiple outbreaks. The multiple

We speculate that the history of previous catheterization has resulted in the colonization in the urinary tract. However, we could not trace down to the source or the portal of entry of the bacterium is known to be less virulent and never associated with mortality due to the lack of typical lipopolysaccharide content, our patient too had a favorable outcome. Smalley et al. reported the susceptibility pattern of 20 isolates, all of which were susceptible to ceftriaxone, ceftazidime, amikacin, netilmicin, kanamycin, cotrimoxazole, and tetracycline and resistant to moxalactam, cefotaxime, cefoperazone and piperacillin. A similar sensitivity pattern was noted by Morrison et al. However variability is noted to exist in the antibioticgram and hence treatment should always be guided by respective sensitivity patterns. Our isolate was found to be sensitive only to cefotaxime, cefoperazone and piperacillin. A similar sensitivity pattern was noted by Morrison et al. However variability is noted to exist in the antibioticgram and hence treatment should always be guided by respective sensitivity patterns. Our isolate was found to be sensitive only to cefotaxime, cefoperazone and piperacillin.

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