ORIGINAL RESEARCH PAPER
Volume-6 | Issue-9 | September-2017 | ISSN No 2277 - 8179 | IF : 4.176 | IC Value : 78.46
INTERNATIONAL JOURNAL OF SCIENTIFIC RESEARCH

Fluoroquinolone resistant Salmonella enterica subspecies enterica serotype Typhi causing urinary tract infection in a patient operated for posterior urethral valve

Microbiology

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ABSTRACT

Salmonella urinary tract infections (UTIs) are rare. Salmonella Typhi bacteriuria, an unusual finding, can be seen following a recent episode of typhoid fever, or in chronic carrier state involving the urinary system, especially with local abnormalities of urinary tract. We report a rare case of Salmonella urinary tract infection in a young male patient having bladder dysfunction with vesico-ureteric reflux with history of fulguration for posterior urethral valves in his childhood, not reported so far to the best of our knowledge.

KEYWORDS

Posterior urethral valve, Salmonella Typhi, Urinary tract infections

Key Messages:

Salmonella UTIs are unusual and occur following a recent episode of typhoid fever or in chronic carrier state involving urinary system due to local abnormalities. We report a rare constellation of findings of *Salmonella* Typhi UTI associated with impaired bladder urodynamics with bilateral reflux nephropathy previously operated for posterior urethral valves, previously unreported in literature.

Introduction:

Salmonella UTIs are unusual and occur most often in an individual with predisposing factors like nephrolithiasis, hydronephrosis, anatomic abnormalities, schistosomiasis, tuberculosis and neoplasms of kidney.[1] It has been postulated that *Salmonella* enters the urinary tract either hematogenously or by direct invasion of the bladder via urethra.[2] We report a case of *Salmonella* Typhi UTI associated with impaired bladder urodynamics with bilateral reflux nephropathy previously operated for posterior urethral valves. This rare constellation of findings has not been reported in literature previously to the best of our knowledge.

Case History:

A twenty two years old male, presented to surgical department with chief complaints of intermittent fever, dysuria and generalized weakness for last one and half months. He had undergone posterior urethral valve fulguration in his early childhood in 1995 with history of recurrent episodes of urinary symptoms since then. His urine sample was received in the microbiology laboratory for culture and sensitivity. Grossly, urine sample was turbid. On Gram staining, uncentrifuged urine sample revealed plenty of pus cells with few Gram negative bacilli. Sample was inoculated onto CLED agar (Cystine Lactose Electrolyte Deficient agar) and incubated at 37^0C. On culture, non-lactose fermenting colonies in significant counts ($> 10^5$colony forming units/ml of urine) were grown after overnight incubation. (Fig.1a:CLED showing growth of non lactose fermenting colonies) The isolate was presumptively identified as *Salmonella* Typhi on the basis of motility and biochemical reactions.[3] (Fig.1b:Biochemical reactions of the isolate (right to left: Indole +, Citrate -, Urease -, TSI: K/A with H2S, Hugh Leifson's Oxidation / Fermentation Glucose Medium: Fermentative, Nitrate reduction test +, Phenylalanine Deaminase Test +, Glucose: Fermented with production of acid and without gas in Durham tube, Lactose not fermented, Sucrose not fermented, Maltose fermented, Mannitol fermented.) The isolate was confirmed by serotyping (slide agglutination test).[3] Our isolate was positive for agglutination with *Salmonella* serogroup O9 antisera. (Fig.1c :Slide agglutination test: Positive with Salmonella Antisera Group O9) *Salmonella enterica* subspecies *enterica* serotype Typhi was reported. Antibiotic susceptibility testing was done on Muller Hinton Agar (MHA) plate by Kirby Bauer disc diffusion method as per CLSI guidelines 2017.[4] Our isolate was sensitive to ampicillin, ceftriaxone, cotrimoxazole and nitrofurantoin but resistant to fluoroquinolones (ciprofloxacin and norfloxacin). Abdominal ultrasonography of the patient showed shrunken scarred left kidney with pelvicaliectasis and grossly hydronephrotic right kidney with chronic cystitis and bladder outflow obstruction with significant post voiding residual urine. Dynamic renal scan revealed poorly functioning left kidney with evidence of vesico-ureteric reflux with normally excreting right kidney with dilated non-obstructive hydronephroureterosis. His blood investigations showed that leucocyte count was 10,500/mm^3 with slight lymphocytosis. Renal function tests revealed elevated serum creatinine (1.6 mg/dl) and blood urea (55 mg/dl). Liver function tests were within normal limits. His serum was non-reactive for anti HIV 1 and anti HIV 2 antibodies.VDRL test was non-reactive. Blood culture using BacT-Alert system revealed no growth after five days of incubation. Stool culture showed growth of lactose fermenting commensal flora.

Discussion:

Congenital mucosal membrane in the prostatic urethra is called posterior urethral valves (PUV).[5] Posterior urethral valves are the commonest cause of lower urinary tract outflow obstruction in male infants with an estimated incidence of 1:5000 male infants and 1:25,000 live births.[6] Cystoscopic valve ablation or fulguration is the definitive treatment with temporary vesicotomy or urinary diversion done for interim relief in very young patients.[7] Long-term complications include impaired bladder urodynamics in majority of cases of treated PUV with incontinence and vesicoureteric reflux leading to impaired renal function and predisposition to to urinary infections.[8]

Salmonellae are facultative anaerobic, non-spore-forming, Gram-negative bacilli. *Salmonella* have been classified into 2541 serotype by the Kaufmann-White scheme based on the O and H antigens.[9,10] The laboratories perform a few simple agglutination reactions that define *Salmonella* somatic O antigens into serogroups, designated as groups A, B, C1, C2, D and E *Salmonella*. *S*. Enteritidis and *S*. Typhi are both group D.[2,3,10]

Our patient had *Salmonella* Typhi UTI associated with impaired bladder urodynamics with right gross hydronephroureterosis and shrunken left kidney with pelvicalicectasis likely due to secondary

vesico-ureteric reflux. The absence of growth of *Salmonella* on stool culture and blood culture rules out possible fecal contamination and hematogenous source of infection with primary urinary infection.

Figure 1. (Original)

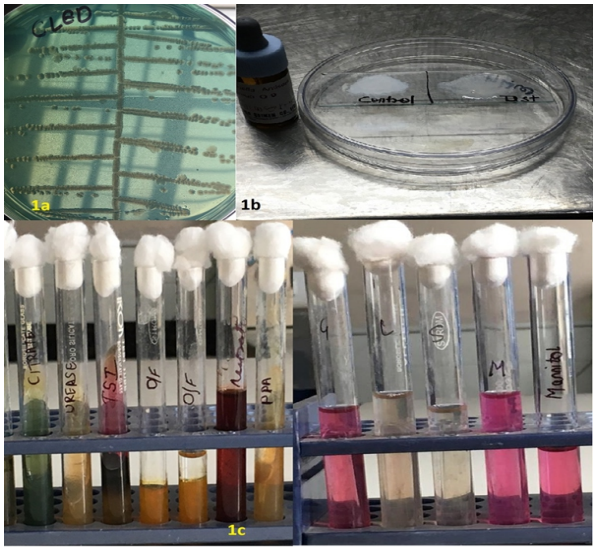

Figure1a. CLED showing growth of non lactose fermenting colonies. Figure1b. Biochemical reactions of the isolate (right to left: Indole +, Citrate -, Urease -, TSI: K/A with H2S, Hugh Leifson's Oxidation / Fermentation Glucose Medium: Fermentative, Nitrate reduction test +, Phenylalanine Deaminase Test +, Glucose: Fermented with production of acid and without gas in Durham tube, Lactose not fermented, Sucrose not fermented, Maltose fermented, Mannitol fermented. Figure 1c. Slide agglutination test: Positive with Salmonella Antisera Group O9.

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