Case Report

Isolation of *Salmonella paratyphi A* from a patient with nephrolithiases

Fawzia E. Al-Otaibi, *MBBS, KSFPath (Micro).*

ABSTRACT

We describe a case of *Salmonella paratyphi A* isolated from urine of a 37-year-old Saudi patient who is a known case of nephrolithiasis and hydronephrosis with frequent admission for management of renal stones. History of enteric fever was not documented and urinary schistosomiasis in such a patient from endemic area is a strong possibility. Relevant literature was discussed.

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Salmonella urinary tract infections (UTIs) are uncommon.[1] Fifty-four cases of culture-proven symptomatic *Salmonella* UTIs during the antibiotic era have been reported. The mean age was 41 years and a male predominance was observed. Most of the infections were located in the upper urinary tract (infection is defined as culture – positive urine obtained from ureters or kidneys). Nearly all patients were immunocompromised (having malignancies or were transplant recipients), had pre-existing pathological changes such as nephrolithiasis,[2,3] hydronephrosis, and anatomical anomaly. In endemic areas such as Egypt, urinary schistosomiasis is an important predisposing factor for *Salmonella* infection, as the organisms establish themselves in the damaged tissues and produce chronic infection. Urolithiasis was present in 19% patients.[2,3] The presence of stones, which may harbor organisms often, leads to multiple relapses or to the development of chronic urinary carrier. *Salmonella typhimurium* was the most commonly isolated serotype (37% of patients) specifically in renal transplant recipients.[1] *Salmonella typhi (S.typh*i) was also frequently cultured from patients most of whom were chronic urinary carriers with underlying abnormalities such as nephrolithiasis, renal tuberculosis or schistosomiasis.[4] Other reported species includes *Salmonella choleraesuis, Salmonella group B, Salmonella enteriditis* and *Salmonella serby*.[3,4] Although in a study on UTI at a specialist hospital in the Kingdom of Saudi Arabia (KSA), *Salmonella paratyphi A* (*S. paratyphi A*) comprised 1/854 (0.1% of urinary isolates),[5] to the best of our knowledge no specific case of *S. paratyphi A* UTI was reported from the Gulf States in general and KSA in particular.

Case Report. A 37-year-old Saudi man was presented with bilateral intermittent loin pain over the last 3 months. He was seen and investigated in Najran, KSA, for the same complain and a diagnosis of left renal stones with hydronephrosis was made for which he had received shock wave therapy. He was then referred to the King Khalid University Hospital, Riyadh, KSA for further management. His past medical report was not complete and there was no past history suggestive of typhoid or paratyphoid fever. On physical examination the patient was not febrile and had normal vital signs. He was pale but not jaundiced. Abdominal examination

From the Department of Pathology/Microbiology, King Khalid University Hospital, Riyadh, *Kingdom of Saudi Arabia*.

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Address correspondence and reprint request to: Dr. Fawzia Eida Al-Otaibi, Department of Pathology/Microbiology (32) College of Medicine, King Khalid University Hospital, PO Box 2925, Riyadh 11461, *Kingdom of Saudi Arabia*. Tel. +966 (1) 4671088. Fax. +966 (1) 4672462.

revealed bilateral flank tenderness. No masses were felt. Laboratory tests revealed anemia (hemoglobin concentration = 11.9 gm/dL), leukocyte count of 9.2 x 109/L with 50% lymphocyte and 8.2% eosinophil, serum urea, creatinine and electrolytes were normal. Urine microscopy showed pyuria, hematuria, and microscopic ex. of the urinary sediment for schistosoma ova was negative. Initial urine culture showed mixed growth of coliforms including non lactose fermenter and skin organisms. But a repeated specimen showed a pure growth (>105 CFU) of *S. paratyphi A.* The organism was identified by characteristic colonial morphology (non lactose fermenter), negative oxidase reaction and biochemical test by Appareils et Procedes d' Identification (API) (Bio, Merieux SA, France). Serological identification was positive for both somatic O antigen (group O-2) and flagella antigens (type H-a). The organism was sensitive to ampicillin (Amp), cotrimoxazole (Cot), ceftriaxone (CRO), cefuroxime (CXM), ciprofloxacin (Cipro) and chloramphenicol (CHL) as determined by disc diffusion using a rotating stoke's technique.[6] Minimum inhibitory concentrations were also determined for the isolate by E test (AB Biodisk, Solona, Sweden) against Amp (3 µg/ml), CRO (0.19 µg/ml), CHL (4 µg/ml), Cipro (0.047 µg/ml) and Cot. (0.064 µg/ml). Although important, stool and blood culture as well as serology were not carried out initially as there was no indication, the patient was not systemically ill and does not have any gastrointestinal symptoms. The patient has a short stay and he did not return to the hospital for follow-up. Plain film showed amorphous (staghorn type) stones in the upper and mid pole of the left kidney along with small multiple stones **Figure 1.** An excretory urogram **Figure 2a and 2b** revealed bilateral calcified hydronephrosis with mild loss of renal cortex in the left kidney and right hydroureter. Visualization of both ureters was delayed. The left kidney was confirmed to be non functioning on isotope dimercapto succinic acid (DMSA) scan (function of 33%). The patient was taken to operating room for cystoscopy and retrograde study of the right kidney. Cystoscopy revealed a normal anterior but narrow bulbar urethra and yellowish sandy patches at bladder neck and trigon, probably from old urinary schistosomiasis. The right ureter was narrow, but no stricture. Balloon dilation was carried out and double J (DJ) stent was inserted. The patient was discharged home on Cefuroxime tablet 250 mg twice a day for 7 days and one month later he underwent elective removal of right DJ stent and bilateral retrograde study with insertion of left DJ stent and shock wave therapy of left renal stones. After the repeated urine culture confirmed the presence of S. paratyphi A, the patient was started on augmentin 625 mg per os 3 times a day for 10 days.

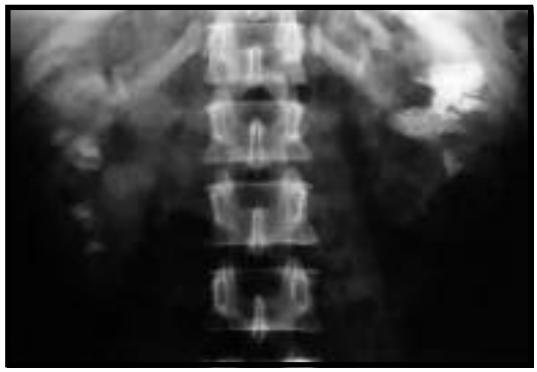
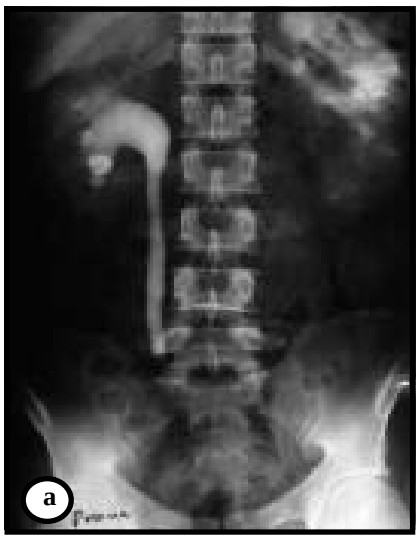


Figure 1 - Plain film showing multiple amorphous radiopaque densities overlaying the left kidney and the largest stone located in mid and upper portions.



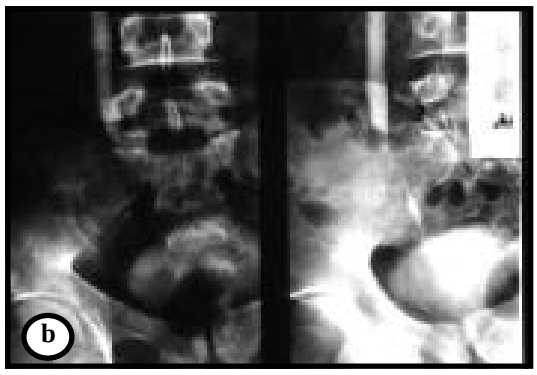


Figure 2 - Plain film showing **a)** 15 minutes post-contrast image of bilateral calcified hydronephrosis with mild loss of renal cortex in the left kidney and **b)** post-contrast one hour delayed of both ureters with hydroureter in the right side up to the inferior margin of right transverse process of lumbar segment of the vertebral column number 5 (L5).

Discussion. *Salmonella* organisms are subdivided into different groups from A to Z on the basis of their somatic O and flagellar H antigens. Some serotypes of group A to D are associated with typhoid and

paratyphoid fevers. *Salmonella bacteriuria* is rare even where the organisms are endemic.[7,8] In a review of 360 cases of typhoid fever from the United States of America in the pre-antibiotic era, 81 patients (23%) had *S. typhi* cultured from the urine; non of the these patients became chronic urinary carriers.[9] However, UTI occurring as late as 52 years after the original typhoid infection has been reported.[10] In this report, a 72-year-old woman develop repeated attacks of UTI with mixed bacterial growth of coliform and other organisms. Pure culture of coliform was then identified as *Salmonella sp.* Later this *Salmonella* was identified as *S. paratyphi B,* which was traced to 1946. This infection has contributed to the formation of a stag horn calculus in her right kidney, chronic inflammation, and squamous-cell carcinoma and ultimately died. Urinary tract manifestations in *Salmonella* infections are uncommon and they occur in 5 clinical forms, transient bacilluria (following a recent typhoid fever as part of the natural history of the disease or in chronic carrier status), perinephric abscess, pyelonephritis, nephrolithiasis and cystitis. Is nephrolithiasis the cause or the result of Salmonella infection of the urinary tract opinions vary. In our case, it is difficult to deny the existence of previous stones or to pinpoint the date of *Salmonella* infection. Multiplicity of stones and presence of bladder pathology suggestive of old urinary bilharziasis would favor their development secondary to infection in an already damaged kidney. Meizer et al[4] reported on 6 patients with clinically significant UTI associated with nephrolithiasis. We would agreed that the pre-existence of stones, deformities or local damage predisposed to the development of chronic *Salmonella* infection of the kidney. The vital clinical point is that in patients with *Salmonella* carrier state bacilluria will not be permanently eliminated by treatment with antibiotics unless stones are removed, obstruction is corrected and diseased renal tissue is resected.

We conclude that in patient who are from endemic areas for schistosomiasis, with renal stones, the isolation of a non-lactose fermenter even in mixed culture should raise the suspicion of a *Salmonella* organism. If *Salmonella* UTI is confirmed, full investigation for evidence of systemic infection by *Salmonella* is advised. The investigation should include serology for *Salmonella* antibodies, and the possibility of carrier state.

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