JOURNAL OF CASE REPORTS 2015;5(1):75-78

Carrier status or Uro-pathogenic Salmonella Paratyphi – A: Urinary Isolation of Variant Salmonella Paratyphi- A in a case of Catastrophic Systemic Lupus Erythematosus

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Abstract:

We communicate this simple but informative report which describes an isolation of a rare variant of *Salmonella paratyphi* A from a known case of SLE in urine causing clinical and microbiological confusion in diagnosis. Urinary isolation of *Salmonella* often is unanticipated and a high index of suspicion is required for adequate therapy. Awareness of association between *Salmonella spp*. and SLE and the timely use of reference laboratories in India is highlighted.

Key words: Salmonella paratyphi A, Urinary Tract Infections, Salmonella, Edema, Jaundice, Humans.

Introduction

Salmonella urinary tract infections (UTIs) are uncommon and are limited to case reports even in areas endemic for this infection [1-3]. Much of these reports are decades older. *S. typhi* and still to a lesser extent *S. paratyphi* can be isolated from urine following a recent episode of typhoid fever, in chronic carrier states involving the urinary system, and also following localized urinary tract infection occasionally [4]. Acute symptomatic UTI is regarded not a recognized manifestation of *S. typhi* infection. They are infrequently seen in immunocompromised (malignancies or transplant recipients), pre-existing pathological changes such as nephrolithiasis, hydronephrosis or anatomical anomaly [5,6]. The presence of stones may harbor microorganisms leading to multiple relapses or to the development of chronic urinary carrier status. Systemic lupus erythematosus (SLE) patients, in particular on immunosuppressive therapy, are known to be associated with *Salmonella* infections [10]. We report a rare case of catastrophic SLE with a variant *Salmonella paratyphi* A urinary isolation, a rare case from South India which caused diagnostic confusion both clinically and microbiologically.

Case Report

A 35 year old lady, case of systemic lupus erythematosus (SLE) on corticosteroids since last one and half year, was admitted with fever, burning micturition from 2 weeks with one episode of vomiting. Fever was intermittent, high grade, no

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Received: October 2, 2014 | **Accepted:** January 24, 2015 | **Published Online:** February 20, 2015

Conflict of interest: None declared | **Source of funding:** Nil | **DOI:** http://dx.doi.org/10.17659/01.2015.0019

diurnal variation, not associated with chills, lower abdomen, back pain or hematuria and loose stools. On examination, she was poorly nourished lady with pallor, icterus and mild pitting pedal edema. There were palpable lymph nodes in the neck with oral candidiasis. She had tachycardia, BP 120/80 mmHg; temperature of 102^{0}F. Liver was palpable 15 cm below the right coastal margin, firm and tender. No splenomegaly or evidence of free fluid or tenderness in the abdomen.

Investigations revealed anaemia of chronic disease (Hb 8.5 gm%, hematocrit 25%). WBC 3600/mm^3 with 91% neutrophils with toxic changes and left shift, platelet count of 1 lakh, elevated ESR and CRP (>100 mg/L). Renal parameters were normal. Liver enzymes were elevated (AST 242 mg/dL, ALT 64 mg/dL, Alkaline phosphatase 1182 IU/mL, GGT 1335, serum bilirubin 9 mg/dL). Ultrasound abdomen showed hepatomegaly with fatty infiltration. Endoscopic ultrasound done revealed multiple upper abdominal lymph nodes without features of secondary malignancy, or evidence of pancreatic biliary disease. Granulomatous disease (tuberculosis/fungal) was thence suspected and transjugular liver biopsy was scheduled with viral markers and ANA testing. HCV and HBsAg status were negative. CT abdomen showed bulky pancreas, hepatomegaly with fatty infiltration, prominent portal vein, diffuse gall bladder wall thickening with pericholecystic fluid collection and minimal ascitis. CT findings suggested ruling out infiltration of liver and pancreas. Urinary system on ultrasound and computed tomography (CT) abdomen appeared normal. She was empirically started on cefaporazone-sulbactum with the presumptive diagnosis of UTI. Fever kept spiking every alternate day despite the antibiotic therapy. The agglutination tests for *Brucella spp*. and WIDAL were negative. Blood and bone marrow culture were sterile throughout. Urine microscopy showed proteinuria (+++), numerous WBCs, 30-35 RBCs per HPF with granular casts. Two of urine samples in an interval of 5 days sent for culture grew *Salmonella spp*. > 10^5 CFU/mL. The non-lactose fermenting colonies produced abundant H_2S and were biochemically not showing the typical reactions of any particular *Salmonella*. Automated identification, agglutination test results for serotyping were also inconclusive. Serotype was confirmed by the reference laboratory as *S. paratyphi A*- an atypical variant. Isolates on both occasions were sensitive to ampicillin, ceftriaxone, cotrimoxazole, norfloxacin, and chloramphenicol. Patient was started on ceftriaxone, received her first pulse therapy of methylprednisolone and cyclophosphamide. Regarded as a case of catastrophic SLE in severe hemolysis and positive Coomb's test with anemia of chronic disease, she also received 3 units packed cells and 2 units of whole blood transfusion. She slowly became afebrile on day 10, liver size decreased and general condition improved. She later completed five of her cycles of cyclophosphamide therapy uneventfully. A repeat urine culture after the completion of antibiotic therapy was sterile.

Discussion

Salmonella bacteriuria is rare and occurs in less than 1% cases of enteric fever; particularly in the endemic areas. The patient had no records of previous history of enteric fever as urinary shedding may occur months after infection in a carrier. Prolonged shedding is seen encountered with pathological or anatomical abnormalities of the urinary tract like the renal calculi and she had none. Although enteric fever was considered and blood culture and WIDAL were sought (which can be non-contributory to diagnosis if not rightly timed), isolation in urine was not anticipated.

Isolation of *Salmonella* in urine with findings of leukocyturia, proteinuria and >10^5 colony forming units/mL in the patient suggested more in favor of a local infection, rather than pure excretion

[7]. Biochemically, a typical *Salmonella paratyphi* A does not routinely produce H_2S and variants such as this isolate producing H_2S can be easily misidentified as *Citrobacter spp*. or other *Salmonella spp*. (all are non-lactose fermenters) leading on to inadequate antibiotic therapy. Many times, on-spot serotyping/ agglutination tests which are routinely done for identification remain inconclusive and the strain needs confirmation from the reference laboratories as was done for this patient. Microbiologists need to be aware of this before recommending the treatment. In certain parts of the world like Egypt, *Salmonella* UTI is known to be associated with schistosomiasis, which of course is not encountered in India. Also, literature suggests that in the absence of schistosomiasis, there is almost a remote possibility of patient becoming a *S. paratyphi A* carrier [8].

Lehman JS Jr *et al*. quote bacterial infection of the urinary tract led to decreased renal function [9] but patient's renal functions remained normal throughout. Fever and leucocyturia can also be present without an infectious etiology in SLE patients. The patient symptomatically got better with ceftriaxone, but she soon landed in catastrophic state because of the disease activity of SLE and we believe the isolate did not have a contributory role in this event. Sharahm *et al*. argued that the duration of antibiotic therapy should be extended for longer time as they noted a recurrence rate of 29% primarily from blood isolates of non-typhoidal *salmonella* in SLE patients [10].

This is one of the rare cases from South India of H_2S producing *Salmonella* paratyphi A plausibly attributed to be causing UTI. Being the only single isolate, it was noted that this pathogen did not suppress the growth or over-grow any other uro-pathogen on culture plates. Awareness of this possibility with associations like SLE is important. Complete work up with investigations like ultrasound, intravenous pyelogram, CT abdomen may be required to rule out the co-existing anomalies.

Conclusion

This report highlights that high index of suspicion is required to identify the non-lactose fermenting *Salmonella* colonies on urine culture plates and to pursue the biochemical identification of atypical isolates upto the serotype which would aid in the administration of appropriate antibiotic therapy. *Salmonella* reporting and serotyping must be made mandatory by regulation in India.

Acknowledgements

The authors thank National Salmonella Reference center, Kasauli, India for confirming the strain and Mrs. Manjula, senior technician, KMC, Manipal for maintaining the strain.

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