Asymptomatic Nontyphoidal *Salmonella* Bacteriuria in a Patient with SLE Nephritis: An Ominous Threat or Innocent Bystander?

Sir,

Nontyphoidal salmonellosis is a common infection in systemic lupus erythematosus (SLE).[1] Nontyphoid *Salmonella* (NTS) infection in immunocompetent individuals mostly presents as acute self-limited gastroenteritis, whereas NTS infections present with various extraintestinal manifestations and sepsis rather than gastroenteritis in SLE.[1] The routine use of antibiotics in the treatment of uncomplicated NTS gastroenteritis is not recommended as it leads to prolonged stool carriage and an increased incidence of clinical relapse;[1] however, sparse data exist on treating asymptomatic NTS bacteriuria to prevent seeding of other sites.

A 14-year-old girl, a diagnosed case of SLE with class IV nephritis, presented to our hospital with chief complaints of decrease in urine output, puffiness of face, itching all over the body, easy fatiguability, and rashes over the legs. She had history of three previous blood transfusions since her diagnosis 1 year back. There was no history of urinary complaints, such as dysuria, urinary frequency, urgency, suprapubic pain or tenderness, and costovertebral tenderness. She had been immunized as per schedule till date. She was on prednisolone, mycophenolate mofetil (MMF), calcium, pantoprazole, and enalapril for the past 1 year.

On examination, the patient was afebrile and hemodynamically stable. General, physical, and systemic examinations were unremarkable except for the presence of pallor and blood pressure of 125/92 mm Hg. Laboratory investigations revealed hemoglobin of 9.7 g/dL, a total leukocyte count 18.81 × 10^3/mm^3, and absolute neutrophil count 15,600/mm^3. Renal function tests were normal with blood urea of 34 mg/dL and serum creatinine 0.8 mg/dL. Urine analysis revealed proteinuria (2+), presence of one to two pus cells/HPF, and one to two epithelial cells/HPF. Urine culture grew nonlactose fermenting translucent colonies on CLED agar having colony count greater than 10^5 colony-forming units per milliliter. The isolate was identified as *Salmonella* group by conventional biochemical reactions and by Vitek 2 (bioMerieux, Hazelwood, MO, France). On serotyping, agglutination was observed with Poly O, Phase 1 and 2 of H antigen, and O_4, identified as *Salmonella* Group D. The isolate was sensitive to ciprofloxacin, levofloxacin, ceftriaxone, and cefixime. Paired blood cultures were sterile and stool cultures did not yield growth of NTS. The patient was treated initially with injection ceftriaxone followed by oral cefixime for 7 days. The patient had improved clinically and was discharged on MMF, low-dose steroid (5 mg/day), enalapril, and hydroxychloroquine. Clinical and microbiological follow-up has continued till date. She has disease activity under control till last follow-up.

There are three possible suggested hypotheses about the association of NTS and SLE: (1) NTS infections trigger SLE, (2) SLE (or its treatment) predisposes to NTS, (3) similar underlying immunological defect predisposes to both lupus and NTS infections.[1] The frequent occurrence of

NTS infections during active SLE or periods of intensified immunosuppression support the second hypothesis. In our case, the patient was on immunosuppressive medication since 1 year and hence had a risk factor for NTS. The modes of NTS urinary tract infection (UTI) include hematogenous spread from gastroenteritis or contamination from fecal flora through direct urethral invasion, which is more common in women. In our case, the patient did not have complaints of gastroenteritis and stool culture was also negative for NTS. High frequency of complications due to NTS UTI in patients with SLE such as pyelonephritis, renal insufficiency, nephrotic syndrome, nephrolithiasis, genitourinary abscesses, recurrence, and chronic bacteriuria warrants prolonged treatment. Prolonged courses of antibiotics are indicated to prevent chronic bacteriuria, relapse, or bacteremia, especially in patients with immunosuppression. In the largest series of NTS infections in patients with SLE by Lim *et al*., serogroup B was the most common (28/50), followed by serogroup D (22/50).[1] All episodes were treated with appropriate antibiotics for an average initial course of 3 weeks. There are other reports of symptomatic NTS UTI in SLE and treatment recommendations. However, there is no established guideline on treating asymptomatic NTS bacteriuria to prevent seeding of other sites.

Patients with active SLE (especially nephritis) on intensified immunosuppression are most at risk for NTS infections.[2,3] In view of this association, there is a need for heightened microbiological surveillance with appropriate cultures to detect colonization. The risk of subsequent symptomatic UTI and associated complications after antibiotics are discontinued demonstrates the importance of careful follow-up with repeat urine cultures. Adequate follow-up is necessary to decide the optimal duration of treatment in colonized patients.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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