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Case Report

Salmonella Paratyphi B bacteremia in Systemic Lupus Erythematosus with an unusual presentation- a case report

Madhuri Kulkarni[1*], Rajeshwari K.G[1], Tejashree A[1], Subramanian R[2]

[1]Department of Microbiology, JSS Medical College, JSS University Mysore, India
[2]Department of Rheumatology, JSS Medical College, JSS University Mysore, India

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ABSTRACT

Background- Systemic Lupus Erythematosus (SLE) is an inflammatory and multisystem autoimmune disorder. Patients of SLE are at increased risk of infections owing to underlying immunological derangements and to the use of therapeutic regimens like immunosuppressive agents. Among the bacterial infections presenting as bacteremia in these patients, non typhoidal and typhoidal salmonellosis are commonly encountered. We report a rare case of Salmonella Paratyphi B bacteremia in a patient with juvenile onset SLE on treatment with corticosteroids.

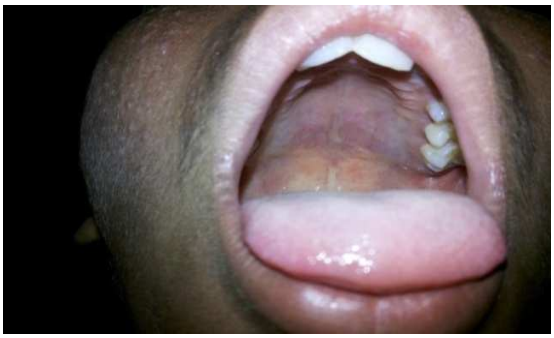
Introduction

Systemic Lupus Erythematosus (SLE) is an inflammatory and multisystem autoimmune disorder. Patients of SLE are at increased risk of infections owing to underlying immunological derangements and to the use of therapeutic regimens like immunosuppressive agents[1]. Among the bacterial infections presenting as bacteremia in these patients, non typhoidal and typhoidal salmonellosis are commonly encountered[2].

Case Report

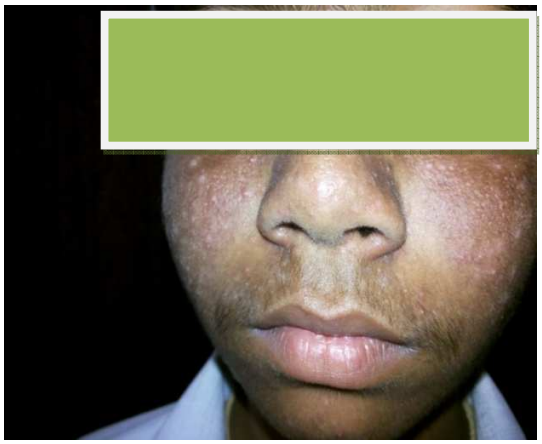
A 16 year old male patient presented to the Rheumatology OPD of JSS Medical College, Mysore with complaints of intermittent fever and headache for 2 weeks .The fever was of high grade, associated with chills. Patient was a known case of Systemic Lupus Erythematosus (SLE) on treatment with steroids for 1month. SLE was diagnosed on the basis of malar rash, oral ulcer, fever and serological markers.

Clinical examination revealed a febrile (102^0 F) toxic patient, with an oral ulcer over the soft palate (Fig a) together with a facial butterfly rash characteristic of SLE (Fig b). Pulse rate was 106/mt, BP 100/70mm Hg. There was mild hepatosplenomegaly.Other systemic examination did not reveal any significant findings.



a. Oral Ulcer over the soft palate

[*]**Corresponding Author: Dr. Madhuri Kulkarni,** Professor & Head, Department of Microbiology, JSS Medical College, JSS University, Mysore, India. **E-Mail:** mkmysore@gmail.com



b. Malar Rash of SLE

Hemoglobin was 12.3 gm/dl with normocytic normochromic blood picture. Total count was 9900 cells per cu mm. Differential count revealed neutrophillia (84%). ESR was 110mm/hr, platelets 2.75 lakhs/cumm. Antinuclear antibodies detected by immunofluorescence showed homogeneous pattern with dividing cell chromatin positive. Anti ds DNA negative by ELISA. ALT was 239 IU/ml, AST 138 IU/ml, Blood urea 52mg/dl, creatinine 1.2mg/dl, Sodium 137mmol/ltr, potassium 4.5mmol/ltr, chloride 98mmol/ltr. Urine microscopy showed 2 + albuminuria with 10 to 12 pus cells and culture was sterile. USG abdomen showed mild ascites with hepatosplenomegaly along with moderate hydrocele on the left side.

Using Bactec automated system and Vitek 2 automated bacterial identification and antimicrobial susceptibility testing system, Salmonella Paratyphi B (L+, Tartrate negative) was isolated on two consecutive blood cultures. The phenotypic identification was further confirmed by serotyping with 0-4 & H-I antisera The isolate was sensitive to chloramphenicol, Ciprofloxacin, Gentamycin, Ceftriaxone, Nalidixic acid and Tetracycline.

Patient was treated with Ceftriaxone and the symptoms resolved in two days. On the third day of stay in the hospital, patient developed ascites and left sided massive epididymoorchitis and hydrocele.

Follow up:

With the completion of 10 days of parenteral antibiotics, the patient's fever had subsided and the left sided hydrocele had subsided. He was put on tapering doses of steroids and he was not on any second line agent. There was no recurrence of albuminuria and his 24 hour urine protein was always less than 500 mg /24 hrs.

Discussion

Infections loom like the sword of Democles, over patients with SLE[3]. About half the patients with SLE will suffer a major infection in their lives and a great proportion of them have an infection attributable death. Inspite of this, only a few studies have addressed the issues that would provide clinicians with better management alternatives for infection in SLE and its prevention[4]. The use of high dose corticosteroids, high disease activity, organ dysfunction and use of immunosupressants are the strongest risk factors for the development of infections in SLE . Fever, among other findings, challenges the clinician into a discriminative endeavor to establish its relation with disease activity and /or infection[5]. Intercurrent infections may mimic flare up and the differential diagnosis becomes difficult. SLE patients are more prone to develop common (pneumonia, urinary tract infection, cellulitis, sepsis), chronic (tuberculosis), and opportunistic infections possibly due to inherited genetic and immunologic defects (complement deficiencies, mannose-binding lectin [MBL] polymorphisms, elevated Fc gamma III and GM-CSF levels, osteopontion polymorphisms[1].

About 80% of SLE infections are caused by bacteria[6].Non typhoidal salmonellosis is the main cause of Gram negative bacteremia in patients who have SLE[7]. Salmonella infections can either manifest as gastroenteritis, bacteremia, osteomyelitis and meningitis. In a case series by Shahram *et al*[2], recurrence of infections and mortality rates were upto to 29 % due to salmonella infections despite appropriate antibiotic therapies. Some experts have argued for longer duration of antimicrobial therapy in patients of SLE with salmonellosis.

Salmonellosis continues to be a major public health problem, especially in developing countries. The formation of local abscesses may occur following either hematogenous or lymphatic spread.

Recurrence of infection in 29% of the patients and the high mortality rate(28.5%) despite the conventional period of antimicrobial treatment, show a poor prognosis of Salmonellosis in SLE patients. This special picture of Salmonellosis requires a much longer period of treatment in these patients.

There are a large number of serious and life threatening clinical manifestations of Salmonellae species ranging from osteomyelitis to infective endocarditis and meningitis. However Salmonella epididimoorchitis is a rare clinical manifestation reported most often in male babies and adolescent boys.

A few case reports of Salmonella species being isolated from the testicular abscesses are available. [8]

The cause for epididymo-orchitis and hydrocele in this patient is uncertain. Peritoneal serositis is a rarely reported aspect of SLE, occurring when complicated by nephrotic syndrome, CCF or cirrhosis.Salmonella Typhi is endemic in India and S.Paratyphi A is endemic and is showing an increasing trend over the last few years but S.Paratyphi B infection is rare.

Conflict of interest statement

We declare that we have no conflict of interest.

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Conclusion

The immunocompromised state of the patients of SLE predisposes them to blood stream infections, sometimes with rare agents as in our case. Vigilance on the part of the attending physician will warrant early diagnosis, prompt treatment and thus curtail the morbidity.

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