Case Report

TYPHOID FEVER COMPLICATED BY LEUKOCYTOCLASTIC VASCULITIS

JAMSHID AYATOLLAHI*

SUMMARY: We report a case of typhoid fever with an unusual presentation: prolonged fever with painful purpuric skin lesions of the legs, splenic abscess and pancreatitis. The diagnosis was made upon isolation of S. typhi in blood cultures, and ruling out other causes of leukocytoclastic vasculitis. The outcome was favorable with antibiotics alone without surgery.

Key Words: Typhoid fever, leukocytoclastic vasculitis

INTRODUCTION

Typhoid fever is a systemic disease characterized by fever and abdominal pain caused by dissemination of *S. typhi* (1). Rare complications whose incidences are reduced by prompt antibiotic treatment include pancreatitis, hepatic and splenic abscesses, endocarditis, pericarditis, orchitis, hepatitis, meningitis, nephritis, myocarditis, pneumonia, arthritis, osteomyelitis, and parotitis (2-4). To our knowledge, we report the first case of typhoid fever with cutaneous leukocytoclastic vasculitis.

Case report

A 31-year-old man was admitted with the 3-week history of fever and a 2-day history of painful purpuric skin lesions of the legs.

Previously in good health, he had traveled through Iraq for 2 weeks, and had come back to Iran in the first day of April 2004. Almost immediately after coming home he complained of fever, abdominal pain, headache and mild diarrhea. Diarrhea had spontaneously resolved within a few days but fever persisted. Two days before admission, painful skin lesions appeared on his legs. He had not taken any drugs. On physical examination, his weight was 63 kg for 1.78 m, with a 9 kg weight loss. His temperature was 39°C, pulse 120, and blood pressure 120/90 mmHg. Skin examination showed purpuric infiltrated spots limited to the legs, with non-pitting edema and painful calves. He was well oriented and there were no other clinical abnormalities.

The CBC showed WBC count per mm^3 9700 (neutrophils 7400 despite high fever), platelet count 220000, and hemoglobin 10.2 g/dl. The sedimentation rate was 100 mm at the first hour. Serum urea and creatinin and urinary sediment analysis were normal. Amylasemia was 270 U/L (N<120 U/L), lipasemia 500 U/L (N<80 U/L), AST, ALT and LDH were slightly increased and bilirubin and other laboratory values were normal. Tests for antinuclear antibodies, ANCA, cryoglobulinemia and circulating immune complexes were negative. CH50 and C3 complement fractions were slightly increased. The first sets of blood cultures were negative. Serological tests for HBV, HCV, HIV, wright, coombs wright and widal were negative.

Chest radiograph, echocardiography and electrocardiogram were normal. An abdominal ultrasonography showed a liquid splenic lesion, and a CT scan revealed a splenomegaly with an abscess of 15 mm in diameter, whereas examination of pancreas, liver and biliary ducts showed no abnormalities. The main histopathological finding of the skin biopsy was a leukocytoclastic vasculitis with superficial and deep perivascular infiltrate of lymphocytes and neutrophils.

*From Infectious and Tropical Diseases Research Center, Shahid Sadoughi Hospital, Safaeieh, Yazd, Iran.

At rest, the patient became apyretic and the skin lesions disappeared within 48 hours. He remained asymptomatic for the following 14 days. AST, ALT and LDH levels normalized and pancreatitis improved. Because of the general improvement, splenic surgery was postponed. On the 15th day the fever recurred: 39°C, then 40.5°C, on the 16th day. Two blood cultures from the 15th day grew with drug- sensitive *S. typhi*. The patient was treated with ceftriaxone 2g intravenously for 7 days, then ciprofloxacin 500 mg twice a day taken orally for 7 days.

Apyrexia was obtained in 96 hours and the evolution was unremarkable, with normalization of pathological changes and disappearance of the splenic abscess assessed with a second CT scan. Serological controls, performed 2 weeks and 1 month after the first ones, remained negative, showing only minor dilution increase for widal test (1/160).

DISCUSSION

Skin lesions in salmonella infections are rose spots (1,4), pustular dermatitis, purpura, and petechiae (2). Purpura or skin petechiae in *Salmonella* infection is rare, and always described in the setting of endocarditis (4). We report a cutaneous leukocytoclastic vasculitis associated with a typhoid fever without endocarditis. Two cases of purpuric skin lesions without endocarditis have been described with *Salmonella* infection: firstly, *Henoch-Schonlein purpura* with renal insufficiency after a *S. Hirschfeldii* infection (5) and, secondly, a leukocytoclastic vasculitis complicating a *S. typhimurium* bacteremia in a patient with sickle and cell disease (6).

Salmonella typhi is the probable causative factor of the vasculitis, since the vasculitis simultaneously occurred with *S. typhi* infection and cutaneous leukocytoclastic vasculitis has been reported in association with numerous infectious diseases (7), including Gram-negative rod infections. Among many gastrointestinal pathogens are *Campylobacter* and *Yersinia*, but *Salmonella* species have been recorded (4,5). Other etiologies such as autoimmune diseases or drugs were ruled out: immunological tests were negative, and the patient had not taken any drugs before the occurrence of the vasculitis.

Pancreatitis has been infrequently described in typhoid fever (4,8). Its spectrum ranges from biological abnormalities to pancreatic abscesses requiring surgery. Splenic abscess is one of the abdominal complications of untreated typhoid fever, developing frequently in the third or fourth week of the infection (9). It represents nearly 30% of *Salmonella* abdominal infections (4).

Although splenectomy and percutaneous drainage remain the treatment of choice, our case demonstrates that medical treatment alone can be sufficient with a careful monitoring.

This observation reports the association of a cutaneous vasculitis and abdominal lesions developed during a typhoid fever. This observation adds a new infectious cause for leukocytoclastic vasculitis. It illustrates that typhoid fever is a disease representing with a variety of symptoms, including systemic vasculitis, sometimes unexpected, which may lead to misdiagnosis.

REFERENCES

1. Parry CM, Hein TT, Dougan G : Typhoid fever. N Engl J Med, 374:1770-82, 2002.

2. Maskalyk J : Typhoid fever. CMAJ, 169:132, 2003.

3. Wilkins EGL, Roberts C : Extraintestinal salmonellosis. Epidemiol Infect, 100:361-368, 1988.

4. Cohen JI, Bartlett JA, Corey GR : Extra-intestinal manifestations of Salmonella infections. Medicine, 66:349-388, 1987.

5. Fiocchi O, Stabellini G, Squerzanti R, et al : Henoch-Schonlein purpura after Salmonella hirschfeldii infection. Nephron, 55: 316-320, 1990.

6. Fincher RME, Todd Threadgill S, Cranford MS, et al : Case report: salmonellosis complicated by leukocytoclastic vasculitis. Am J Med Sci, 302:296-297, 1991.

7. Somer T, Finegold SM : Vasculitides associated with infections, immunization, and antimicrobial drugs. Clin Infect Dis, 20:1010-1036, 1995.

8. Coffernils M, Salame M, Paulet P : Salmonellosis- associated pancreatitis and pancreas divisum? J Clin Gastroenterol, 16:270, 1993.

9. Pegues DA, Ohl ME, Miller SI : Salmonella species, (including Salmonella typhi). In: Principles and practice of infectious diseases. Ed by GL Mandell, JE Bennett, R Dolin. 6th ed. NY: Churchill livingstone, 2636-2650, 2005.

Correspondence:
Jamshid Ayatollahi
Infectious and Tropical Diseases,
Research Center,
Shahid Sadoughi Hospital,
Safaeieh, Yazd,
IRAN.
e-mail: jamshidayatollahi@yahoo.com