

Cutaneous Leukocytoclastic Vasculitis, Pleural Effusion and Splenic Abscess as Unusual Presentation of Typhoid Fever

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Abstract We report a case of typhoid fever with an unusual presentation: prolonged fever with cutaneous vasculitis, pleural effusion and splenic abscess. The diagnosis was made upon Widal serology, and after ruling out other causes of leukocytoclastic vasculitis. The outcome was favourable with antibiotics alone and without surgery. This is the second case of cutaneous leukocytoclastic vasculitis associated with typhoid fever in adults.

Keywords: *typhoid fever, cutaneous vasculitis, pleural effusion, splenic abscess*

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1. Introduction

Typhoid fever has become an uncommon disease in developed countries, most often acquired during travels to endemic areas. It is usually characterized by fever, headache, and gastrointestinal manifestations [1]. However, numerous extra-intestinal manifestations of salmonella typhi infection, such as central nervous system infection [2], cardiovascular infection [3], pulmonary infection [4], osteomyelitis [5], intra abdominal abscess [4,6], and urinary tract infection [7], have been described. We report an unusual presentation of typhoid fever revealed by cutaneous leukocytoclastic vasculitis (CLV) with splenic abscess and pleural effusion.

2. Case Report

A 71 year-old man was admitted for a 2-month history of intermittent fever and a 3-day history of purpuric skin lesions and chest pain with dry cough. He had no diarrhea and no drugs intake. On physical examination, his weight was 75kg for 178cm. He was a pyretic with a 90bpm pulse, blood pressure at 130/90 mmHg, and Glasgow scale of 15/15. Pulmonary examination revealed dullness on the right lung. Skin examination showed purpuric infiltrated lesions limited to the legs. The hemogram showed 6200 WBC and 412 000 platelets per mm³, and a hemoglobin rate of 10.2g/dl. The sedimentation rate was 75 mm in the first hour and the C reactive protein rate 67 mg/l. Serum urea and creatinine, 24-hour proteinuria and urinary sediment analysis were normal. Amylase, lipase and liver enzymes were normal. Sputum examination was

negative. Tests of antinuclear antibodies and antineutrophil cytoplasm antibodies were negative. Chest X-Ray revealed a mild unilateral pleural effusion. The echocardiography was normal. The thoracentesis revealed a lymphocytic pleuritis, the culture of which was negative, and biopsy revealed a non specific inflammation with mesothelial hyperplasia. The histopathological finding of the skin biopsy was a leukocytoclastic vasculitis and bacterial culture of the skin sample was negative. Three weeks later, the patient developed a 39°C fever. 3-day stool culture remained negative. The Widal serology was positive at 1/800. Blood culture isolated salmonella. CT scan revealed a spleen enlargement with an abscess of 10 cm x 9 cm whereas examination of liver biliary ducts showed no abnormalities. The patient was treated with 500 mg of ciprofloxacin twice a day taken orally for 15 days. Apyrexia was obtained in 72 hours and the purpuric lesions disappeared. The splenic abscess disappeared in the second CT scan.

3. Discussion

The pathogenesis of CLV is still unknown, but the association of infectious agents with vasculitis was recognized decades ago [8]. During typhoid fever, the characteristic skin lesions are pink spots and pustular dermatitis [1]. Purpura or skin petechiae in Salmonella infections are rare, and always described in the setting of an endocarditis. To our best knowledge, this is the second case in adults and the seventh for all ages, of a CLV associated to a typhoid fever without endocarditis [9,10]. Al-Mayouf et al reported a series of five children who developed CLV after salmonella infection and all patients had recurrent salmonella infection as manifested by

enteritis, urinary tract infection, abscess formation and sepsis [9]. Lambotte et al described the same extra-intestinal complication in a 25-year-old patient without any recurrent infection [10].

Salmonella typhi is the probable causative factor of the vasculitis. Skin biopsy showed a histopathological finding of leukocytoclastic vasculitis with superficial and deep perivascular infiltrate of lymphocytes and neutrophils. Direct immunofluorescence was negative. *Salmonella typhi* was not identified in the skin biopsy and the search of *S. typhi* DNA by PCR in the skin sample was not ruled out [10]. C3 deposit in the derma vessel wall was demonstrated, suggesting an immune complex mediated process [10]. The pathogenic mechanisms of infection related to vascular inflammation appear to be complex: immune complex formation, vessel damage or altered vessel function mediated directly by infectious agents, humoral or cellular immunologic response [8]. Other etiologies such as auto-immune diseases or drugs were ruled out: immunological tests were negative, and the patient had not taken any drugs before the occurrence of vasculitis. The causal link of *Salmonella typhi* in the occurrence of cutaneous leukocytoclastic vasculitis is based on the positivity of bacteriological investigations, the negativity of the remaining explorations, and the favorable outcome after adequate antibiotics.

The pulmonary system involvement occurs in 1-6% of cases and pleural effusion could be due to a bronchopleural fistula [11], which is not the case reported here. We notice a primitive pleural involvement according to the findings of thoracocentesis and pleural biopsy and we suggest the immune mediated process hypothesis in this aseptic lymphocytic pleural effusion.

Splenic abscesses are one of the abdominal complications of untreated typhoid fever, developing frequently in the third or fourth week of infection. 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 with a careful monitoring can be sufficient.

4. Conclusion

This observation reports the association of a cutaneous vasculitis, splenic abscess and pleural involvement developed during a typhoid fever. It adds a new infectious cause for leukocytoclastic vasculitis. This shows that typhoid fever is a disease which may have various symptoms, including systemic vasculitis and aseptic pleural effusion.

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