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(Citation)
The Kobe journal of the medical sciences,46(5):201-204

(Issue Date)
2000-10

(Resource Type)
departmental bulletin paper

(Version)
Version of Record

(URL)
https://hdl.handle.net/20.500.14094/E0001059

Kobe J. Med. Sci. 46, 201/204, October 2000

SPLENIC ABSCESS, PLEURAL EFFUSION AND SEVERE ANEMIA CAUSED BY *Salmonella typhi*

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KEY WORDS

Salmonella typhi; splenic abscess; pleural effusion

ABSTRACT

Various complications related to gastrointestinal system, central nervous system, and skeletal system may be observed during typhoid fever, but splenic abscess and pleural effusion are rarely encountered. A 12-year-old boy was admitted with fever, fatigue and pallor. On examination he had hepatosplenomegaly and severe anemia. He was diagnosed as having *Salmonella typhi* infection complicating with splenic abscess, pleural effusion and severe anemia, and successfully treated with percutaneous drainage with ultrasonography and antibiotics. In conclusion we would like to emphasize that typhoid fever should also be considered in patients with hepatosplenomegaly and severe anemia, and percutaneous drainage with ultrasonography may successfully be used in management of splenic abscess in typhoid fever.

Received for publication : August 7, 2000

INTRODUCTION

While salmonellosis is often considered to affect primarily the gastrointestinal tract, infection at other sites may occur, producing characteristic clinical syndromes. [1,2] Although splenic abscess was observed in 1.5-1.8% patients with salmonella infection prior to antibiotic era, this ratio has markedly decreased after widespread using of antibiotics. [1] Similarly, pleural effusion and severe anemia caused by *Salmonella typhi* (*S. typhi*) are rarely seen. [2,4] In this article we report a case of splenic abscess, pleural effusion and severe anemia caused by *S. typhi* due to rare presentation.

CASE REPORT

A 12-year-old boy was admitted with a two-month history of fever, fatigue and pallor. The personal and family history were unremarkable, and he had not received any drug such as chloramphenicol before admission. Physical examination revealed; fever 39 °C, pulse rate 130/min, respiratory rate 26/min and arterial tension 100/60 mmHg. The weight and height were in normal ranges. His general condition was moderate, and he had severe pallor. The enlarged liver was palpable 4 cm below the right costal margin and the spleen was enlarged to palpate 5 cm below the left costal margin. Other systemic findings were normal.

Laboratory studies disclosed; hemoglobin 3.5 g/dl, leukocyte count 6500/mm^3, platelet count 67000/mm^3, reticulocyte count 0.4%, erythrocyte sedimentation rate 17 mm/h. On peripheral blood smear lymphocytes were predominant, the morphological characteristics of red blood cells were normochromia and normocytosis, and thrombocytopenia was noted. The bone marrow was hypocellular, with normal megakaryocyte series. Serum electrolytes, renal and liver function tests (serum total bilirubin 0.8 mg/dl, indirect bilirubin 0.6 mg/dl, aspartate aminotransferase 15 U/L, alanine aminotransferase 29 U/L and lactate dehydrogenase 132 U/L) were normal. Direct Coombs and Sickling tests were negative. Hemoglobin electrophoresis, osmotic fragility and glucose 6 phosphate dehydrogenase activity were normal. Stool examination for leukocyte and blood were unremarkable.

The patient was hospitalized in order to investigate the etiology of anemia and hepatosplenomegaly. The routine tests were received and ampicillin-sulbactam (200 mg/kg/day) plus amikacin (15 mg/kg/day) were empirically started because it was thought that he had a septicemic condition. In addition, a packed of erythrocyte suspension was given. On the 3rd day of admission amikacin was replaced by chloramphenicol (100 mg/kg/day) because the antibody against O antigen of *S. typhi* was found to be 1/800. On the 6th day of admission *S. typhi* was isolated from blood culture. On the 8th day of admission, chloramphenicol was replaced by ceftriaxone (100 mg/kg/day) according to the result of antibiogram because his general condition and fever did not improve. Because his fever persisted in spite of ceftriaxone therapy, further investigations including abdominal ultrasonography (USG) and then abdominal computerized tomography (CT) were performed. CT of the abdomen displayed hepatosplenomegaly and a hypodens area which is 2x2 cm in diameter on center pole of the spleen. This appearance was compatible with splenic abscess (Figure 1). While abdominal USG was performed, a presence of mild pleural effusion was observed on the left pleural space, and then this was confirmed with CT of the thorax. Percutaneous drainage with USG was performed and approximately 20 ml purulent material was aspirated. *S. typhi* was isolated from spleen aspiration culture as well, and it was resistant to ampicillin-sulbactam, chloramphenicol and ceftriaxone. Therefore, imipenem (50 mg/kg/day) was initiated to the result of antibiogram and his fever disappeared on the 4th day of imipenem therapy. On the 28th day of admission he was discharged from hospital without symptom, and on the 3rd month of follow up the splenic abscess completely disappeared.

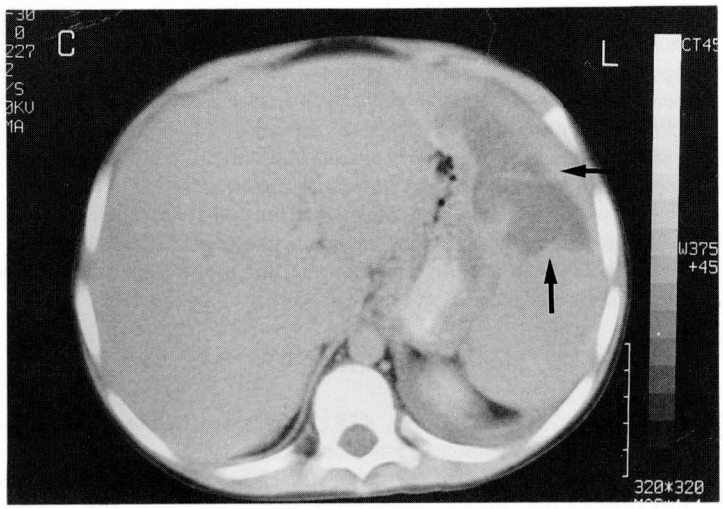

Figure 1. Computerized tomography of the abdomen shows a hypodens area which is 2x2 cm in diameter on the center pole of the spleen (see arrows).

DISCUSSION

In the last years, the number of cases of splenic abscess caused by Salmonella species have decreased parallel with decreasing of cases of typhoid fever. [4,8,10] In a study performed in our country it was noted that of 680 cases of typhoid fever requiring laparotomy during childhood two (0.29 %) had splenic abscess. [7] This study shows that the incidence of splenic abscess in typhoid fever is fairly low.

In the literature there are some reports showing the association between severe anemia and abscess caused by Salmonella, and a case of splenic abscess, pleural effusion and hemolytic anemia by *S. typhi* has been reported. The last patient with hemolytic anemia by *S. typhi* had hemosiderinuria, decreased haptoglobin concentration and high levels of lactate dehydrogenase and reticulocyte. [4-6] To our knowledge, however, a patient associated with splenic abscess, pleural effusion, non-hemolytic severe anemia and thrombocytopenia as in our case has not been recorded. It has been noted that the cause of anemia in Salmonella infection is related to intestinal blood loss or bone marrow suppression. [2] In our patient anemia was non-hemolytic because there was not anisocytosis and poikilocytosis on the peripheral blood smear, and reticulocyte count, serum indirect bilirubin and lactate dehydrogenase levels were normal. We thought that severe anemia was related to bone marrow suppression because our patient had not intestinal blood loss and the bone marrow was hypocellular.

In the diagnosis of splenic abscess USG and CT are used. [3] Although USG is a cheap and practical method, CT is more useful and reliable. Because the factors such as the capability and experience of radiologist, and hindering of appearance of spleen by the other organs such as colon affect the reliability of ultrasonographic examination. Although abscesses in a few mm diameter can also be observed and localized in CT, USG is more practical and useful method in percutaneous drainage of splenic abscess as in our case. [3]

In the previous years splenectomy was performed in the treatment of splenic abscesses caused by typhoid fever. [7,10] However, in the last years percutaneous drainage with USG plus antibiotic therapy have been successfully used in the treatment of splenic abscess caused by *S. typhi*. [9] In our case ampicillin-sulbactam and amikacin were initially used and then ceftriaxone was given, but no clinical response could be obtained. The cause of failure was the resistance to the antibiotics and splenic abscess. We obtained a complete clinical improvement with percutaneous drainage plus appropriate antibiotic therapy including imipenem.

In conclusion we would like to emphasize that typhoid fever should also be considered in patients with hepatosplenomegaly and severe anemia, and salmonella infections may be associated with splenic abscess, pleural effusion and severe anemia, and percutaneous drainage with USG may successfully be used in the management of splenic abscess in typhoid fever.

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