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CASE REPORT

Polyserositis due to *Salmonella enterica* serovar Enteritidis

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Summary *Salmonella enterica* serovar Enteritidis (S. Enteritidis), a non-typhoid *Salmonella* is an important emerging pathogen that usually causes gastroenteritis. Here, we report polyserositis with right-sided pleural effusion and loculated collection of fluid in the peritoneum caused by S. Enteritidis in a 60-year-old man from southern India. The patient was immunocompetent and did not have preceding gastroenteritis or any local structural abnormality. Malnutrition and old age might have been the predisposing factors. The patient received intravenous ceftriaxone for 2 weeks followed by oral ciprofloxacin. Pleurocentesis and abdominal paracentesis were also done. The patient was cured as evidenced by clinical improvement and radiological disappearance of the fluid collection.

1. Introduction

Salmonella enterica serovar Enteritidis (S. Enteritidis), a non-typhoid *Salmonella*, is an important emerging pathogen causing infections that are most often zoonotic, transmitted by the consumption of raw or improperly cooked eggs or chicken.[1] It usually causes gastroenteritis, but it can also cause bacteraemia and subsequent metastatic extra-intestinal manifestations in immunocompromised patients and those with local structural abnormalities.[2] Here, we report a case of empyema and loculated peritoneal fluid collection due to S. Enteritidis in a patient without preceding gastroenteritis or any underlying structural abnormality.

2. Case report

A 60-year-old retired postman from a village presented to R.L. Jalappa Hospital, Kolar, India, in August 2006, with a 4 month history of easy fatigability, abdominal distension and swelling of the lower limbs. Two weeks prior to his illness he had fever with chills and rigors that subsided after antipyretics, but fever recurred intermittently. He had loss of appetite with drastic weight loss. There was no history of chest pain, cough, paroxysmal nocturnal dyspnoea, orthopnea, vomiting, abdominal pain or diarrhoea. The patient gave informed consent for his investigation and treatment.

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On examination he was afebrile and weighed 45 kg. Bilateral, pitting, pedal oedema extending to the knee, marked pallor, bald tongue and platynychia were observed. Vital signs and jugular venous pressure were normal. The abdomen was uniformly distended and the veins over the abdominal wall were engorged with blood flow from below. There was dullness around the umbilicus and the liver and spleen were not palpable. Examination of the respiratory system revealed right-sided pleural effusion. There were no signs of pericarditis and ECG was normal.

Haemoglobin was 4.2 g/dl, total leukocyte count 12.2×10^9/l (neutrophils 90%; lymphocytes 8%; eosinophils 2%) and platelet count 424×10^9/l. A peripheral blood smear showed dimorphic anaemia with neutrophilic leukocytosis. ESR was 150 mm/h. Liver function tests showed total protein of 68 g/l (albumin 21 g/l, globulin 47 g/l; with a reversed albumin/globulin ratio of 0.4). Serological tests for HIV were negative.

Chest X-ray confirmed the right-sided pleural effusion. Ultrasonography of the abdomen showed loculated ascites with septations. Liver and spleen were normal and the diameter of the portal vein was 1.1 cm. A CT scan showed right-sided pleural effusion and loculated collection of fluid in the peritoneum along the anterior abdominal wall with a normal pericardium. Upper gastrointestinal endoscopy was normal. A provisional diagnosis of polyserositis due to tuberculosis was made.

Pleural fluid from diagnostic aspiration was turbid: protein 32 g/l, LDH 422 IU/l and cell count 16.0×10^9/l; cytology: 85% neutrophils, a few lymphocytes and degenerative cells. Peritoneal fluid aspirate was yellow and thick: protein 30 g/l, LDH 300 IU/l and cell count 18.0×10^9/l; cytology: 75% neutrophils, a few lymphocytes and occasional reactive mesothelial cells. Malignant cells were not found in pleural or peritoneal fluid samples.

A moderate number of pus cells and a few Gram-negative bacilli were seen in the Gram smear of the pleural fluid; the peritoneal fluid showed numerous pus cells but no organisms. Ziehl-Neelsen staining of both the fluids was negative for acid-fast bacilli. Pleural and peritoneal fluid cultures yielded S. Enteritidis (antigenic formula 9,12:g,m) as identified by biochemical reactions and serology. Both the isolates were sensitive to ampicillin, ceftriaxone, amoxicillin–clavulanic acid, tetracycline, chloramphenicol, cotrimoxazole and ciprofloxacin. Repeat culture from pleural fluid after 4 days yielded the same organism with a similar sensitivity pattern.

Blood cultures (one collected before and another after 48 h of antibiotic therapy) were sterile. Stool culture did not yield any pathogens and urine culture was sterile. A Widal test done 1 week after admission showed a titre of 1:160 against O and H antigens of *Salmonella* Typhi; there were no detectable antibodies against H antigens of *Salmonella* Paratyphi A and *Salmonella* Paratyphi B.

In light of these findings a diagnosis of polyserositis due to S. Enteritidis was made. The patient received 2 g intravenous ceftriaxone twice daily for 2 weeks followed by ciprofloxacin 500 mg orally, twice daily for 6 weeks. Anaemia was treated with packed cell transfusion and oral haematinics. Pleurocentesis was done at admission and 1 week later. Similarly, peritoneal fluid was aspirated twice. Culture of pleural fluid after 21 days did not grow any pathogens, although the smear showed many neutrophils. After 2 months the patient had no fever and had gained weight significantly. Ultrasonography showed minimal right-sided pleural effusion and no fluid collection in the abdomen. Oral administration of ciprofloxacin 500 mg twice daily was continued for another 2 months and the patient recovered completely.

3. Discussion

Non-typhoidal salmonellosis is an important public health problem worldwide. Among non-typhoidal *Salmonellae*, S. Typhimurium and S. Enteritidis are the most common serovars reported.[2] *Salmonella* Enteritidis typically causes diarrhoea but can cause empyema, lung abscess, endocarditis, cystitis, pyomyositis, arthritis of the hip and cutaneous abscesses in immunocompromised persons.[3] Rarely, S. Enteritidis causing anterior chest-wall abscess, brain abscess and pleural empyema in immunocompetent patients has been described.[4,5]

Our patient presented with easy fatigability, breathlessness, abdominal distension and swelling of the lower limbs for 4 months. The symptoms were clinically suggestive of cardiac or hepatic dysfunction but, after investigation, the patient was diagnosed with polyserositis. He was apparently immunocompetent, and old age and malnutrition may have been the predisposing factors for his illness. Severe nausea and decreased food intake may have worsened his anaemia.

To the best of our knowledge this is the first report of polyserositis due to S. Enteritidis. In developing countries like India, tuberculosis is the most common cause of polyserositis. This case shows that S. Enteritidis can, rarely, cause polyserositis in an apparently immunocompetent individual without gastrointestinal symptoms.

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Conflicts of interest: None declared.

Ethical approval: Not required; the assessment and treatment of the patient were part of the standard clinical procedures at Sri R.L. Jalappa Hospital, Kolar, India. Informed consent for the publication of this case report was given by the patient.

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