

## BRIEF REPORT

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***Salmonella* serovar Give: an unusual pathogen causing splenic abscess**Published online: 25 March 2006  
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*Salmonella enterica* subsp. *enterica* serovar Give is an enteric serotype frequently isolated from ruminants and pigs but rarely found in human hosts [1]. Between April and June 2004, an unusual increase was observed in the number of *S. ser. Give* isolates identified by several European national reference laboratories [2]. In 11 countries, 103 cases were detected, and only a minority of these infections was related to travel. Further European data suggest a recent increase in the prevalence and pathogenicity of *S. ser. Give*. For example, from March to July 2004, 115 cases of gastroenteritis caused by *S. ser. Give* were identified in Germany and these were mainly responsible for the increased number of cases reported in the Enter-net data of the international surveillance network for enteric infections during the second quarter of 2004 [3]. This nationwide outbreak was strongly associated with human consumption of raw minced pork (OR, 8.0; 95%CI, 2.3–27.7;  $p=0.001$ ), and common outbreak strains were isolated from patients and pork samples. Compared to

patients infected by *S. ser. Enteritidis*, the most common serovar in Germany, infections caused by *S. ser. Give* led to a higher hospitalization rate in patients older than 60 years (*S. ser. Give* 55%, *S. ser. Enteritidis* 33%; OR, 2.5; 95%CI, 1.1–5.4;  $p=0.02$ ) [3]. The higher hospitalization rate observed for *S. ser. Give* possibly indicates this serotype has a higher virulence compared to other non-typhoidal *Salmonella* spp. Reported here is a clinical case of an immunocompetent patient presenting with splenic abscess due to *S. ser. Give*, which was possibly associated with consumption of raw minced meat.

In March 2004, a 24-year-old Caucasian male returning from a vacation in southern Thailand with his family was admitted to our walk-in clinic with increasing fever, dyspnea and left shoulder pain. He had no medical history of note. He had been well until February 2004, when he suddenly developed a high fever, malaise, nausea, dry cough and increasing dyspnea. He received intramuscular penicillin G injections for 2 days without any improvement, then a combination of oral amoxicillin and clavulanic acid (625 mg tid) over 7 days for suspected bronchopneumonia of the left lower lobe. Five days following initiation of the second antibiotic therapy, the patient felt better and fever disappeared. Following completion of the antibiotic regimen, however, symptoms relapsed, with fever up to 40°C, chills, abdominal discomfort and unbearable left shoulder pain triggered by deep inspiration and coughing. Two days after completion of the oral regimen, the patient returned for further investigations.

Physical examination revealed a body temperature of 38°C, dyspnea and polypnea (28/min), tachycardia (122/min), diffuse subcutaneous edema, dull percussion at the left inferior thoracic base and hepatosplenomegaly. Laboratory tests revealed a raised C-reactive protein level (301 mg/l), leukocytosis ( $15 \times 10^9/l$ ) with a left shift (8%), thrombocytosis ( $804 \times 10^9/l$ ), hypoalbuminemia (17 g/l), and elevated liver enzymes (alanine aminotransferase 80, aspartate aminotransferase 37 U/l, gamma-glutamyl transpeptidase 288 U/l). Chest radiographs showed a raised and thickened left diaphragm with signs of an expanding mass with air fluid level and stomach compression in the left

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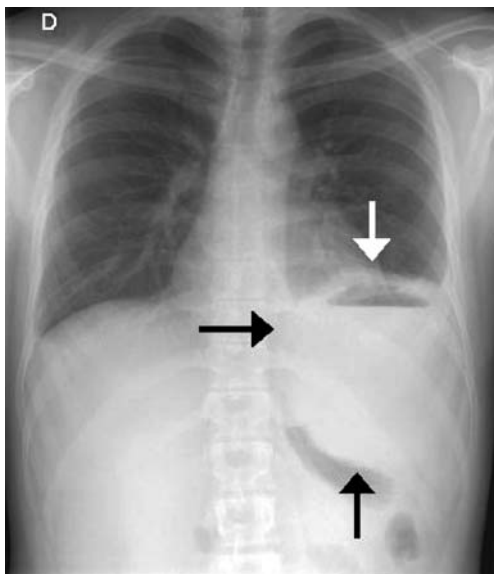
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abdominal quadrant (Fig. 1). A splenic abscess was confirmed by computed tomography, which revealed a huge hypodense mass in the spleen with gas formation displacing adjacent organs (Fig. 2). Considering the location and extent of the abscess, as well as the risk of a splenic rupture and septic complications following drainage, splenectomy was performed.

Stool and blood cultures, blood slides and serological tests were negative for melioidosis, malaria, amoebiasis (ELISA, 0.00 OD), and helminthiasis (ELISA, 0.00 OD for trichinellosis, toxocarasis, *Echinococcus granulosus*, *Echinococcus multilocularis*, fascioliasis, schistosomiasis, anguilluliasis, and filariasis). Postoperative samples of the splenic abscess were positive for *S. ser. Give*. The strain was susceptible to piperacillin/tazobactam, ceftazidime, ciprofloxacin, cotrimoxazole, imipenem, gentamicin, netilmicin, amikacin, nitrofurantoin, and colistin. However, it also demonstrated intermediate resistance to amoxicillin/clavulanic acid and resistance to ampicillin, ticarcillin, ticarcillin/clavulanic acid, cephalothin, chloramphenicol, kanamycin, streptomycin, spectinomycin, and tetracycline. This resistance profile could explain the unfavorable course of the administered antimicrobial therapy.

The patient had a non-penetrating trauma as a single risk factor: he remembered having fallen on slippery stairs and experiencing subsequent transient pain of the left flank 5 weeks before the first symptoms appeared. He also mentioned a transient episode of non-bloody diarrhea that occurred around the same time following consumption of raw minced meat. It is thus probable that intestinal *S. ser. Give* spread to a hematoma arising from the accident-related injury during transient bacteremia, which then caused the splenic abscess. The patient's left shoulder pain could be explained by the infradiaphragmatic irritation. We postulate that this clinical picture is a result of secondary



**Fig. 1** Chest radiograph showing signs of a prominent splenic abscess with air fluid level and stomach displacement



**Fig. 2** Computed tomography confirming the presence of a prominent splenic abscess with air fluid level

infection and suppuration of the previously contused spleen. To our knowledge, this is the first report of a case of splenic abscess due to *S. ser. Give*.

Splenic abscesses are more prevalent in males and in younger age groups. Most of them are preceded by infections elsewhere in the body, which act as a focus for dissemination of microorganisms, unless the spleen is infected primarily through direct penetration [4, 5]. Streptococci and staphylococci, *Escherichia coli*, *Bacterioides* spp, enterococci and *Salmonella* Typhi are frequently cultured organisms: fungi and mycobacteria and, especially in Asia, *Burkholderia pseudomallei* are also reported to cause splenic abscess [6–9]. Immunosuppression, intravenous drug abuse, HIV, non-penetrating abdominal trauma and hemoglobinopathies are considered as predisposing factors.

Splenic abscess must be considered in a large number of differential diagnoses, especially in febrile patients with symptoms of polymorphic pain. Left upper quadrant tenderness with fever and leukocytosis are the most common clinical features. Computed tomography remains the gold standard and the most sensitive tool for the definitive diagnosis of splenic abscess [10]. Splenectomy is considered the treatment of choice by most authors, while percutaneous drainage and antimicrobial therapy can be considered as therapeutic alternatives for select clinical conditions and when the pathogen has been clearly identified.

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