



Salmonella paratyphi a sepsis presenting as acute ovarian cyst rupture

Authors

G Suresh Reddy^{1*}, Basava Prabhu¹ Dr Satya Prasad²

¹Department of Internal Medicine, Kasturba Medical College, Mangalore, India

²Department of Internal Medicine, Apollo Main Hospitals, Chennai

*Corresponding Author

Dr G Suresh Reddy

Kasturba Medical College, Mangalore, India

Abstract

We are reporting a case of Salmonella paratyphi A in a young female, who presented with acute abdomen, ruptured ovarian cyst, who was operated in emergency. Pus which was aspirated from the ruptured cyst and the peritoneal cavity isolated Salmonella Paratyphi A. She has an episode of Acute Gastro Enteritis 2 months ago treated conservatively then with IV antibiotics-? Sub optimally treated Enteric Fever. Now the patient presented as said above with an infected and a rupture ovarian cyst.

Extra intestinal manifestations of enteric fever are quite rare, however the possibility of the infection may be suspected in patients presenting acutely post an intestinal infection or a fever as we have seen in our case.

With prompt recognition and multidisciplinary management, the patient recovered with no serious sequela.

Introduction

This is a case of Salmonella paratyphi A in a young female, who presented with acute abdomen, ruptured ovarian cyst, who was operated in emergency. Pus which was aspirated from the ruptured cyst and the peritoneal cavity isolated Salmonella Paratyphi A. She has an episode of Acute Gastro Enteritis 2 months ago treated conservatively then with IV antibiotics - **A Sub optimally treated Enteric Fever.** Now the patient presented as said above with an infected and a rupture ovarian cyst. Extra intestinal manifestations of enteric fever are quite rare, however the possibility of the infection may be suspected in patients presenting acutely post an intestinal infection or a fever as we have seen in our case. With prompt recognition and

multidisciplinary management, the patient recovered with no serious sequela.

Background

Enteric fever is more common in children and young adults than in older patients^[1]. Worldwide, enteric fever is most prevalent in impoverished areas that are overcrowded with poor access to sanitation. Incidence estimates suggest that south-central Asia, Southeast Asia, and southern Africa are regions with high incidence of *S. Typhi* infection (more than 100 cases per 100,000 person-years^[2-4]). Other regions of Asia and Africa, some parts of Latin America, the Caribbean, and Oceania have a medium incidence of 10 to 100 cases per 100,000 person-years. These estimates, though, are limited by lack of consistent reporting from all areas of the world and are based on

extrapolation of data across regions and age groups. *S. Paratyphi A* remains uncommon in Africa^[5], but accounts for a substantial proportion of enteric fever cases in areas of South Asia^[6].

Because humans are the only reservoir for *S. Typhi*, a history of travel to settings in which sanitation is poor or contact with a known typhoid case or carrier is useful for identifying people at risk of infection outside of endemic areas, although a specific source or contact is identified in a minority of cases. Transmission from an index case to a contact is rarely documented in resource-rich settings^[7].

In a study of 428 cases of enteric fever reported among travelers from resource-rich countries through the multinational Geo Sentinel Surveillance Network between 2006 and 2011, 67 percent of cases were acquired in south-central Asia (34, 13, 7, and 6 percent of total from India, Nepal, Pakistan, and Bangladesh, respectively) (figure 1)^[9]. Individuals visiting relatives in endemic countries accounted for 28 percent of the typhoid cases.

Many travelers who subsequently develop enteric fever have not received appropriate vaccination despite guideline recommendations. Among 580 cases of vaccine-preventable diseases among returned international travelers reported to the GeoSentinel Surveillance Network between 1997 and 2007, confirmed or probable enteric fever (due mainly to *S. Typhi*, but also *S. Paratyphi*) was the most common^[10]. Only 38 percent of those with enteric fever had a pre-travel clinical encounter. However, the possibility of *S. Typhi* infection in returning travelers with a history of vaccine receipt should not be discounted, since the vaccine is not completely effective.

Patients who acquire infection abroad are usually older than those who acquire disease in United States outbreaks and are more likely to have drug-resistant infection. *S. Typhi* outbreaks in the United States are most often food borne; they are generally limited in size but can cause substantial morbidity^[8, 11].

The risk factors for the development of enteric fever due to *S. Typhi* or *S. Paratyphi* may differ. In an Indonesian study, transmission of paratyphoid fever was more frequently observed outside the home (eg, via consumption of food purchased from street vendors); transmission of typhoid fever was more frequently observed within the household (e.g., via sharing utensils, presence of a patient with typhoid, lack of soap or adequate toilet facilities)^[12]. Some evidence suggests that *S. Paratyphi* may be more likely to be transmitted by food, while *S. Typhi* may spread more via contaminated water supply^[13], though this remains to be confirmed. *S. Paratyphi* also appears to be an increasing cause of enteric fever among vaccinated travelers, as the Vi polysaccharide typhoid vaccine is ineffective against most *S. Paratyphi*^[14,15], which lack the Vi antigen targeted by the vaccine. Vaccines for *S. Typhi* induce detectable immune responses to *S. Paratyphi* in vitro, but the clinical significance of this finding is unknown^[16,17].

Enteric (typhoid) fever is a systemic disease characterized by fever and abdominal pain caused by dissemination of *Salmonella enterica* serotype typhi (*S. typhi*) and *S. enterica* serotype paratyphi (*S. paratyphi*) A, B, C^[18] and *Salmonella Choleraesuis*.^[19] Contaminated food and water are the major sources of infection, enteric fever develops as a febrile illness with onset of symptoms post 5 to 21 days after ingestion. Generally, lower inocula are associated with longer incubation times. However, both the incubation period and inoculum needed to cause disease vary depending upon host factors such as age, gastric acidity, and immunologic status.

The majority of patients with enteric fever present with abdominal pain, fever, and chills.

Untreated individuals may present with classic characteristic stages of enteric fever.^[20] In the first week of illness, slow rising "stepwise" fever and bacteremia develops^[21]. Though chills are typical, frank rigors are rare to be seen^[17]. Relative bradycardia or pulse-temperature dissociation is also seen. Second week of illness, abdominal pain

develops and "rose spots" (faint salmon-coloured macules on the trunk and abdomen) may be seen. During the third week of illness, hepatosplenomegaly, intestinal bleeding, and perforation due to ileocecal lymphatic hyperplasia of the Peyer's patches may occur, together with secondary bacteremia and peritonitis. Septic shock, altered level of consciousness may develop. A study done on 300 patients of typhoid fever in Indonesia, these findings were observed in approximately 15 percent of patients.^[22] In the absence of acute complications or death from overwhelming sepsis, symptoms gradually resolve over weeks to months.

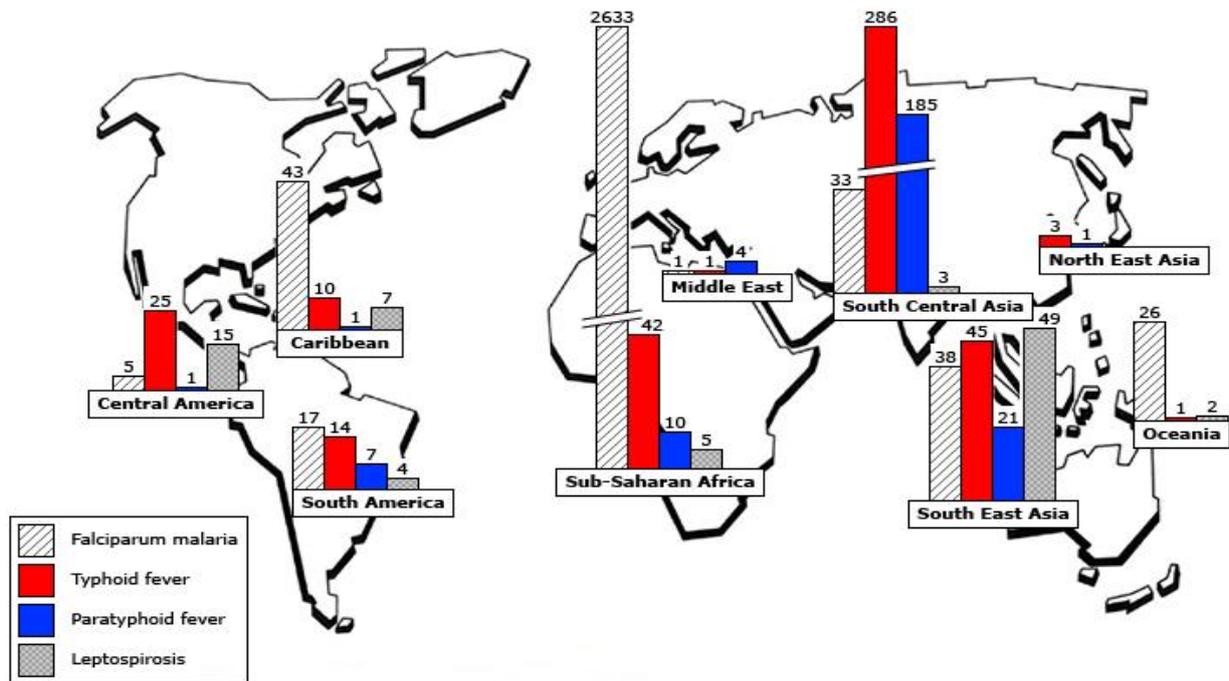
Certain underlying conditions increase this risk of bacteremia such as immunodeficiency, immunosuppressive therapy and young age.^[23] Asymptomatic carriers of Salmonella can also be identified.^[24]

Enteric fever –

Enteric (typhoid) fever is a systemic disease characterized by fever and abdominal pain caused by dissemination of Salmonella enterica serotype

typhi (*S. typhi*) and *S. enterica* serotype paratyphi (*S. paratyphi*) A, B, C and *Salmonella* Choleraesuis. Contaminated food and water are the major sources of infection; enteric fever develops as a febrile illness with onset of symptoms post 5 to 21 days after ingestion. The majority of patients with enteric fever present with abdominal pain, fever, and chills. Untreated individuals may present with classic characteristic stages of enteric fever. In the first week of illness, slow rising "stepwise" fever and bacteraemia develops. Second week of illness, abdominal pain develops and "rose spots" (figure 2) (faint salmon-coloured macules on the trunk and abdomen) may be seen. During the third week of illness, hepatosplenomegaly, intestinal bleeding, and perforation due to ileocecal lymphatic hyperplasia of the Peyer's patches may occur, together with secondary bacteraemia and peritonitis. Septic shock, altered level of consciousness may develop. In the absence of acute complications or death from overwhelming sepsis, symptoms gradually resolve over weeks to months.

Figure 1



This map indicates the number of cases of selected acute and potentially life-threatening diseases region reported among 82,825 travelers from resource-rich countries to various tropical regions between 1996 to 2011. Data are from the Geo Sentinel surveillance network.

Figure 2

Rose spots are small (1 to 5 mm), erythematous, blanchable, nontender papules, which begin early during the acute febrile period of typhoid fever. Crops of lesions (10 to 20) appear at irregular intervals for approximately 10 to 14 days, typically distributed on the abdomen, chest, and back. Rarely, vesicular or hemorrhagic lesions appear. The lesions persist for two to three days.

Case Report

A 31-year-old nulliparous woman without any known co-morbidities was received in the Emergency Unit with chief complaints severe abdominal pain, burning micturition and vomiting. She was admitted for further evaluation and management. Detailed history suggestive of abdominal pain since 2 weeks worsened since 2 days, crampy and diffuse in nature, multiple episodes of non bilious and non projectile vomiting for 2 days and also burning micturition since 3 days, no history hematuria or dysuria. History of fever associated with chills and rigors.

Past history suggestive of admission one month ago with complaints of abdominal pain, high grade fever with chills and rigors, multiple episodes of vomiting, loss of appetite and loss of weight of about 7 kgs in past 3 months. She was evaluated for the same with blood, biochemistry and sonological investigations thoroughly. Blood cultures did not isolate any organism. Upper GI endoscopy was normal. Widal test was negative. Stool and urine routines were normal. Liver and renal parameters were well within limits. Ultrasound whole abdomen suggested of Bilateral

ovarian cysts. She was managed conservatively for the same and discharged.

She also had a history of admission at an elsewhere Hospital 2 months ago for fever, loose stools and dehydration managed conservatively with IV Fluids and antibiotics and discharged. She underwent laparotomy in the year 2006 for endometriotic cyst.

On clinical examination patient looked dehydrated with diffuse abdominal tenderness. In view of a suspicion of ruptured ovarian, a general surgery consult was given. Contrast Enhanced CT abdomen was done which showed bilateral cystic lesions in ovaries with moderate ascites. An emergency exploratory laparotomy was planned and right salpingo-oophorectomy was done.

Intraoperative finding suggested of an aspiration of frank pus from the peritoneal cavity and a ruptured right sided ovarian cyst. The pus was sent for culture and sensitivity. Intraoperatively patient developed hypotension patient. Patient needed inotropes support and broad spectrum antibiotics.

Salmonella Paratyphi A was isolated from the pus which was sensitive to Ceftriaxone,

cotrimoxazole, meropenem and cefixime. Patient was treated with meropenem and metronidazole, later changed to oral cefixime. Clinically she improved. Hence she was discharged.

Literature Review

There have been at least 17 case reports of *Salmonella* spp. causing ovarian or tubo-ovarian abscesses in the literature since 1975,^[27] mostly due to *S. typhi* and nontyphoid salmonella. This would be the third involving *S. paratyphoid A*, and given the lack of effective vaccine may become a more prominent pathogen causing localised suppurative complications.^[28]

A Review of the Literature was published in 2017 and reported previous ovarian cysts and endometriomas infected with *Salmonella* between 1963 and 2016.^[29] There were 33 cases reported from 2 to 48 years old, mostly from Asia and Europe, only 55% of them had preceding diarrheal illness and most of the time, the diagnostic test was ultrasound or CT. Most of them had endometrioma or teratoma and only three of them had sepsis. The most common types of *Salmonella* were enteritis and typhi. All of these patients were treated with surgery and antibiotics such as penicillins, third-generation cephalosporins and fluoroquinolones.^[30-37]

Discussion

Both *Salmonella typhi* and *S. paratyphi* are known for spread beyond the alimentary track and can infect almost every organ, causing localised suppurative complications.^[25] These bacteria avoid significant host immune response when translocating the gut mucosa. After phagocytosis they replicate within cells of the gut-associated lymphoid tissue and of other cells of monocytic lineage that are disseminated throughout the reticuloendothelial system.^[26]

S. paratyphi which is the causative organism in this case, is presumed to have resulted in haematogenous spread to a pre-existing ovarian cyst. The cystic medium provided a harbour from lethal concentrations of antibiotics, given in

previous admissions, with survival and relapse of a febrile illness and culture of the organism from the tubo-ovarian and peritoneal collection.

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