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SALLMOMELLOSIS-AN ACUTE INFECTIOUS DISEASE THAT IS TRANSMITTED BY THE FECAL-ORAL MECHANISM, MAINLY DAMAGE TO THE GASTROINTESTINAL TRACT

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Annoation

Bacteria of the genus Salmonella are highly adapted for growth in both humans and animals and cause a wide spectrum of disease. The growth of serotypes S. Typhi and S. Paratyphi is restricted to human hosts, in whom these organisms cause enteric (typhoid) fever. The remaining serotypes (nontyphoidal Salmonella, or NTS) can colonize the gastrointestinal tracts of a broad range of animals, including mammals, reptiles, birds, and insects. More than 200 serotypes are pathogenic to humans, in whom they often cause gastroenteritis and can be associated with localized infections and/or bacteremia.

Key words

Salmonellae, gram-negative bacilli, S. bongori, S. choleraesuis, Typhoid fever, Enteric fever, fluoroquinolones, amoxicillin, trimethoprim-sulfamethoxazole (TMP-SMX), ciprofloxacin.

This large genus of gram-negative bacilli within the family Enterobacteriaceae consists of two species: S. choleraesuis, which contains six subspecies, and S. bongori. S. choleraesuis subspecies I contains almost all the serotypes pathogenic for humans. Because the designation S. choleraesuis refers to both a species and a serotype, the species designation S. enterica has been recommended and widely adopted. According to the current Salmonella nomenclature system, the full taxonomic designation Salmonella enterica sub species enterica serotype Typhimurium can be shortened to Salmonella serotype Typhimurium, or simply Salmonella Typhimurium.

Members of the seven Salmonella subspecies are classified into >2400 serotypes (serovars) according to the somatic O antigen [lipopolysaccharide (LPS) cell-wall components], the surface Vi antigen (restricted to S. Typhi and S. Paratyphi C), and the flagellar H antigen. For simplicity, most Salmonella serotypes are named for the city where they were identified, and the serotype is often used as the species designation.

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Salmonellae are gram-negative, non–spore-forming, facultatively anaerobic bacilli that measure 2–3 by 0.4–0.6 µm. The initial identification of salmonellae in the clinical microbiology laboratory is based on growth characteristics. Salmonellae, like other Enterobacteriaceae, produce acid on glucose fermentation, reduce nitrates, and do not produce cytochrome oxidase. In addition, all salmonellae except S. Gallinarum-Pullorum are motile by means of peritrichous flagella, and all but S. Typhi produce gas (H_2S) on sugar fermentation. Notably, only 1% of clini cal isolates ferment lactose; a high level of suspicion must be maintained to detect these rare clinical lactose fermenting isolates.

Although serotyping of all surface antigens can be used for formal identification, most laboratories perform a few simple agglutination reactions that define specific O-antigen serogroups, designated A, B, C1, C2, D, and E. Strains in these six serogroups cause ~99% of Salmonella infections in humans and warm-blooded animals. Molecular typing methods, including pulsed-field gel electrophoresis, are used in epidemiologic investigations to differentiate Salmonella strains of a common serotype.

Typhoid fever is a systemic disease characterized by fever and abdominal pain and caused by dissemination of S. Typhi or S. Paratyphi. The disease was initially called typhoid fever because of its clinical similarity to typhus. However, in the early 1800s, typhoid fever was clearly defined pathologically as a unique illness on the basis of its association with enlarged Peyer's patches and mesenteric lymph nodes. In 1869, given the anatomic site of infection, the term enteric fever was proposed as an alternative designation to distinguish typhoid fever from typhus. However, to this day, the two designations are used interchangeably.

In contrast to other Salmonella serotypes, the etiologic agents of enteric fever—S. Typhi and S. Paratyphi serotypes A, B, and C—have no known hosts other than humans. Most commonly, food-borne or waterborne transmission results from fecal contamination by ill or asymptomatic chronic carriers. Sexual transmission between male partners has been described. Health care workers occasionally acquire enteric fever after exposure to infected patients or during processing of clinical spec- imens and cultures.

Enteric fever is a misnomer, in that the hallmark features of this disease—fever and abdominal pain—are variable. Although fever is documented at presentation in >75% of cases, abdominal pain is reported in only 30–40%. Thus a high index of suspicion for this potentially fatal systemic illness is necessary when a person presents with fever and a history of recent travel to a developing country. The incubation period for S. Typhi averages 10–14 days but ranges from 3 to 21 days, with the duration likely reflecting the inoculum size and the host's health and

immune status. The most prominent symptom is pro longed fever (38.8°–40.5°C; 101.8°–104.9°F), which can continue for up to 4 weeks if untreated. S. Paratyphi A is thought to cause milder disease than S.Typhi, with pre dominantly gastrointestinal symptoms. However, a prospective study of 669 consecutive cases of enteric fever in Kathmandu, Nepal, found that the infections were clinically indistinguishable. In this series, symptoms reported on initial medical evaluation included headache (80%), chills (35–45%), cough (30%), sweating (20–25%), myalgias (20%), malaise (10%), and arthralgia (2–4%). Gastrointestinal symptoms included anorexia (55%), abdominal pain (30–40%), nausea (18–24%), vomiting (18%), and diarrhea (22–28%) more commonly than constipation (13–16%). Physical findings included coated tongue (51–56%), splenomegaly (5–6%), and abdominal tenderness (4–5%) Early physical findings of enteric fever include rash ("rose spots"), hepatosplenomegaly (3–6%), epistaxis, and relative bradycardia at the peak of high fever. Rose spots make up a faint, salmon-colored, blanching, maculopapular rash located primarily on the trunk and chest. The rash is evident in ~30% of patients at the end of the first week and resolves without a trace after 2–5 days. Patients can have two or three crops of lesions, and Salmonella can be cultured from punch biopsies of these lesions. The faintness of the rash makes it difficult to detect in highly pigmented patients. The development of severe disease (which occurs in ~10–15% of patients) depends on host factors (immune-suppression, antacid therapy, previous exposure, and vaccination), strain virulence and inoculum, and choice of antibiotic therapy. Gastrointestinal bleeding (10–20%) and intestinal perforation (1–3%) most commonly occur in the third and fourth weeks of illness and result from hyperplasia, ulceration, and necrosis of the ileocecal Peyer's patches at the initial site of Salmonella infiltration. Both complications are life-threatening and require immediate fluid resuscitation and surgical intervention, with broadened antibiotic coverage for polymicrobial peritonitis and treatment of gastrointestinal hemorrhages, including bowel resection. Neurologic manifestations occur in 2–40% of patients and include meningitis, Guillain Barré syndrome, neuritis, and neuropsychiatric symptoms (described as "muttering delirium" or "coma vigil"), with picking at bedclothes or imaginary objects.

Rare complications whose incidences are reduced by prompt antibiotic treatment include disseminated intravascular coagulation, hematophagocytic syndrome, pancreatitis, hepatic and splenic abscesses and granulomas, endocarditis, pericarditis, myocarditis, orchitis, hepatitis, glomerulonephritis, pyelonephritis and hemolytic uremic syndrome, severe pneumonia, arthritis, osteomyelitis, and parotitis. Up to 10% of patients develop mild relapse, usually

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within 2–3 weeks of fever resolution and in association with the same strain type and susceptibility profile.

Up to 10% of untreated patients with typhoid fever excrete S. Typhi in the feces for up to 3 months, and 1–4% develop chronic asymptomatic carriage, shedding S. Typhi in either urine or stool for >1 year. Chronic carriage is more common among women, infants, and persons with biliary abnormalities or concurrent bladder infection with Schistosoma haematobium. The anatomic abnormalities associated with the latter conditions presumably allow prolonged colonization.

The definitive diagnosis of enteric fever requires the isolation of S.Typhi or S.Paratyphi from blood, bone marrow, other sterile sites, rose spots, stool,or intestinal secretions.The yield of blood cultures is quite variable; sensitivity is as high as 90% during the first week of infection and decreases to 50% by the third week. A low yield in infected patients is related to low numbers of salmonellae (<15 organisms/mL) and/or to recent antibiotic treatment. Since almost all S.Typhi organisms in blood are associated with the mononuclear-cell/platelet fraction, centrifugation of blood and culture of the buffy coat can substantially reduce the time to isolation of the organism but does not increase sensitivity. Unlike blood culture, bone marrow culture remains highly (90%) sensitive despite ≤5 days of antibiotic therapy. Culture of intestinal secretions (best obtained by a non-invasive duodenal string test) can be positive despite a negative bone marrow culture. If blood, bone marrow, and intestinal secretions are all cultured, the yield is >90%. Stool cultures, although negative in 60–70% of cases during the first week, can become positive during the third week of infection in untreated patients. Several serologic tests, including the classic Widal's test for "febrile agglutinins", are available. None of these tests are sufficiently sensitive or specific to replace culture-based methods for the diagnosis of enteric fever in developed countries. Polymerase chain reaction and DNA probe assays to detect S.Typhi in blood are being developed.

Treatment: Prompt administration of appropriate antibiotic therapy prevents severe complications of enteric fever and results in a case-fatality rate of <1%.The initial choice of antibiotics depends on the susceptibility of the S.Typhi and S.Paratyphi strains in the area of residence or travel. For treatment of drug-susceptible typhoid fever, fluoroquinolones are the most effective class of agents, with cure rates of ~98% and relapse and fecal carriage rates of <2%.Experience is most extensive with ciprofloxacin. Short-course ofloxacin therapy is similarly successful against infection caused by nalidixic acid susceptible strains. However, the increased incidence of nalidixic acid–resistant (NAR) S.Typhi in Asia, which is

probably related to the widespread availability of fluoroquinolones over the counter, is now limiting the use of this drug class for empirical therapy. Patients infected with NAR S.Typhi strains should be treated with ceftriaxone, azithromycin or high-dose ciprofloxacin. However, high-dose fluoroquinolone therapy for NAR enteric fever has been associated with delayed resolution of fever and high rates of fecal carriage during convalescence. Ceftriaxone, cefotaxime and (oral) cefixime are effective for treatment of MDR enteric fever, including NAR and fluoroquinolone-resistant strains. These agents clear fever in ~1 week, with failure rates of ~5–10%, fecal carriage rates of <3%,and relapse rates of 3–6%.Oral azithromycin results in defervescence in 4–6 days, with rates of relapse and convalescent stool carriage of <3%. Despite efficient in vitro killing of Salmonella, first-and second-generation cephalosporins as well as aminoglycosides are ineffective in treating clinical infections. Patients with persistent vomiting, diarrhea, and/or abdominal distension should be hospitalized and given supportive therapy as well as a parenteral third-generation cephalosporin or fluoroquinolone, depending on the sus ceptibility profile. Therapy should be administered for at least 10 days or for 5 days after fever resolution.

In a randomized, prospective, double-blind study of critically ill patients with enteric fever (i.e., those with shock and obtundation) in Indonesia in the early 1980s, the administration of dexamethasone (3-mg initial dose followed by eight doses of 1 mg/kg every 6 h) with chloramphenicol was associated with a substantially lower mortality rate than treatment with chloramphenicol alone (10% vs 55%). Although this study has not been repeated in the "post-chloramphenicol era," severe enteric fever remains one of the few indications for glucocorticoid treatment of an acute bacterial infection.

The 1–5% of patients who develop chronic carriage of Salmonella can be treated for 4–6 weeks with an appropriate oral antibiotic. Treatment with oral amoxicillin, trimethoprim-sulfamethoxazole (TMP-SMX), ciprofloxacin, or norfloxacin is ~80% effective in eradicating chronic carriage of susceptible organisms. However, in cases of anatomic abnormality (e.g.,biliary or kidney stones), eradication often requires both antibiotic therapy and surgical correction.

All in all, theoretically, it is possible to eliminate the salmonellae that cause enteric fever since they survive only in human hosts and are spread by contaminated food and water. However, given the high prevalence of the disease in developing countries that lack adequate sewage disposal and water treatment, this goal is currently unrealistic. Thus travelers to developing countries should be advised to monitor their food and water intake carefully and to consider vaccination.

REFERENCE:

1. CENTERS FOR DISEASE CONTROL AND PREVENTION: Preliminary Bacterial Infections FoodNet data on the incidence of infection with pathogens transmitted commonly through food—10 states, 2008. MMWR Morb Mortal Wkly Rep 58:333,2009
2. COHEN JI et al: Extra-intestinal manifestations of Salmonella infections. Medicine 66:349, 1987
3. FRASER A et al: Vaccines for preventing typhoid fever. Cochrane Database Syst Rev Jul 18(3):CD001261, 2007
4. GLYNN MK et al:Emergence of multidrug-resistant Salmonella enterica serotype typhimurium DT104 infections in the United States. N Engl J Med 338:1333,1998
5. HOFFMAN SL et al: Reduction in mortality in chloramphenicol treated severe typhoid fever by high-dose dexamethasone. N Engl J Med 310:82, 1984
6. LIN FY et al:The efficacy of a Salmonella typhi Vi conjugate vaccine in two-to-five-year-old children. N Engl J Med 344:1263, 2001
7. MASKEY AP et al:Salmonella enteric serovar Paratyphi A and S. enterica serovar Typhi cause indistinguishable clinical syndromes in Kathmandu, Nepal.Clin Infect Dis 42:1247, 2006
8. OHL ME, MILLER SI: Salmonella:A model for bacterial pathogenesis. Annu Rev Med 52:259,2001
9. STEINBERG EB et al: Typhoid fever in travelers: Who should be targeted for prevention. Clin Infect Dis 39:186, 2004
10. SU LH et al: Antimicrobial resistance in nontyphoid Salmonella serotypes:A global challenge. Clin Infect Dis 39:546, 2004
11. VARMA JK et al: Antimicrobial-resistant nontyphoidal Salmonella is associated with excess bloodstream infections and hospitalizations. J Infect Dis 191:554, 2005