Splenic Abscess Caused by *Shigella flexneri* and *Bacteroides fragilis*

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We report a case of splenic abscess from which only *Shigella flexneri* (serotype 1a) and *Bacteroides fragilis* were isolated. The patient was a 59-year-old diabetic female who displayed minimal gastrointestinal symptoms. The *S. flexneri* isolate was resistant to chloramphenicol and tetracycline. The possible synergistic relationship between the two organisms is discussed.

Splenic abscesses are uncommon and occur primarily in patients with sickle cell hemoglobinopathies, trauma, bacteremia, and compromised host defenses. Mortality rates in patients with splenic abscesses, have ranged from 40 to 100% (1). There has been only one previous report of a splenic abscess caused by *Shigella flexneri*, and it occurred in a 13-year-old boy with classic symptoms of shigellosis (6). We report here a case of splenic abscess caused by *S. flexneri* and *Bacteroides fragilis* in a patient with minimal gastrointestinal symptoms. A previously healthy 59-year-old diabetic female was admitted with complaints of abdominal pain, nausea, and vomiting of approximately 12 h duration. Physical examination revealed the patient to have moderate upper-left-quadrant abdominal tenderness. The stool was guaiac positive. Her hemoglobin was 7.7 g/dl, her hematocrit was 26% and her leukocyte count was 16.6 × 10^3/µl. The differential leukocyte count showed 76% neutrophils, 2% bands, 22% lymphs. Chemistry tests on admission were unremarkable except for glucose, which was 349 mg/dl. On the evening of admission, the patient developed a temperature of 38.4°C, and the leukocyte count rose to 23 × 10^3/µl. Abdominal computerized tomograph scan revealed a collection of fluid suggestive of purulent material in the left upper quadrant. On the fifth hospital day, the patient underwent an exploratory laparotomy. A large encapsulated splenic abscess almost replacing the spleen was found, and it contained approximately 300 ml of foul-smelling purulent material. The spleen was removed, and the patient was placed on antibiotics consisting of 2 g of cefamandole every 6 h, 100 mg of tobramycin every 8 h, and 600 mg of clindamycin every 6 h for 7 days postoperation, at which time her fever had resolved and her leukocyte count had returned to normal. There was no evidence of direct spread of organisms from the colon, which was grossly normal at operation. Stool cultures performed postoperation were negative. We presume that the spleen was seeded via the bloodstream. Splenic tissue and aspirated fluid were sent to the laboratory for culture. The postoperative course of the patient was uncomplicated.

Routine blood, stool, and urine cultures were negative. The splenic material removed at surgery was plated to sheep blood agar plates, anaerobic brucella agar plates, chocolate agar, MacConkey agar, and supplemented thioglycolate broth. Direct smears made from the splenic material revealed gram-negative rods. The aerobic cultures grew only *S. flexneri* (serotype 1a); *B. fragilis* was isolated from the anaerobic cultures. Identification of the shigella was based on the following reactions: triple sugar iron agar, alkaline over acid; lysine iron agar, alkaline over acid; and urea slant, negative. API (Analytab Products, Plainview, N.Y.) profile number 0004100 identified the organism as *S. flexneri*, and this identification was confirmed by serotyping. The *S. flexneri* serotype was determined by the Texas Department of Health. A Bauer-Kirby disk diffusion test revealed that the organism was susceptible to ampicillin, carbenicillin, cephalothin, gentamicin, kanamycin, tobramycin, and ceftamandole but was resistant to chloramphenicol and tetracycline. The *B. fragilis* isolate was identified by criteria proposed by Sutter et al. (7) and by an API anaerobic test strip. The colonies on blood agar were gray to white, smooth, and entire, and the Gram stain revealed pale pleomorphic gram-negative rods. There was no pigment, pitting, hemolysis, or fluorescence on anaerobic blood agar, and the organism was resistant to penicillin (2-U disk), kanamycin (1,000-µg disk), vancomycin (5-µg disk), and colistin (10-µg disk).

Our patient made frequent trips to Mexico, where she probably became infected with the *S. flexneri*. Splenic abscesses are preceded by infections elsewhere in the body, and these infections serve as a source of seeding of microorganisms. A study by Chun et al. in 1980 (1) reviewed 173 reported cases of splenic abscesses. Culture results were available from 129 patients. *Streptococcus* spp., *Staphylococcus* spp., *Salmonella* spp., and gram-negative rods accounted for 86% of them, whereas anaerobes were reported in only 7 (5.4%) of the cases. *Bacteroides* spp. were the most common anaerobes isolated. Sterile cultures were reported in 37 (28.7%) of the cases. Anaerobic culture techniques have improved markedly over the past 10 years, and inadequate anaerobic culture techniques may have contributed to a low rate of recovery of anaerobes.

It is presumed that the abscess in this patient resulted from bacteremia from mildly symptomatic bacterial enteritis. The anemia could have resulted from the chronic infection associated with the splenic abscess. It is possible that the *B. fragilis* and *S. flexneri* isolated from our patient worked in synergy to form the splenic abscess. Although it was assumed previously that anaerobes were merely commensals, recent experimental and clinical data have shown anaerobes to play a significant role in many infectious processes in general and in abscess formation in particular (3–5, 8). Of the few anaerobes important in human infections, by far the most important is *B. fragilis*. It possesses some unique properties, including its capsule, which has been shown to be an important virulence factor (5). In rat models in which

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*Escherichia coli*, *enterococci*, *Bacteroides* spp., and *Fusobacterium* varium were implanted singly or in various combinations, only rats receiving *E. coli* died. Abscesses developed only in animals receiving *E. coli* or enterococci in combination with either anaerobe (4). Purified *B. fragilis* polysaccharide alone is sufficient to produce sterile abscesses (5).

Our case points up a number of important considerations for dealing with splenic abscesses. (i) Cultures for anaerobes as well as aerobes should be performed routinely. (ii) Broad spectrum antibiotic therapy effective against both aerobes and anaerobes should be utilized. Caution should be employed when using chloramphenicol since there have been reports of clinical failures despite indications that the organisms were susceptible (2). Our *S. flexneri* isolate was initially resistant to chloramphenicol in vitro. (iii) Pathogens such as *S. flexneri*, as well as normal flora, must be considered in cases of splenic abscesses.

LITERATURE CITED