Shigella flexneri bacteremia in a child

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ABSTRACT

We report an unusual and lethal case of *Shigella flexneri* septicemia in an 8-year-old Saudi handicapped child from a social home presenting with severe toxic megacolon and acute abdomen secondary to fulminant necrotizing enterocolitis.

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Infection of extra-intestinal sites by *Shigella species* is rare. However, whenever it occurs, such patients, usually, are either immuno-compromised or they have prior bacillary dysentery or are asymptomatic carriers. Nonetheless, anecdotal cases have been reported, especially from the developing tropical countries. We wish to describe a lethal case of *Shigella flexneri* septicemia who presented with acute abdomen secondary to fulminant necrotizing enterocolitis with toxic megacolon and who improved initially following surgical decompression but finally died.
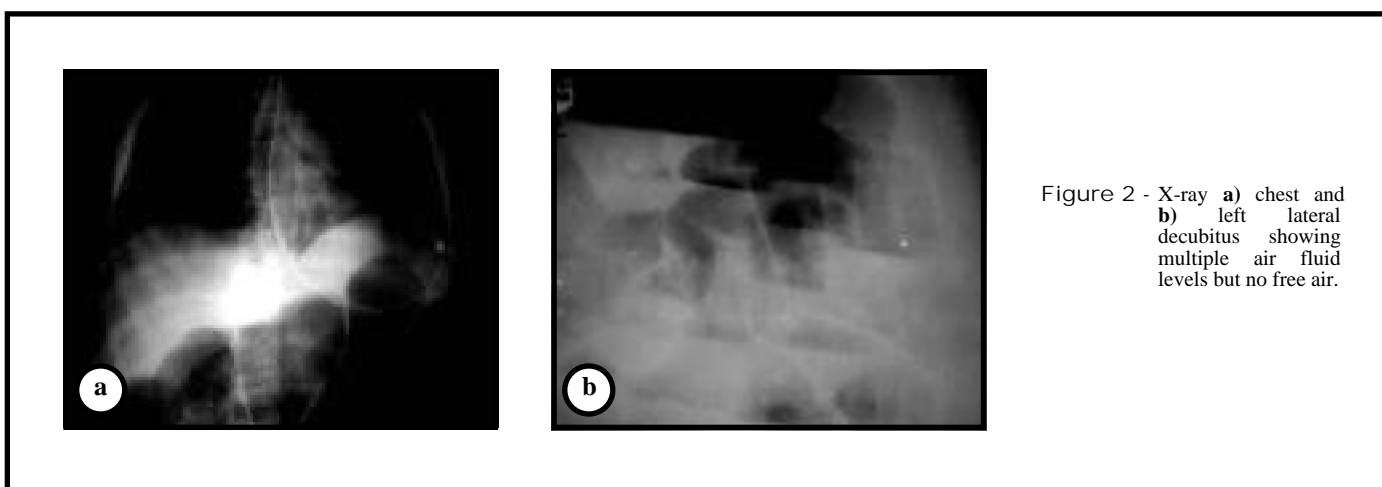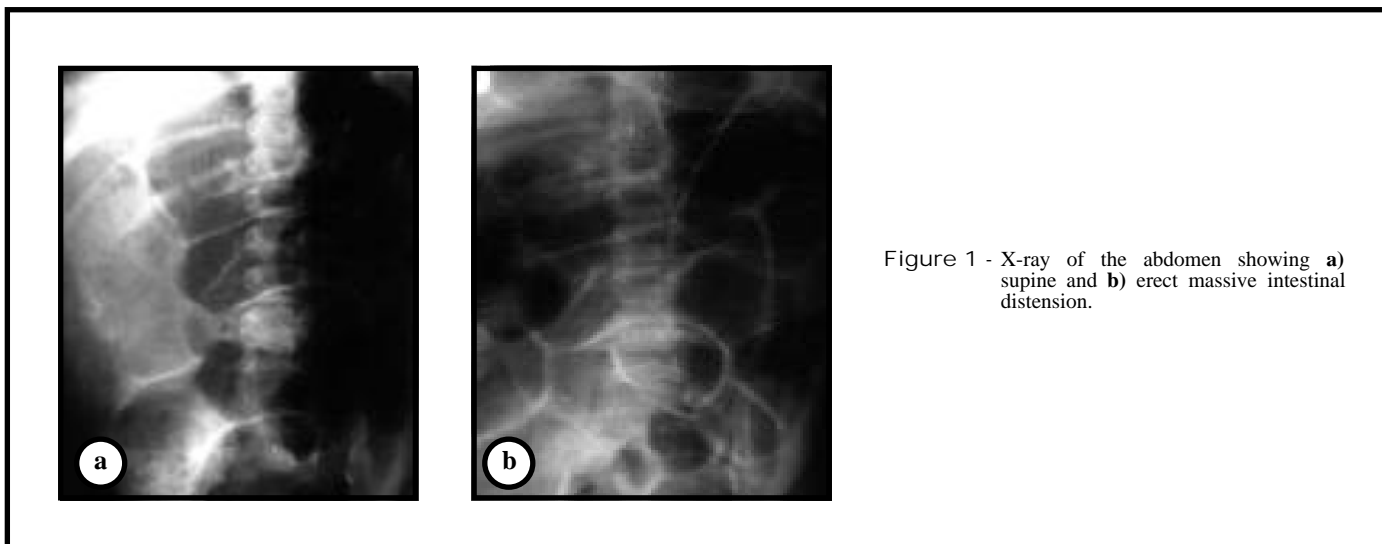
Case Report. An 8-year-old Saudi boy, with mental retardation and cerebral palsy since post-natal period and an inhabitant of one of the social homes, was brought to our Accident and Emergency Department at Assir Central Hospital, Abha, Kingdom of Saudi Arabia with a history of diarrhea of 5 days duration and severe rapidly increasing abdominal distension of one day without any vomiting, fever or blood in the stools. Upon admission to the pediatric intensive care unit, he was unconscious but responsive, breathing spontaneously by an oxygen mask with a thready pulse rate of 120 per minute, blood pressure 106/65 mm Hg, and a rectal temperature of 36.9°C, but with severe dehydration. Abdominal examination showed very tense gross distension with no evidence of ascites, masses or bowel sounds. Per rectal examination showed blood and mucus but no stools. A rectal swab, urine and blood for culture and sensitivity were taken. X-ray chest and abdomen showed massive bowel distention (**Figure 1**), no free air but with few air-fluid levels (**Figure 2**). White blood count 5000/mm^3, hemoglobin 12.7 g/dl, hematocrit 36.4%, platelets 49,000/mm^3, sodium (NA+)/potassium (K+) 107/3.4 mEq/l, BUN 57 mg; calcium++ 7 mg, arterial blood gases, showed compensated metabolic acidosis; serum albumin 2.1 g/dL, enzymes were elevated, prothrombin time/activated partial thromboplastin time was 22/16, 55/31. Fibrin degradation products >1000 mg. Stool cultures were ordered, but patient failed to produce stools. Patient was managed conservatively with nasogastric suction, intravenous fluids, ventilatory support, inotropics and antibiotic cover (ceftriaxone + metronidazole), pending the culture and sensitivity report. When patient did not show any sign of improvement, amikacin was added. On the third day of admission, the patient developed huge abdominal distension with very tense shining abdomen requiring very high ventilatory pressure settings. His platelets dropped to 14,000/mm^3 and coagulation parameters were prolonged. A diagnosis of acute fulminant necrotizing enterocolitis with toxic mega colon was made and surgical decompression was decided after the patient was prepared with platelets and fresh frozen plasma infusions. At exploration, turbid yellowish peritoneal fluid, measuring 500 ml, with

Figure 1 - X-ray of the abdomen showing **a)** supine and **b)** erect massive intestinal distension.

Figure 2 - X-ray **a)** chest and **b)** left lateral decubitus showing multiple air fluid levels but no free air.

massive distension of right colon and terminal ileum with multiple necrotic patches were seen. Liver and spleen were enlarged and gall bladder was distended. Enterotomy decompression, peritoneal toilet and a decompression of the right transverse colostomy with a piece of colon biopsy at colostomy site concluded the procedure. The patient improved markedly in the postoperative period with scaphoid abdomen and started moving the colostomy with bile-stained and mucoid blood discharge on the second postoperative day. The peritoneal fluid culture, swab from the colostomy site plus the repeat blood cultures twice, were all negative. The negative results were ascribed to the bacteriostatic effect of antibiotics. The initial blood culture became positive on day 2 and the organism was subsequently identified as *Shigella flexneri*. It must be emphasized that the patient never passed stools throughout the period of admission. The catheter-specimen urine grew 400 cfu/ml *Candida albicans*. This was considered as colonization. The catheter was removed and the urethra irrigated with sterile saline. The *Shigella flexneri* isolate was sensitive to augmentin, cefoxitin, gentamicin, tobramicin, amikacin, cephalothin, cefotaxime, ceftazidime, ceftriaxone, tetracycline, imipenem, meropenem, ciprofloxacin, piperacillin, aztreonam, and resistant to: Trimethoprim-sulphamethoxazole, Ampicillin, chloramphenicol, and carbenicillin. Suddenly on the 9th postoperative day, the patient started downhill course with florid disseminated intravascular coagulopathy with multiple hemorrhages in the lungs, subcutaneous tissues, venepuncture sites, nasogastric tube, colostomy and per-rectally and continued to deteriorate despite all resuscitative measures. He finally succumbed on the tenth day with multiple organ failure.

Discussion. For a long time, it is believed that *Shigella species* remain confined to the bowel and neither invade the lymphatics nor extend to other organs of the body.[1] However, several reports have now appeared in the literature of *Shigella bacteriuria* in

many parts of the world[2] and of bacteremia from across the continents.[3-5] Despite the intense superficial destructive process in the colonic epithelium that typifies acute *Shigellosis*, bacteremia and disseminated infection are relatively rare. Unlike infections by *Salmonella* typhi and paratyphi, the mechanism of bacteremia by *Shigella species*, remains unclear. When we learnt that this patient had dysentery followed by colostomy, we thought his blood must have been contaminated by this procedure. However, we later discovered that he completed the blood culture 3 days before the colostomy operation. His *Shigella* bacteremia, probably had occurred by a physiological process. When the bowel is obstructed, fluid fills the lumen due to combination of diseased intestinal absorption and increased secretion with seepage of fluid into the bowel wall and eventually into the peritoneum.[6] Vascular compromise along the antimesenteric aspect of distended bowel from rising pressure can lead to relative ischemia and faster translocation of intraluminal bacteria and ingested milk acts as culture media.[7,8] Intestinal flora then penetrate the compromised bowel wall. They proliferate and spill into the peritoneum and blood stream causing bacterial peritonitis and sepsis. Once this occurs, mortality reaches 70%.[9] Virulent *Shigella* and other non-toxigenic invasive *Escherichia coli* strains produce disease after invading the intestinal mucosa, with subsequent multiplication and destruction of the mucosa. *Shigella* infection is superficial and only rarely is there penetration beyond the mucosa. This explains the rarity of obtaining positive blood cultures in *Shigellosis* despite the common occurrence of hyperpyrexia and toxemia. The property of invasiveness of *Shigella* is associated with a mixture of soluble bacterial proteins encoded by a 140-MD plasmid. The traditional drugs for treating *Shigellosis* are: ampicillin, tetracycline, trimethoprim or sulphamethoxazole, and the fluoroquinolones. Unfortunately, several clinical isolates of this organism have developed resistance to these drugs and many more, especially in the developing countries.[10] A recent report quotes 50% of *Shigella flexneri* isolates from this area as resistant to ampicillin, cephalothin and trimethoprim-sulphamethoxazole.[11] This underscores the need for routine antibiotic testing of all isolates. Our patient was started on ceftriaxone and amikacin before our sensitivity report was out. Since our isolate was sensitive to both drugs, no change in therapy was made. The patient suddenly developed disseminated intravascular coagulation (DIC) and pulmonary hemorrhage and died on the 10th day. The staff and inmates of the social home from which the patient was admitted were screened for *Shigellae* and other enteric pathogenic bacteria, but none was positive.

We conclude that though its a rare phenomenon, *Shigella* bacteremia does occur and both Clinicians and Laboratorians should look out for it especially in situations where either the patient is immunocompromised or has had a prior history of dysentery.

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