

Case

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The patient was a 4-year-old male who presented to the emergency room with a 2-h history of vomiting, diarrhea, fever, irritability, and lethargy. The child had gone to sleep on the living room couch at 11 p.m. His grandmother found him on the floor at 3 a.m. covered with feces. When she picked him up to carry him to the bathtub, she noticed he was febrile. She bathed him, and brought him to the emergency room. The patient's medical history was significant for his being in group day care.

In the emergency room, he had two episodes of vomiting. His temperature was 38.9°C, pulse 160 beats per min, and respiratory rate 36/min, and he was noted to be dehydrated. His stool contained bloody streaks, and a methylene blue stain of his feces is shown in Fig. 1. Other laboratory studies included a cerebrospinal fluid examination, done because of his lethargy, which was within normal limits; a peripheral white blood cell count of 13,200/ μ l with 85% neutrophils; a negative blood culture; and a negative stool examination for ova and parasites. Figure 2 shows a MacConkey agar plate culture of the organism recovered from the feces of this patient. Figure 3 shows the biochemical reactions obtained in a triple sugar iron (TSI) tube, a urea-motility-indole (UMI) tube, and a UMI tube with added Kovacs reagent (to detect indole production).

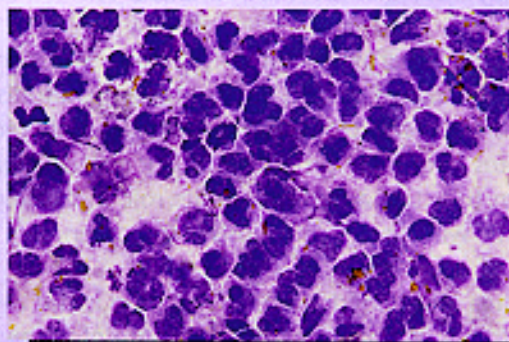


Figure 1

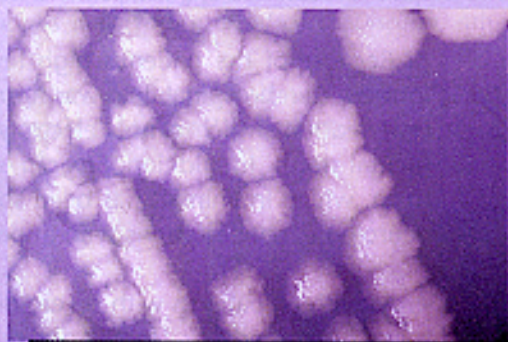


Figure 2

1. Why were white blood cells present in his stool?
2. Given his clinical picture, what bacterial pathogens are likely in this patient? Based on the laboratory results seen in Fig. 2 and 3, what organism is likely causing his illness?
3. What factors contributed to his lethargy?
4. What would be the appropriate treatment strategy for this child?
5. What was the significance of his being in group day care? What special characteristics of this organism lead to its spread?
6. Was it surprising that this patient had a negative blood culture? Explain.

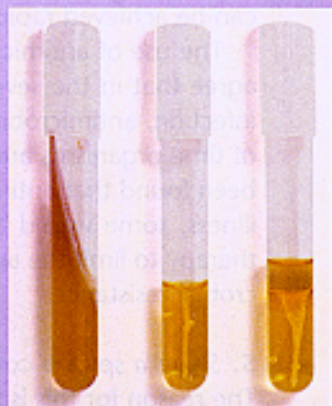


Figure 3

Case Discussion

1. The presence of white blood cells in feces indicates that the patient has an inflammatory type of diarrhea. Inflammatory diarrhea is frequently associated with infection with invasive bacteria and selected protozoans.
2. This patient had a clinical picture consistent with severe invasive diarrhea. Organisms which frequently cause invasive diarrheal disease include *Shigella* spp., *Salmonella* spp., *Campylobacter jejuni*, *Yersinia enterocolitica*, enteroinvasive *Escherichia coli*, and *Entamoeba histolytica*. *E. histolytica* was ruled out on the basis of a negative examination for parasites. The colonial morphology and inability to ferment lactose on MacConkey agar (Fig. 2) are consistent with *Salmonella* and *Shigella* spp. The TSI agar slant shows an organism that does not ferment sucrose or lactose and does not produce H_2S as a product of metabolism. *Salmonella* spp. generally are H_2S positive, making this organism less likely. In the UMI tube, the isolate is nonmotile and urea and indole negative. All three results are typical for *Shigella* spp. In a clinical laboratory further testing could be done to confirm the identity of this organism, but the results seen in Fig. 2 and 3 are consistent with *Shigella* spp. The particular species isolated from this patient was *Shigella sonnei*.
3. Because of the patient's high fever and lethargy, a lumbar puncture was performed. Cerebrospinal fluid was normal, indicating that he did not have meningitis. Dehydration, which causes electrolyte imbalances, can cause lethargy and altered mental status. In addition, *Shigella* isolates frequently produce a toxin, called Shiga toxin, which in animals has been shown to act as a neurotoxin. Its role in causing neurologic manifestations seen in patients with severe shigellosis is unproven.
4. As with all patients with severe diarrhea and dehydration, rehydration is the key therapeutic modality. Usually oral rehydration is used because of its ease of administration and its low cost. Because this child was vomiting, intravenous rehydration therapy was given. Stool volume is relatively low in *Shigella* diarrhea, so rehydration can be achieved rapidly.
The use of antimicrobial agents in diarrheal disease is controversial. Most would agree that in the severely ill child, antimicrobial therapy is appropriate. In *Shigella* infection, antimicrobial therapy shortens the disease course and reduces the length of time organisms are excreted in feces. In the less severely ill patient, where it has been found that antimicrobial therapy may not significantly shorten the duration of illness, some would argue that the infected individual should forgo antimicrobial therapy to limit the antimicrobial pressure on this organism and thus reduce antimicrobial resistance.
5. *Shigella* sp. is a common cause of outbreaks of diarrhea in the day care setting. The reason for this is that shigellae are much more resistant to the low pH found in the stomach than are other enteric pathogens such as *Salmonella* spp. or *Vibrio*

cholerae. As a result, only a small number of organisms (estimated at less than 200 viable bacteria) need be ingested for infection to occur. *Shigella* can spread from person to person by fecally contaminated hands, a common occurrence in the day care setting whether it be a day care worker who does not adequately wash his or her hands after changing the diaper of a *Shigella*-infected toddler or the *Shigella*-infected child touching toys or placing fingers which are fecally contaminated into the mouths of other children. Food- and waterborne outbreaks of *Shigella* infection are uncommon but do occur. Unlike many other enteric pathogens which can infect a variety of domestic animals, e.g. *Salmonella* spp., *Shigella* infects only humans and non-human primates.

6. The finding that this child had a negative blood culture was not surprising. Although the organism is locally invasive, destroying cells in the colonic mucosa, the organism rarely penetrates beyond the lamina propria. Therefore, blood cultures are rarely positive with this organism.

References

1. Dupont, H. L. 1995. *Shigella* species (bacillary dysentery), p. 2033–2039. In G. L. Mandell, J. E. Bennett, and R. Dolin (ed.), *Principles and Practice of Infectious Diseases*, 4th ed. Churchill Livingstone, Inc., New York.
2. Salyers, A. A., and D. D. Whitt. 1994. Dysentery caused by *Shigella* species, p. 169–181. In *Bacterial Pathogenesis: a Molecular Approach*. ASM Press, Washington, D.C.