Especially on the immunoblot, serum levels of IgM antibody to B. burgdorferi were increased early and later decreased, whereas serum IgG antibodies remained negative (table 1). Slightly increased levels of IgM antibodies to herpesviruses and IgG antibodies were also found, however, without time-dependent changes in titers. At a follow-up 18 months after the tick bites, serological results for herpesviruses remained unchanged. Meanwhile the patient had become negative for IgM antibodies to B. burgdorferi. Because of the high risk of thrombosis or thromboembolism due to pregnancy, lumbar puncture was not carried out. Immunofluorescence assay and ELISA of umbilical cord blood from both children were negative for serum IgG and IgM antibodies, as was PCR analysis of placenta tissue for B. burgdorferi.

Pregnant women have an increased risk of developing idiopathic facial nerve palsy (Bell’s palsy) [6]. The distinction between LD-associated facial palsy and Bell’s palsy is important since B. burgdorferi infection must be treated early and appropriately to ensure rapid and complete recovery [7]. Conversely, corticosteroids, which are often prescribed for treatment of Bell’s palsy, are unwarranted and perhaps deleterious in cases of infection [8]. Although Arkansas is not an area where LD is endemic (reported incidence for 1996 was 1.1 cases per 100,000 inhabitants [9]), assessment of the specific bands on the immunoblot for our patient is suggestive of true infection [8] rather than nonspecific positive results due to pregnancy. Early therapy may have prevented an IgG response. The slightly increased levels of IgM antibodies to herpessviruses suggest polyclonal stimulation rather than appearance of an acute or persistent viral infection.

This case reemphasizes the fact that for pregnant women presenting with facial palsy and a history of a recent tick bite the possibility of infection with B. burgdorferi must be taken into consideration.

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References

Fatal Gastrointestinal Mucormycosis That Invaded the Postoperative Abdominal Wall Wound in an Immunocompetent Host

Mucormycosis of the gastrointestinal tract is a rare form of the disease that (because of the vague presentation and rapid progression) is often fatal, with the diagnosis being made only at autopsy. The entire gastrointestinal tract is susceptible to infection. Most commonly, initial presentation involves severe abdominal pain and distention, nausea and vomiting, and occasionally fever and hematochezia. Predisposing factors include malnutrition, carcinoma, uremia, hematologic malignancy, abdominal trauma, and deferoxamine therapy [1]. Diabetes rarely predisposes to this form of infection [1]. Approximately one-third of all reported cases of gastrointestinal mucormycosis occur in children [2]. Gastrointestinal mucormycosis in healthy patients without predisposing conditions is extremely rare; we describe an immunocompetent man with fatal gastrointestinal mucormycosis that invaded the postoperative abdominal wall wound.

A 54-year-old man presented to the emergency department complaining of constipation for 1 month. He had no significant medical history. He was afebrile and hypotensive with mild abdominal tenderness but no rebound or guarding. Laboratory and radiological tests were unremarkable. Testing for HIV was negative. The patient was admitted to the hospital for treatment with fluids and antibiotics.

The next day, the patient underwent cardiopulmonary arrest. The abdomen became distended and tense, and laparotomy was promptly performed. Extensive ischemia and necrosis of all except the proximal 4 ft of the small bowel was demonstrated, and small bowel resection was performed. Examination of the resected bowel revealed areas of transmural necrosis with hemorrhage interspersed within the normal bowel. Examination of the infarcted areas showed branching, nonseptate, broad, ribbonlike hyphae consistent with fungi causing mucormycosis. The organisms were seen within the vascular walls and lumina.

Liposomal amphotericin B (5 mg/[kg · d] intravenously) was started. Three days later, mold was noted to be growing from the surgical wound (figure 1); cultures of wound scrapings and wound packing confirmed the spread of Rhizopus to the abdominal wall. Despite repeated surgical debridements, the wound progressed to become dark and necrotic with continued growth of Rhizopus. The patient died on hospital day 25.

The Mucorales are vasculotropic fungi that cause extensive tissue infarction, preying on immunocompromised debilitated hosts. Diagnosis is made by histological identification of broad (10–20 μm), nonseptate hyphae with right-angled branches in resected necrotic tissue with evidence of tissue invasion [1].

Gastrointestinal mucormycosis in healthy patients without any predisposing factors is extremely rare. The only two reported cases...
have involved the distal small intestine [3] and the sigmoid colon [4]. Both patients presented with acute abdominal pain and distention, and despite combined medical and surgical intervention, both patients died.

The case presented here is remarkable on two counts. The patient had no identified predisposing cause for the development of this invasive opportunistic infection. However, the second, and to the best of our knowledge previously unreported, aspect of this case was the spread of \textit{Rhizopus} to involve the abdominal wall surgical wound. Although cases of abdominal wall necrosis following abdominal trauma and routine surgery have been reported, all were described as "necrotizing fasciitis" with no evidence of clinically visible mold [5]. We propose that in the case presented, the patient’s severely debilitated and catabolic postoperative state promoted the growth of \textit{Rhizopus}, which spread rapidly and aggressively from the intraabdominal cavity externally to invade the abdominal wound. Not surprisingly, the patient rapidly died.

Early diagnosis and treatment of gastrointestinal mucormycosis may result in survival from this otherwise deadly disease [6]. Early and aggressive combined medical and surgical intervention including resection of the infected nonviable areas is required. Frequently, antifungal treatment is often instituted only once histological confirmation of mucormycosis is made, usually several days following surgical intervention and frequently too late to alter the clinical course of this serious infection. On the basis of this case, clinicians should view the growth of mold in the postoperative wound in cases of gastrointestinal mucormycosis as an event portending an extremely dismal prognosis.

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\textbf{Figure 1.} Abdominal wound of a patient with fatal gastrointestinal mucormycosis on postoperative day 3. Note extensive fuzzy mold (\textit{Rhizopus}) invading the wound (arrow).