Cryptosporidiosis was first reported in humans in 1976 [4]. Since then, cryptosporidiosis has been recognized as a common gastrointestinal-tract infection in humans, primarily among those infected with HIV [5]. In immunocompetent hosts Cryptosporidium causes a self-limited diarrheal illness, lasting from 4–20 days [6]. Generally the clinical course is relatively benign, although protracted and severe cases have been reported. Several reports describe a variety of extraintestinal manifestations for patients with AIDS; however, in contrast, such reports are rare for immunocompetent hosts. Indeed, we found only one report of pancreatitis associated with Cryptosporidium [7].

There are several possible sources of infection for our patient. First, a pet dog, which had been boarded at a kennel, had had watery diarrhea several days before the onset of the patient’s symptoms. Although no confirmed cases of Cryptosporidium transmission from infected household pets to humans have been reported, there are reports of episodes in which infected cats were found in the households of immunocompromised individuals with cryptosporidiosis [8]. In addition, a study conducted at an animal shelter documented that 10% of the puppies examined were shedding cryptosporidia oocysts [9]. A fecal sample from our patient’s dog, obtained 5 weeks after the dog’s illness, tested negative for Cryptosporidium antigen by use of ELISA. An alternative source of infection for our patient is person-to-person transmission; the patient’s friend had a self-limited diarrheal illness before our patient developed symptoms. Finally, the patient may have acquired the parasite through contaminated food or water.

We assert that this patient had acute pancreatitis on the basis of the following clinical criteria: severe upper abdominal pain, nausea, and emesis in the setting of elevated serum amylase and lipase levels. Although a contrast-enhanced CT scan of the abdomen did not reveal evidence of pancreatic inflammation, the diagnosis of acute pancreatitis should not be excluded [10]. Rather, absence of findings on the CT scan indicates a self-limited pancreatitis caused by transient pancreatic edema. This explanation is consistent with our patient’s clinical course.

In conclusion, C. parvum generally causes a relatively benign, self-limiting diarrheal illness in immunocompetent individuals. However, the diagnosis of cryptosporidiosis should be considered, and an ELISA for antibodies to Cryptosporidium should be undertaken for patients with diarrhea and acute pancreatitis.

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A Case of Endocarditis with Vasculitis Due to Actinobacillus actinomycetemcomitans: A 16S rDNA Signature for Distinction from Related Organisms

Actinobacillus actinomycetemcomitans, a fastidious gram-negative coccobacillus that is part of the indigenous flora of the oral cavity, may be significant in the pathogenesis of juvenile local periodontitis [1]. Renal involvement is noted among ~26% of patients with endocarditis due to A. actinomycetemcomitans [2]. To our knowledge, we describe the second case of endocarditis due to A. actinomycetemcomitans associated with vasculitis and glomerulonephritis [3].

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Clinical Infectious Diseases 1998;27:224–5
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A 54-year-old female was admitted to our hospital for evaluation of fever, night sweats, and fatigue. Perinuclear antineutrophil cytoplasmic autoantibody (ANCA) positive systemic vasculitis and glomerulonephritis had been diagnosed several months earlier. The patient’s current treatment regimen consisted of prednisolone, 50 mg/d, and cyclophosphamide, 150 mg/d. At the time of admission, she appeared critically ill. Physical examination revealed a 1/6 tricuspid systolic murmur and splenomegaly. Laboratory results revealed signs of moderate anemia, thrombopenia, and leukopenia, and the C-reactive protein level was 12.7 mg/dL. There was no history of recent dental procedures, travelling abroad, iv drug abuse, peripheral edema, or weight loss. Cultures of blood obtained at admission yielded gram-negative coccobacillary rods after 5 days; identification was not achieved by use of biochemical means. Echocardiography revealed a vegetation on the tricuspid valve. Treatment with iv imipenem, 0.5 g t.i.d., and iv gentamicin, 240 mg q.d., was instituted, and 3 days later the patient was afebrile.

 Sequencing of amplified 16S rDNA fragments was undertaken to identify the pathogen [4]. Similarity searches by use of the basic local alignment search tool (BLAST) program were accomplished with query sequences of various lengths ≈530 bases [5]. All of
the five highest-scoring homologous sequences were derived from various isolates of \textit{A. actinomycetemcomitans}. Principally, a signature sequence of only 26 bases (\texttt{ttaacacatgcaagtcggacggtagc}), corresponding to positions 48 through 73 of the \textit{Escherichia coli} gene was required to distinguish \textit{A. actinomycetemcomitans} from other \textit{Actinobacillus} species, \textit{Haemophilus} species, and other unusual gram-negative bacteria such as Pasteurellaceae. An advantage of this approach over other recently described molecular-identification techniques for \textit{A. actinomycetemcomitans} is the applicability to many other bacteria without preliminary classification [6, 7]. Members of the \textit{Haemophilus}, \textit{Actinobacillus}, \textit{Capnocytophaga}, \textit{Eikenella}, and \textit{Kingella} (HACEK) group of bacteria comprise \(\sim 3\)% of isolates causing community-acquired infective endocarditis [8].

In the present case, it remains unclear whether immunosuppressive therapy predisposed to infection with \textit{A. actinomycetemcomitans} or whether subclinical \textit{A. actinomycetemcomitans} infection preceded and possibly precipitated the development of vasculitis.

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