characteristic yellow colonies, which were the only type of organism identified. Cultures were negative for Salmonella, Shigella, Campylobacter, and Escherichia coli O157:H7.

Antimicrobial susceptibility testing for the isolate revealed susceptibility to aminoglycosides, fluoroquinolones, trimethoprim-sulfamethoxazole, and tetracycline and resistance only to ampicillin. The patient was treated with a single 1-g oral dose of ciprofloxacin and subsequently reported complete resolution of his diarrhea.

V. alginolyticus has only rarely been associated with acute diarrheal illness [2–8]. In the seven studies, a total of 15 cases of diarrhea due to V. alginolyticus were reported from 1980 to 1995. In four of these 15 cases, another enteric pathogen along with V. alginolyticus, including Campylobacter, Shigella, E. coli, and Vibrio parahaemolyticus, was isolated, suggesting that V. alginolyticus may not have even been the causal agent in these cases [5–7]. In an additional eight cases, it was not specified whether V. alginolyticus was the sole organism recovered [3, 4]. Only three prior cases clearly demonstrate pure isolation of V. alginolyticus from a patient with acute diarrhea [2, 6, 8]. Chronic diarrhea was not described in any of the 15 cases. A MEDLINE search of the English-language literature from 1966 to 1999 revealed no reports of chronic diarrhea associated with V. alginolyticus.

Our case is also of interest because it involved a homosexual man who was immunocompromised secondary to AIDS. This situation raises the question of whether patients with AIDS or homosexual males are at increased risk of developing V. alginolyticus infections. An AIDSLINE search of the English-language literature from 1980 to 1999 revealed no other case reports of such infections. There have, however, been several case reports of gastroenteritis caused by other Vibrio species in patients with AIDS. One report described acute diarrhea caused by Vibrio vulnificus in a man with AIDS [9], and another discussed a case of sepsis, peritonitis, and gastroenteritis in a homosexual man with AIDS that was found to be caused by Vibrio vulnificus [10]. In both of these cases, the patients had eaten seafood (scallops and raw oysters, respectively) prior to symptom onset, suggesting that the infections were food-borne.

Acalculous Cholecystitis Associated with Plasmodium falciparum Infection

Acute acalculous cholecystitis (AAC) has been described in association with various infectious agents [1–3]. Although gastrointestinal manifestations are not uncommon in malaria [4], AAC as a complication of this infection has not been previously reported. We report the first case of AAC attributed to an acute infection with Plasmodium falciparum.

A 26-year-old female was admitted to the hospital with a 5-day history of abdominal pain, nausea, vomiting, fever, and chills. She had been seen elsewhere 2 days previously and was diagnosed with gastroenteritis. She was told to take acetaminophen, but when her symptoms persisted, she came to the hospital. At admission, she was lethargic but capable of being aroused with a temperature of 39.5°C, pulse rate of 120, and blood pressure of 80/35 mm/Hg. The neck was supple, and no rashes were present. Results of chest and cardiovascular examinations were normal. The abdomen was diffusely tender with rebound in the right upper quadrant.

Laboratory investigations showed a WBC count of 4.5 × 10^9/L, hemoglobin level of 104 g/L, and platelet count of 56 × 10^9/L. The total bilirubin concentration was elevated to 60 mmol/L (indirect bilirubin level, 51 mmol/L). Transaminase and alkaline phosphatase values were normal. Abdominal ultrasonography was performed and revealed a thickened gallbladder wall (5 mm) surrounded by a thin rim of fluid. No stones were visible, and a diagnosis of acalculous cholecystitis was made. The patient was treated with intravenous fluids and broad-spectrum antibiotics, and an emergency cholecystectomy was scheduled. However, on further questioning, it was determined that the patient had returned from Togo, Africa, 2 weeks before hospitalization.

It was her first trip home in 10 years, and she had stayed there for 2.5 weeks but did not take any prophylaxis. Examination of a

References

The daily urinary output fell to 400 mL.

Cidofovir dosage was reduced to 2.5 mg/kg according to protocol. The presence of proteinuria (protein level in urine, 1 g/L); the Normoglycemic glycosuria developed after the third cycle [2].

decreased transiently after each cidofovir administration (figure 1). Indinavir was substituted by ritonavir. The creatinine clearance necid (4 g) and intravenous normal saline (2 L). At the same time, cidofovir (5 mg/kg once every 2 weeks) with concomitant oral probe-

included Kaposi's sarcoma and hypertension treated by nifedipine. Triple nucleoside analogues were started. Treatment with quinine and doxycycline was started. Over the next few days, the patient’s condition improved clinically, with resolution of abdominal pain and normalization of her laboratory and ultrasonography findings. Cultures of blood obtained before the start of antibiotic therapy remained negative.

The clinical presentation of falciparum malaria may include gastrointestinal symptoms such as nausea, vomiting, and diarrhea [4]. Severe abdominal pain mimicking an acute abdomen is unusual and can lead to a delay in the diagnosis [5]. In this case, results of physical examination and ultrasonography were compatible with AAC, and the surgeon was prepared to perform cholecystectomy. The usual management of AAC is cholecystectomy, but when the diagnosis of falciparum malaria was made, surgery was postponed. Medical treatment of malaria resulted in resolution of the cholecystitis.

The pathogenesis of AAC is believed to be a combination of bile stasis and gallbladder ischemia [1]. Our patient had a period of hypotension along with hemolysis that may have resulted in injury to the gallbladder. Although other infectious agents have been reported to cause AAC, we were unable to find a previous case report of malaria associated AAC in our search of the English-language medical literature. Falciparum malaria should be added to the list of infectious causes of AAC.

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Cidofovir-Induced End-Stage Renal Failure

Cidofovir, previously known as HPMPC, is a nucleotide analogue of cytosine with potent and prolonged activity against herpesviruses and polyomaviruses including JC virus, the etiologic agent of progressive multifocal leukoencephalopathy [1]. To our knowledge, we report the first case of subacute tubulointerstitial nephritis caused by cidofovir that led to end-stage renal failure.

A 64-year-old HIV-positive man was diagnosed with progressive multifocal leukoencephalopathy (established by brain biopsy) in March 1998. His CD4 cell count was 180 × 10⁶/L, and his HIV RNA load was <400 copies/mL. The serum creatinine level was 1.2 mg/dL. Urinalysis was unremarkable. He had been taking cotrimoxazole prophylaxis since 1993 and stavudine, didanosine, nevirapine, and indinavir treatment since April 1997. His medical history included Kaposi’s sarcoma and hypertension treated by nifedipine.

On 24 April 1998, treatment was started with intravenous cidofovir (5 mg/kg once every 2 weeks) with concomitant oral probenecid (4 g) and intravenous normal saline (2 L). At the same time, indinavir was substituted by ritonavir. The creatinine clearance decreased transiently after each cidofovir administration (figure 1). Normoglycemic glycosuria developed after the third cycle [2]. After the fourth cycle, the serum creatinine level was 1.8 mg/dL. In the presence of proteinuria (protein level in urine, 1 g/L); the cidofovir dosage was reduced to 2.5 mg/kg according to protocol. The serum creatinine level rose to 3.2 mg/dL 4 days later (all medications were withdrawn) and to 9.9 mg/dL within 3 weeks. The daily urinary output fell to 400 mL.

References

Figure 1. Evolution of creatinine clearance (mL/min; •) calculated according to the Cockcroft-Gault formula and of proteinuria (dipstick test result, 0 or +; g/d; ○) in a patient with cidofovir-induced end-stage renal failure. B = renal biopsy; S = steroid administration.

Urinary sediment remained normal with glycosuria (glucose level in urine, up to 4.5 g/L) and proteinuria (protein level in urine, up to 3.9 g/d). Renal ultrasound examination was unremarkable. On 3 July 1998, renal biopsy showed diffuse incipient interstitial fibrosis with focal tubular atrophy. Interstitial edema was associated with moderate to severe mixed inflammatory infiltration, extensive acute tubular epithelial cell degeneration, and focal tubulitis. There were no glomerular lesions. Treatment with prednisone was ineffective. Hemodialysis was started and is still required.

The close temporal relationship between the onset of renal failure and cidofovir administration and the transient deterioration in renal function that was observed after each cycle strongly support the causative role of cidofovir in our case.