Acute Cholecystitis Complicating Mumps

Sir—We describe a case of mumps complicated by hepatitis and acute acalculous cholecystitis. There are few published reports of hepatitis complicating mumps infection, and no previous reports of cholecystitis complicating mumps infection.

A 23-year-old man presented with a 1-week history of fever, acute bilateral parotitis, and orchitis, followed by 24 h of abdominal pain and vomiting. Several members of his soccer team had recently had mumps. He had never received mumps vaccine. He was febrile, with a temperature of 38.8°C, with a palpable, tender liver edge, and positive Murphy’s sign. Initial blood test results showed a WBC count of 13,100 cells/mm³, a neutrophil count of 9500 cells/mm³, and a lymphocyte count of 1400 cells/mm³. He had a C-reactive protein level of 248 mg/L, a bilirubin level of 1.05 mg/dL, an alanine aminotransferase level of 160 IU/L, an alkaline phosphatase level of 480 IU/L, a γ-glutamyl transferase level of 494 IU/L, and an albumin level of 45 g/L. His blood urea nitrogen, creatinine, electrolyte, and serum amylase levels were all within normal limits, and blood cultures showed no bacterial growth at 5 days.

The patient was given supportive treatment with analgesics and intravenous fluids. During the next few days, his bilirubin and alanine aminotransferase levels rose to 3.63 mg/dL and 432 IU/L, respectively. IgM against hepatitis A, C, and E; hepatitis B surface antigen; and IgG against cytomegalovirus were not detected. IgG against Epstein-Barr virus nuclear antigen was detected, which indicated previous Epstein-Barr virus infection. Tests for antinuclear antibody; antineutrophil cytoplasmic antibody; rheumatoid factor; complement C3 and C4; immunoglobulins; and smooth muscle, mitochondria, and liver and kidney microsomal antibodies all had normal results. Abdominal ultrasound showed a normal sized liver of decreased echogenicity, no biliary dilatation, and a distended, thick-walled edematous gallbladder that was tender on direct scanning, in keeping with a diagnosis of acute acalculous cholecystitis (figure 1). The patient made a full clinical recovery. Two weeks later, his C-reactive protein levels and liver biochemistry values had returned to normal, and IgM and IgG against mumps virus were detected, which confirmed recent mumps infection.

Mumps, measles, and rubella vaccine was introduced in the United Kingdom in 1988 for children 12–15 months old, and in 1996, a pre-school booster dose was added to the schedule. However, a cohort of young adults born between ~1982 and 1992, who received only 1 or no doses of the vaccine, and who did not acquire natural immunity through infection with circulating wild-type virus, remain susceptible to infection. The current UK mumps epidemic predominantly affects this cohort [1].

Common complications of mumps include orchitis and meningitis; less commonly, oophoritis, pancreatitis, arthritis, mastitis, thyroiditis, and myocarditis may occur. The relative frequency of compi-
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Symptomatic Schistosoma mansoni Infection as an Immune Restoration Phenomenon in a Patient Receiving Antiretroviral Therapy

Sir—We describe a case of symptomatic Schistosoma mansoni–associated enteritis presenting as an immune reconstitution inflammatory syndrome in an African man receiving antiretroviral therapy (ART).

A 36-year-old South African man received the diagnosis of HIV-1 infection in 1990. He had moved to the United Kingdom in 1988 and had made no visits to Africa in subsequent years. In November 2002, his CD4+ cell count was 170 × 10^6 cells/L, with an HIV load of 49,700 copies/mL. ART (with tenofovir, abacavir, zidovudine, and lamivudine) was initiated. After 4 weeks, his viral load decreased to 1330 copies/mL, and his CD4+ cell count increased to 230 × 10^6 cells/L. Two weeks later, he developed fever, vomiting, diarrhea, and abdominal pain. Attributing these symptoms to his medication, he stopped his ART and experienced prompt symptomatic improvement. The gastrointestinal symptoms recurred 3 weeks after reinitiation of ART. During the next 2 years, his symptoms led him to stop treatment on 5 occasions; each time, the temporal association between egg excretion and immune status supports the hypothesis that this is an immune restoration phenomenon [1, 2]. The inability to generate granulomas is a well-described consequence of advanced HIV infection [3]. Evidence suggests that this inability may occur in patients coinfected with S. mansoni and HIV. In animals infected with schistosomes, functional immune responses are required to generate granulomas and thus to efficiently discharge schistosome eggs across the gut mucosa [4]. A study of S. mansoni in western Kenya showed evidence of immune-facilitated excretion of schistosome eggs from patients with S. mansoni–HIV coinfection [5]. The efficiency of fecal egg excretion decreased as the peripheral CD4+ cell count decreased. A Zambian study of Schistosoma haematobium infection also showed reduced egg excretion and fewer reports of hematuria in those who were coinfected with HIV [6]. Although not all studies have replicated these findings [7, 8], this association between egg excretion and immune status supports the concept that immune reconstitution could restore antischistosomal granulomatous responses in the mucosa and lead to symptomatic enteritis, as seen in this patient. The increasing availability of ART in areas of the world where both schistosomiasis and HIV are endemic raises the possibility that Schistosoma–associated morbidity may be seen more commonly.

Schistosoma eggs are highly immunogenic; granuloma formation around the ova is the characteristic histopathological appearance and is responsible for both the long-term fibrotic sequelae in the liver and the gastrointestinal symptoms seen in patients with chronic S. mansoni infection. This patient must have acquired S. mansoni infection at least 14 years previously, but he only experienced symptoms after initiation of ART. The recurrent temporal association between his symptoms and his ART regimen, together with the florid inflammatory response visualized at colonoscopy, supports the hypothesis that this is an immune restoration phenomenon [1, 2]. The inability to generate granulomas is a well-described consequence of advanced HIV infection [3]. Evidence suggests that this inability may occur in patients coinfected with S. mansoni and HIV. In animals infected with schistosomes, functional immune responses are required to generate granulomas and thus to efficiently discharge schistosome eggs across the gut mucosa [4]. A study of S. mansoni in western Kenya showed evidence of immune-facilitated excretion of schistosome eggs from patients with S. mansoni–HIV coinfection [5]. The efficiency of fecal egg excretion decreased as the peripheral CD4+ cell count decreased. A Zambian study of Schistosoma haematobium infection also showed reduced egg excretion and fewer reports of hematuria in those who were coinfected with HIV [6]. Although not all studies have replicated these findings [7, 8], this association between egg excretion and immune status supports the concept that immune reconstitution could restore antischistosomal granulomatous responses in the mucosa and lead to symptomatic enteritis, as seen in this patient. The increasing availability of ART in areas of the world where both schistosomiasis and HIV are endemic raises the possibility that Schistosoma–associated morbidity may be seen more commonly.