cations varies with age [2]. The patient we describe had orchitis, hepatitis, and acalculous cholecystitis. Although hepatitis is a recognized complication of other paramyxovirus infections, such as measles, there are few previous case reports of hepatitis complicating mumps infection [3]. Among 2482 reported mumps cases in the pre-vaccination era, only 1 was complicated by hepatitis [2]. This is the first report of acute cholecystitis complicating mumps infection. Acalculous cholecystitis has been reported as complicating acute hepatitis A virus infection, in association with direct viral invasion of the biliary epithelium [4]. A similar pathological process may have occurred in this case. It is possible that a different spectrum of complications may occur in the current mumps epidemic in the United Kingdom because of the increased burden of disease among adults.

Acknowledgments

Potential conflicts of interest. All authors: no conflicts.

A. J. Brent,1 R. Hull,1 K. J. M. Jeffery,1 R. R. Phillips,2 and B. Atkins1
1Nuffield Department of Infectious Diseases & Microbiology, John Radcliffe Hospital, and 2Department of Radiology, Churchill Hospital, Headington, Oxford, United Kingdom

References


Reprints or correspondence: Dr. Andrew J. Brent, Nuffield Dept. of Infectious Diseases & Microbiology, Level 7, John Radcliffe Hospital, Headington, Oxford OX3 9DU, United Kingdom (andrew.brent@doctors.org.uk).

Clinical Infectious Diseases 2006;42:302–3 © 2005 by the Infectious Diseases Society of America. All rights reserved. 1058-4838/2006/4202-0026$15.00

Symptomatic Schistosoma mansoni Infection as an Immune Restoration Phenomenon in a Patient Receiving Antiretroviral Therapy

Str—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 × 10⁶ 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 × 10⁶ 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 ART and the gastrointestinal symptoms was demonstrated. The findings of microscopic evaluation of stool specimens were normal on several occasions.

Colonoscopy revealed proctitis and patchy sigmoid colitis. Mucosal biopsies demonstrated numerous live and dead S. mansoni ova, with florid eosinophilic granulomatous reactions surrounding the live eggs. Schistosomal antibody ELISA results were positive. The patient was given praziquantel (40 mg/kg), and his symptoms resolved over the next 6 weeks. He remains free of gastrointestinal symptoms 2 years later. His most recent CD4+ cell count was 420 × 10⁶ cells/L.

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 gastroenteritis 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.

CORRESPONDENCE • CID 2006;42 (15 January) • 303
Correspondence

Revisiting Combination Antibiotic Therapy for Community-Acquired Invasive Streptococcus pneumoniae Pneumonia

Sir—In a previously published article [1], we showed that adults ≥50 years of age with invasive Streptococcus pneumoniae pneumonia given combination antibiotic therapy consisting of a macrolide and a β-lactam had a significantly lower case-fatality rate, compared with those given monotherapy with a β-lactam (7% vs. 22%, respectively). Because we did not analyze the clinical comparability of our 2 treatment groups, other investigators who subsequently reported similar findings in invasive pneumococcal pneumonia [2–7] cited our omission as a shortcoming. In this report, we provide that analysis.

For 2 groups of adults with invasive pneumococcal pneumonia, who were aged ≥50 years, were admitted to hospitals from 1978 to 1997, and were treated with combination therapy or monotherapy, data were analyzed for comparability regarding age, severity of illness, and underlying comorbid conditions [1]. Treatment with antibiotics in both groups was started on the day of admission. S. pneumoniae was isolated from blood or pleural fluid samples. Clinical and demographic data were abstracted from the hospital chart. Only deaths that occurred during the first 7 days of hospitalization were counted, unlike in our previous article [1], when all deaths were counted. The institutional review board of Marshall University and of each of the affiliated hospitals approved this research study.

The clinical features of the combination antibiotic therapy group and the monotherapy group were comparable (table 1). They did not differ significantly on the basis of all parameters examined, including age, vital signs at admission, total leukocyte count at admission, number of lobes involved, and preexisting underlying diseases. Both groups had, on average, slight hypothermia, tachycardia and tachypnea, normal blood pressure, and leucocytosis. Mainly, only 1 lobe was involved. Approximately four-fifths of each group had ≥1 underlying comorbid condition(s). The case-fatality rate in the first 7 days of hospitalization was significantly lower in the combination therapy group, compared with the monotherapy group (1.8% vs. 14.5%, respectively; \( P = .009 \), 2-tailed Fisher’s exact test). Two persons in the combination therapy group also received a fluoroquinolone (ciprofloxacin) and neither died. Penicillin resistance was not a factor, because ~94% of persons were infected with penicillin-susceptible strains. Infection with penicillin-resistant strains was equally common in the 2 treatment groups, but no such case was fatal. No patients had HIV or AIDS.

Thus, compared with monotherapy, combination antibiotic therapy effectively lowered the case-fatality rate of invasive pneumococcal pneumonia among ill adults ≥50 years of age. Reason dictates that a β-lactam is the antibiotic of choice for the treatment of invasive pneumococcal pneumonia. Why does use of 2 antibiotics, 1 of which is a macrolide, more effectively lower case-fatality rates in invasive pneumococcal pneumonia than does use of a β-lactam alone? Apparently, it is not because of antibiotic synergy, because treatment with erythromycin and a penicillin or a cephalosporin failed to show synergy in vitro [8]. It is unclear whether a proportion of patients with invasive pneumococcal pneumonia also have a second infection with an atypical pathogen, such as Mycoplasma pneumoniae or Chlamydia pneumoniae, and these pathogens respond to treatment with the macrolide. It is important to know that the best evidence supports use of combination antibiotic therapy, and prospective randomized clinical trials might settle this point [2, 4, 6, 7, 10]. However, until such trials can be performed, it seems reasonable to provide severely ill adults who have community-acquired pneumococcal pneumonia—and especially those who have

Acknowledgments

Potential conflicts of interest. All authors: no conflicts.

S. de Silva, J. Walsh, and M. Brown

1 Mortimer Market Centre, Camden Primary Care Trust, Department of Infectious and Tropical Diseases, London School of Hygiene and Tropical Medicine, and 2 St. Mary’s Hospital, Paddington, London, United Kingdom

References


Reprints or correspondence: Dr. Michael Brown, Dept. of Infectious and Tropical Diseases, London School of Hygiene and Tropical Medicine, Keppel St., London WC1E 7HT, United Kingdom (Michael.brown@lshtm.ac.uk).

Clinical Infectious Diseases 2006;42:303–4 © 2006 by the Infectious Diseases Society of America. All rights reserved. 1058-4838/2006/4202-0027$15.00

304 • CID 2006:42 (15 January) • CORRESPONDENCE