among MRSA isolates from patients with persistent infections, as we and others [7] have found.

With regard to the issue of “low” vancomycin trough serum levels and vancomycin response, raised by Howden et al. [8], the majority of patients in our study [3] were required to have vancomycin trough concentrations of 10 μg/mL. This serum trough concentration was chosen because it was the recommended trough concentration used in the phase III studies, in which vancomycin was a comparator to either quinupristin/dalfopristin or linezolid. Patients with endocarditis, an infected device (e.g., prosthetic device in knee), or a bone or joint infection were required to have a vancomycin trough concentration of 15 μg/mL.

We have previously found lower 24-h AUC/MIC (vancomycin 24-h area under the concentration time curve divided by the MIC) values to be associated with vancomycin treatment failure in hospitalized patients with lower respiratory tract infections [9]. In our study [3], we would expect higher vancomycin MICs to be associated with lower trough-to-MIC ratios for patients with similar infections who had similar trough concentrations. In addition, if the 24-h AUC value was similar for all patients, the 24-h AUC/MIC values would be lower for patients with higher vancomycin MICs. However, appropriate levels for 24-h AUC determination were not obtained for all of the patients investigated. As stated above, patients with a variety of infection types were included in this investigation, and vancomycin trough levels were higher in patients with endocarditis, infected devices, or bone and joint infections, conditions which may be considered more “difficult to treat.” Even though vancomycin trough levels were higher in these patients, the majority of these “difficult-to-treat” infections were considered to be associated with vancomycin treatment failure.

Howden et al. [8] are also curious about the geographic dispersion of the accessory gene regulator (agr) specificity group II isolates. We did not find the agr group II isolates to be from a specific US region, as shown in table 1 (P = .386).

Acknowledgment

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Treating HIV Encephalopathy with Antiretroviral Therapy: A Clinical Case Demonstrating the Success of HAART

Sir—Numerous studies have documented that highly antiretroviral therapy (HAART) successfully prevents HIV encephalopathy. However, little has been reported on the clinical approach for HIV-infected patients who present with HIV encephalopathy. We report successful

Table 1. Incidence of agr group II and non–group II methicillin-resistant Staphylococcus aureus infections by US region.

<table>
<thead>
<tr>
<th>US region</th>
<th>No. of group II agr isolates (n = 52)</th>
<th>No. of non–group II agr isolates (n = 35)</th>
<th>Total no. of isolates (n = 87)</th>
</tr>
</thead>
<tbody>
<tr>
<td>West</td>
<td>2</td>
<td>2</td>
<td>4</td>
</tr>
<tr>
<td>Southwest</td>
<td>2</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td>Midwest</td>
<td>6</td>
<td>1</td>
<td>7</td>
</tr>
<tr>
<td>Northeast</td>
<td>41</td>
<td>30</td>
<td>71</td>
</tr>
<tr>
<td>Southeast</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
</tbody>
</table>

CORRESPONDENCE • CID 2004:39 (15 November) • 1545
A 44-year-old Haitian man presented to the hospital with a 6-month history of progressive mental status changes. At the time of hospitalization, he could only follow simple commands and could not verbally answer questions. His medical history included a diagnosis of mild depression, for which he received citalopram and risperidone therapy.

Pertinent findings of physical examination included the following: the patient was oriented to person only with flat affect and occasional utterances; his oral mucosa were covered with thrush; he had hyperpigmentation on his face; and his sclerae were injected bilaterally. Funduscopic examination revealed “cotton wool” and chorioretinal lesions consistent with HIV retinopathy. The patient’s motor strength was documented as 4/5 in all 4 extremities. He was incontinent, and he was unable to walk without assistance.

Pertinent laboratory values measured at the time of hospitalization included a WBC count of 4.9 cells/μL and CSF fluid that was colorless and showed 0 nucleated cells, an RBC count of 1 cell/μL, a protein level of 238 mg/dL, and a CSF glucose level of 45 mg/dL.

HIV testing performed at the time of hospitalization was positive, with a CD4 cell count of 5 cells/mm³ (CD4, 0.9%). Cranial MRI performed at admission showed extensive, bilaterally symmetric, deep edema in the gray matter and extending into the adjacent white matter, which we interpreted as most likely representing a toxic or metabolic encephalopathy or an aggressive viral encephalopathy, all of which are uncharacteristic of common opportunistic infections (figure 1).

CSF test results were negative for a cryptococcal antigen test, india ink staining, a Venereal Disease Research Laboratory test for syphilis, and PCR for cytomegalovirus, herpes simplex virus, and JC virus. In addition, test results were negative for a serum rapid plasma reagin test, a fluorescent treponemal antibody absorption test, and cytomegalovirus PCR. The serum toxoplasma IgG titer was <1:16, and the toxoplasma IgM titer was <1:8. A test for hepatitis C virus antibody yielded a negative result, with hepatitis C virus RNA level of <300 copies/mL. Results of serum staining for bacteria and fungi and bacterial and fungal cultures were also negative.

The patient was discharged from the hospital 1 month after admission with significant improvement in mental status and improvement in plasma HIV load (a decrease from 8946 copies/mL at the time of hospitalization to 711 copies/mL at the time of discharge). Six months after discharge from the hospital, his neurological functioning was almost at baseline levels, according to a report from his wife. The patient resumed all daily activities gradually and without difficulty. At a follow-up examination performed 6 months after hospital discharge, cranial MRI showed almost complete resolution of the edema in the gray matter, with only minimal residual periventricular hyper-intensities in the white matter along the anterior lateral ventricles bilaterally. At this follow-up examination, conducted 6 months after the CD4 count had increased to 46 cells/mm³, his plasma HIV load was undetectable. At a follow-up examination conducted 3 years after his hospital discharge, his CD4 cell count had risen to 209 cells/mm³, and his plasma HIV load had remained undetectable.

Direct infection of the brain with HIV ultimately results in HIV encephalopathy, which, prior to the advent of HAART, was diagnosed in 10%-30% or more of HIV-infected individuals [1]. The symptoms of HIV encephalopathy involve cognitive,
motor, and behavioral changes. The primary cognitive symptom is forgetfulness, which is often associated with slowed mental and motor abilities [2]. HIV encephalopathy generally presents with 2 radiological changes: cerebral atrophy and diffuse signal or density changes in the white matter [3].

The patient we describe presented with a severe HIV-infection–related mental status change that cannot be explained by opportunistic infection. Cranial MRI findings were not typical for common opportunistic infections, including toxoplasmosis, cytomegalovirus infection, progressive multifocal leukoencephalopathy, or herpes simplex virus infection. This patient’s clinical picture was consistent with HIV encephalopathy, and the diagnosis was based on exclusion of other possible causes of mental status change.

In this antiretroviral drug–naive patient, the effects of HAART were dramatic. By clinical standards, this patient had full recovery of all neurological functioning. More importantly, the radiographical evidence of this patient’s HIV encephalopathy almost completely regressed. This case demonstrates that HIV encephalopathy is a potentially reversible condition with effective antiretroviral therapy.

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References


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Fatal Septicemia and Pyomyositis Caused by Salmonella typhi

Sir—Pyomyositis in patients with diabetes mellitus is usually caused by Staphylococcus aureus [1]. Pyomyositis due to Salmonella species is rare and is usually caused by nontyphoid Salmonella [2–7]. We report a diabetic patient with fatal septicemia and pyomyositis caused by Salmonella serotype Typhi.

A 76-year-old man presented at the emergency department of our hospital with a history of pain in the left thigh for 7 days. He had a >30-year history of diabetes mellitus controlled with regular oral hypoglycemic agents. He denied having any recent diarrhea. Findings of physical examination included blood pressure of 99/51 mm Hg, body temperature of 36.4°C, heart rate of 114 beats/min, and respiratory rate of 18 breaths/min. The patient’s abdomen was soft and distended and bowel sound was hypoactive. Local swelling and tenderness of the left thigh were noted. Findings of other examinations were unremarkable. Laboratory data included a WBC count of 12.98×10^9/L with 16% bands and 73% neutrophils, a hemoglobin level of 8.3 g/dL, and a platelet count of 249×10^9/µL. Blood chemistry values were as follows: urea nitrogen, 59.4 mg/dL; creatinine, 2.35 mg/dL; blood glucose, 245 mg/dL; creatinine kinase, 149 mg/dL; and creatine kinase isoenzyme–MB, 9.2 mg/dL. MRI of the upper thigh revealed an abscess involving the pelvic cavity and adductor longus muscle (figure 1). Destruction of muscle architecture involving the left iliopsoas, the left rectus femoris, and the left adductus longus was also found. The patient was empirically treated with intravenous ceftriaxone (1g every 12 h) and clindamycin (600 mg every 6 h). Surgical drainage and debridement were not performed because the patient’s family refused treatment. In addition to the intravenous antibiotic therapy, the patient received aggressive fluid resuscitation and high-dose inotropic agents, but profound septic shock persisted and the patient died 1 day after hospitalization.

Two consecutive blood cultures both yielded Salmonella species belonging to serogroup D (O9). The isolates were initially identified as S. Typhi on the basis of serotyping of H-antigen. Serotyping was done according to the Kauffman and White scheme that uses somatic and flagellar antigens (Becton Dickinson) and conventional biochemical identification methods and the Phoenix System (Becton Dickinson, Cokkeysills, MD) [8]. The 16S rRNA partial sequencing analysis with a pair of universal primers (DG74 and RW01) further confirmed the identification of S. Typhi, with an identity of 99% (1355 of 1362 nucleotides) (GenBank accession number AL627280.1) [9]. Isolates were susceptible to ampicillin, ceftriaxone, ciprofloxacin, and piperacillin with standard disk diffusion susceptibility-testing method.

Clinically, pyomyositis often presents as localized muscle pain, swelling, stiffness, and tenderness with or without systemic signs of sepsis. Most cases of pyomyositis occur in the tropics, and the condition rarely occurs in nontropical areas [1]. Pyomyositis is caused by direct invasion or hematogenous spread. However, only 25%–50% of patients with pyomyositis report a history of trauma [1]. The iliopsoas and quadriceps femoris muscles were most commonly involved [4]. S. aureus is the most common pathogen, accounting for 66%–90% of all pathogens in several series, followed by streptococci (4%–16%) [1]. Infections caused by Salmonella species...