chronic liver disease, celiac disease, mastocytosis, and AIDS, and as an increased immune response in intravenous drug users [3]. Lymphadenopathy occurs as benign hyperplasia and proliferation in response to infection and/or antigenic stimulation. The features of lymph nodes on ultrasound or CT scan, such as size, number, or distribution, are important in the evaluation of abdominal lymphadenopathy. Larger, numerous, rounded nodes; presence of mass effect; and absence of echogenic hilum favor malignant diseases [4–6]. However, in contrast to these reports, certain studies show that lymph node characteristics are of no diagnostic value [2].

To our knowledge, herein we have described the first case of significant abdominal lymphadenopathy in association with malaria and its regression after treatment.

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References

Figure 1. CT scan of abdomen showing marked retroperitoneal lymphadenopathy in a 16-year-old boy with malaria.

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identify emerging infections that might be studied through this network, particularly those encompassed in acutely presenting syndromes that prompt ED presentation. On the basis of public health importance, a prioritized list is developed and reviewed with the emergency physician investigators to determine which studies are most feasible.

Current projects include investigation of the prevalence of and risk factors for infection with Shiga toxin–producing Escherichia coli and other enteropathogens among patients presenting with bloody diarrhea, the appropriateness of rabies prophylaxis practices following animal exposures, the prevalence of and risk factors for neurocysticercosis among seizure patients who have undergone neuroimaging, and nosocomial ED Mycobacterium tuberculosis transmission and hospital isolation bed use for adults admitted for pneumonia or suspected tuberculosis.

Future plans for EMERGE ID NET include studies of antimicrobial use, and meningitis and encephalitis, and consideration of other public health concerns such as injury and national and international network expansion.

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**References**

Paired CSF and plasma specimens were obtained 5 hours after patients received a dose of saquinavir, and were stored at $-70^\circ$C before batch processing to determine saquinavir concentrations (by use of high-performance liquid chromatography [HPLC]; detection limit, 2 ng/mL) and HIV viral loads (AmpliSeq PCR; Roche Diagnostic Systems, Branchburg, NJ; detection limit, 400 copies/mL). When a PCR inhibitor was present, a nucleic acid sequence–based amplification method (NASBA, Organon Teknika, Belgium; detection limit, 2,000 copies/mL) was used. In addition, in those with PCR inhibitors of sampling, the result of a PCR viral load on a sample obtained within 1 day or 35 days (patient 9) of the lumbar puncture is shown.

Saquinavir concentrations in CSF were very low for all patients, irrespective of plasma levels. The mean plasma saquinavir level was 167 ng/mL for patients receiving nucleosides and saquinavir, and 1,094 ng/mL (not significant) for those receiving dual protease inhibitor therapy, supporting earlier findings of elevated plasma saquinavir levels associated with ritonavir co-administration [1a]. The ratio of CSF to plasma level was 0.3% in the single sample (patient 7) for which a CSF saquinavir concentration of 6.5 ng/mL was detected. There are no comparable human data for ritonavir, although animal studies have shown total ritonavir levels in the CSF ranging from 0.03% to 0.08% of total serum levels (personal communication: R. W. McLean, Abbott Laboratories), i.e., 10-fold lower than the saquinavir ratio in patient 7.

Suboptimal concentrations of antivirals in the CNS or CSF may allow persistent viral replication and lead to the selection of drug-resistant HIV mutants, which may then move from the CNS into the plasma/lymphoid compartment. In addition, persistent viral replication in the CNS may lead to cognitive deterioration in individuals who have otherwise achieved clinical remission, or may up-regulate other viruses important to CNS pathology. No conclusions can be drawn concerning the efficacy of the drug combina-

### Table 1. Summary of test results for nine outpatients of a large HIV center (London) receiving antiretroviral therapy.

<table>
<thead>
<tr>
<th>Patient no.</th>
<th>Nadir CD4 count*</th>
<th>CD4 count* at lumbar puncture</th>
<th>Reason for lumbar puncture</th>
<th>Treatment</th>
<th>HIV RNA viral load (copies/mL)</th>
<th>Saquinavir concentration (ng/mL)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Plasma</td>
<td>CSF</td>
<td>Plasma</td>
</tr>
<tr>
<td>1</td>
<td>3</td>
<td>154</td>
<td>ADC</td>
<td>d4T/3TC/SQV</td>
<td>3,500 &lt;400</td>
<td>126    &lt;2.5</td>
</tr>
<tr>
<td>2</td>
<td>3</td>
<td>102</td>
<td>Meningitis ?</td>
<td>d4T/3TC/SQV</td>
<td>1,500 1,000</td>
<td>&lt;2.5   &lt;2.5</td>
</tr>
<tr>
<td>3</td>
<td>1</td>
<td>50</td>
<td>Encephalitis² ?</td>
<td>d4T/ddI/IND/SQV</td>
<td>13,000 &lt;400</td>
<td>102    &lt;2.5</td>
</tr>
<tr>
<td>4</td>
<td>3</td>
<td>36</td>
<td>ADC</td>
<td>AZT/ddC/SQV</td>
<td>&lt;2,000³ &lt;400</td>
<td>22.4   &lt;2.5</td>
</tr>
<tr>
<td>5</td>
<td>5</td>
<td>31</td>
<td>ADC</td>
<td>RIT/SQV</td>
<td>563,000 7,000</td>
<td>377    &lt;2.5</td>
</tr>
<tr>
<td>6</td>
<td>5</td>
<td>5</td>
<td>ADC</td>
<td>RIT/SQV</td>
<td>1,800 13,800</td>
<td>2,080  6.5</td>
</tr>
<tr>
<td>7</td>
<td>10</td>
<td>255</td>
<td>Tremor</td>
<td>RIT/SQV</td>
<td>&lt;2,000³ &lt;400</td>
<td>548    &lt;2.5</td>
</tr>
<tr>
<td>8</td>
<td>84</td>
<td>118</td>
<td>ADC</td>
<td>RIT/SQV</td>
<td>&lt;2,000³ &lt;400</td>
<td>1,370  &lt;2.5</td>
</tr>
<tr>
<td>9</td>
<td>4</td>
<td>183</td>
<td>ADC</td>
<td>RIT/SQV</td>
<td>&lt;2,000³ &lt;400</td>
<td>3,915  &lt;2.5</td>
</tr>
</tbody>
</table>

NOTE. ADC = AIDS dementia complex; AZT = zidovudine; CMV = cytomegalovirus; d4T = stavudine; ddC = zalcitabine; ddl = didanosine; IND = indinavir; NASBA = nucleic acid sequence–based amplification; RIT = ritonavir; SQV = saquinavir; 3TC = lamivudine; ? = symptoms suggestive of.

* cells/mm³.

² Stable CMV retinitis. CSF PCR positive for CMV.

³ NASBA assay.
Pacemaker Endocarditis Due to *Staphylococcus lugdunensis*: Report of Two Cases

*Staphylococcus lugdunensis* is a coagulase-negative staphylococcus responsible for skin and soft-tissue infections and for acute infective endocarditis [1]. The severity and rapid evolution of the symptoms of infective endocarditis due to *S. lugdunensis* closely resemble those of infective endocarditis due to *Staphylococcus aureus*, which are associated with a high mortality rate of 71%. Therefore, early diagnosis and prompt appropriate therapy are needed for a favorable outcome. To our knowledge, we describe the first two cases of acute pacemaker infective endocarditis due to *S. lugdunensis*, which were cured by the removal of the entire pacing system and by valvular surgery.

**Case 1.** In 1997, a 62-year-old man required placement of a transvenous pacemaker because of a symptomatic atrioventricular block. Fever and chills appeared 1 month later. Physical examination at admission revealed fever, a grade 2/6 holosystolic murmur, and a diminished right basilar pulmonary sound. Transesophageal echocardiography demonstrated a voluminous tricuspid vegetation (2 cm) and atrial lead vegetation, together with a small tricuspid regurgitation. *S. lugdunensis* was isolated from three blood cultures. The patient was treated with cloxacillin and gentamicin. The entire pacing system was removed, and a tricuspid bioprosthesis was placed. *S. lugdunensis* was isolated from the native tricuspid valve, the pacemaker leads, and the infective thrombi. A new transvenous pacemaker was implanted 3 weeks later. The patient was cured after 6 weeks of intravenous antibiotic therapy.

**Case 2.** In 1998, 3 days after cutaneous effraction of a toenail by a podiatrist, a 65-year-old diabetic man was admitted to the hospital because of an acute febrile syndrome with chills and dorsolumbar pain. His medical history included pacemaker implantation 1 year before because of an alternating bundle-branch block. Physical examination disclosed no other abnormality. Four cultures of blood and one culture of a toenail specimen yielded *S. lugdunensis*. Macrogenic restriction profiles of the different strains of *S. lugdunensis* by use of pulsed-field gel electrophoresis of blood and toenail isolates were identical. Bony scintigraphy was compatible with spondylitis at the T5 to L1 levels. Transesophageal echocardiography showed five voluminous vegetations on the aortic and ventricular leads and a small tricuspid regurgitation. The entire pacing system was removed and a tricuspid valvuloplasty was performed. Cultures of pacemaker material were negative, but gram-stained preparations of the vegetations revealed gram-positive cocci and numerous polymorphonuclear neutrophils. The patient was treated with cloxacillin, gentamicin, and ofloxacin, and he was cured after surgery and 8 weeks of antibiotic therapy.

We report herein two cases of definitive infective endocarditis on pacemaker leads, as defined by the Duke criteria [2], with two major clinical criteria (persistent positive blood cultures and positive echocardiogram) and, in addition, for the first case, one pathological criterion (isolation of *S. lugdunensis* from the blood, tures used in this study, as the numbers were small. Some patients had CNS infections in addition to HIV, but although they may have been expected to have had inflamed and “leaky” blood-brain barriers, there were no consequent increases in CSF saquinavir concentrations.

However, protease inhibitor concentrations in CSF may not be a reliable means of predicting efficacy of the agent in the CSF. Drug molecular size, protein binding, lipophilicity, and membrane transport may all determine the extent of CNS penetration. Amphotericin is not detected in CSF samples, although it achieves good brain penetration [2], and comparisons between CSF and brain penetration of zidovudine and stavudine in an animal model show that while both have similar CSF levels, brain-tissue uptake of stavudine is far less than that of zidovudine [3].

In addition, in a recent study by Farthing et al. of protease-inhibitor-naïve patients who were started on dual ritonavir/saquinavir therapy, 12 of a subgroup of 13 patients with undetectable plasma viral loads (<400 copies/mL) after treatment for a median of 60 weeks also had CSF HIV levels below detection [4].

Drug concentrations in CSF need to be interpreted with caution, and changes in HIV load may be the best predictor of drug efficacy in compartments such as the CNS where pharmacokinetic data may be misleading.

Reference


4. Farthing C, Japour A, Cohen C, et al. Cerebrospinal fluid (CSF) and plasma Drug concentrations in CSF need to be interpreted with caution, and changes in HIV load may be the best predictor of drug efficacy in compartments such as the CNS where pharmacokinetic data may be misleading.

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4. Farthing C, Japour A, Cohen C, et al. Cerebrospinal fluid (CSF) and plasma Drug concentrations in CSF need to be interpreted with caution, and changes in HIV load may be the best predictor of drug efficacy in compartments such as the CNS where pharmacokinetic data may be misleading.

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the vegetations, and the pacemaker leads). In both cases, the portal of entry was probably the skin. Indeed, *S. lugdunensis* is described as a commensal that colonizes the human skin [3]. For the two cases we described, the possible routes of infection are as follows: patient 1—the skin effraction during pacemaker implantation (interval between surgery and onset of infective endocarditis only 4 weeks), and patient 2—the toenail lesion after the podiatrist’s care (as suggested by the identity of the pulsotypes of the skin and the blood culture isolates).

The clinical courses of these two cases reflect again the severity of infective endocarditis due to *S. lugdunensis* characterized by a rapid evolution of the disease (the onset of fever 3 days after the podiatrist’s care for patient 2), the presence of voluminous vegetations for both patients, and a metastatic bone localization for patient 2. Moreover, staphylococcal pacemaker lead infections generally require the removal of the entire pacing system. For all these reasons, surgery was performed immediately for these two patients with pacemaker endocarditis due to *S. lugdunensis*. Both patients responded favorably to the combined surgical and anti-staphylococcal therapy.

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References

