sessed. Resource constraints and financial pressures have become common in health care; if Saleh et al. [1] practice in settings where such constraints are absent, they are fortunate indeed.

We conclude by agreeing with Hanssen et al. [2] that the use of modeling is not a substitute for well-designed, randomized, controlled trials. These have been rare in the field of orthopedic infectious diseases but are badly needed. Until data from such trials become more widely available, we hope that our model will serve as an aid to decision making for clinicians involved in the management of these challenging infections.

References


Urine Samples for Rabies RNA Detection in the Diagnosis of Rabies in Humans

Sir—Elsewhere, we reported the use of nucleic acid sequence–based amplification (NASBA) for the detection of rabies RNA in the diagnosis of human rabies before death [1]. We were able to detect rabies RNA in samples of saliva and/or CSF obtained from all 4 patients with rabies we studied. Although the NASBA method is relatively simple, it is often difficult to obtain saliva specimens by voluntary spitting or swabbing, because most of the patients are usually dehydrated and not cooperative. Lumbar puncture cannot always be performed safely in a severely agitated patient and may not always yield RNA [1]. An indwelling urinary catheter is inserted in all patients suspected of having rabies at our institution; therefore, urine samples can be obtained throughout the illness. We report the NASBA results with use of urine samples.

From November 2000 through September 2001, we obtained specimens from 4 patients with rabies. None of the patients received postexposure rabies prophylaxis. Three patients with furious rabies had been bitten by stray dogs 24–60 days before the onset of symptoms. Durations of survival ranged from 5 to 13 days. One male patient (patient 1) had an unusual presentation with bilateral arm paralysis and loss of pinprick sensation in both arms. Quadriplegia followed, and coma developed within the next 2 days. We could not obtain a good history of rabies exposure. This patient survived for 13 days. Postmortem examination of brain samples revealed that they were positive for rabies by use of direct fluorescent antibody testing, mouse inoculation testing, and NASBA.

All samples were kept at 4°C for 2 h (for 3 patients) and 24 h (for 1 patient), until studies for the presence of the rabies nucleocapsid gene were conducted. All patients except the patient with the unusual presentation (patient 1) had detectable rabies RNA in samples of saliva and/or urine. Patient 1 had no RNA detectable in 2 saliva samples (obtained on days 5 and 6 after the onset of symptoms) and in CSF and urine samples (1 sample each obtained on day 5 after onset). Saliva and urine samples obtained from patient 2 on day 2 after onset and from patient 3 on day 4 after onset were positive for rabies RNA. A CSF sample obtained at the same time from patient 3 was negative for rabies RNA. Of the 6 samples (3 saliva and 3 urine) obtained from patient 4 on days 4,
5, and 10 after onset of symptoms, only 1 saliva sample, which was obtained on day 4 after onset, was positive for rabies RNA. A CSF sample obtained from this patient on day 4 was also negative. These data confirmed our earlier findings that not every sample can be expected to test positive for rabies RNA. Therefore, serial tests should be performed on saliva, urine, and CSF samples whenever possible. Urine and saliva testing yielded the highest proportion of positive results in this small series (urine testing, 2 of 4 patients; saliva testing, 3 of 4 patients), as compared with testing of CSF samples (none of the samples obtained from 3 patients yielded positive results). Frozen sections of nuchal skin samples are not routinely obtained at our hospital for fluorescent testing techniques, which precluded us from using these samples for RNA detection [2]. Previous analyses using immunohistochemistry failed to demonstrate rabies virus antigen in kidney specimens [3]. Nevertheless, our study suggests that urine samples may be another practical source for rabies RNA detection.

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Kawasaki-like Illness in Human Immunodeficiency Virus–Infected Patients: Is the Etiologic Agent the Same as in Pediatric Kawasaki Disease?

Sir—I read with interest the article by Johnson et al. [1] about Kawasaki-like illness in adult HIV-infected patients. As pointed out by the authors, Kawasaki disease (KD) is an acute, febrile illness of unknown etiology that usually is seen in children of <5 years of age [2]. KD is rarely reported in adults. Until a diagnostic test is available, we will not know whether the etiologic agent of KD is responsible for a similar syndrome seen in some adults. Although the clinical findings noted in the 2 patients Johnson and colleagues [1] described could be compatible with KD, there are other diseases that may present with similar findings that should be part of the differential diagnosis. Specifically, patients with leptospirosis or toxin-mediated disease (staphylococcal or streptococcal disease) can have very similar presentations to that of patients with KD. Many patients with toxin-mediated disease may not have conditions that evolve into classic toxic shock syndrome. These patients sometimes present with trivial-looking sources of infection, such as a paronychia. We have seen several patients who were initially thought to have KD in whom opacification of the sinuses was revealed on CT of the sinus (1 of whom also had a blood culture positive for *Staphylococcus aureus*) [3]. These patients recovered after receiving antibiotics intravenously (1 patient also required sinus drainage) and had no subsequent changes visible on echocardiographs. Patients with toxin-mediated disease may present with fever, nonexudative conjunctivitis, red and/or cracked lips, swollen extremities, red palms and/or soles, and rash, and they often respond to intravenous immunoglobulin therapy. Such patients are sometimes very difficult to distinguish from those with KD. They may display elevated levels of acute-phase reactants, leukocytosis with left shift, low albumin levels, and mildly elevated transaminase levels (consistent with both KD and toxin-mediated disease). Desquamation of the skin of the fingers and toes typically occurs in the convalescent phase of both illnesses.

In the 2 patients described by the authors, many of the laboratory findings are not supportive of KD: neither patient had leukocytosis, left shift, or anemia. Only 1 of the 2 patients had elevations in the erythrocyte sedimentation rate and C-reactive protein level. It is unknown whether either patient had sterile pyuria. Both patients had thrombocytopenia, which is unusual in KD. It is unknown whether either of these patients had thrombocytosis later in the course of the illness, which is typically seen in KD. In terms of clinical findings, neither patient had desquamation in the convalescent phase of infection, which is also unusual. Neither patient had significant adenopathy (>1.5 cm in size); however, adenopathy is often absent in patients with KD. Taken together, the absence of all these findings make it questionable that the etiologic agent of KD caused the Kawasaki-like syndrome observed in these patients.

Johnson and colleagues suggest that immunodeficiency predisposes adults to KD. This suggests that either there is an acquired hypogammaglobulinemia or that the immune system is not functional enough to make protective antibody and/or cause T-cell–mediated responses. If this is true, it is puzzling why there is not an increased incidence of KD in HIV-infected children. Children are already at risk by virtue of age. If altered immune status also