

clinical strains and transformants had bands of β -lactamase activity with an alkaline pI of 7.6 and 5.4. Polymerase chain reaction (PCR) amplification of the 26 clinical isolates was positive for bla_{CTX-M} and bla_{TEM} (7). The 26 strains of *E. coli* had the same profile by repetitive-element PCR and pulsed-field gel electrophoresis, while unrelated control strains had very different profiles. Sequencing in strains isolated from four of the patients identified a CTX-M-15 β -lactamase and a TEM-1 β -lactamase. The four strains were related to the phylogenetic group B2 and produced the iutA (ferric aerobactin receptor), YuA (*Yersinia* siderophore receptor), and fimH (type I fimbriae) virulence factors (8). Incidence of colonization or infection by the culprit strain was 34.3% (12 of 35 patients) within the initial 4-month period and 55.3% (26 of 47 patients) over a 1-year period.

Intensified hygienic procedures implemented in January 2002 contributed to a decrease in the number of cases in February only; since then, a regular increase of new cases extended the outbreak and caused problems with controlling it. All urinary tract infections were successfully treated with a 15-day course of trimethoprim-sulfamethoxazole; however, reinfection occurred in some. Neither incontinence ($p = 0.35$), dementia ($p = 0.22$), nor previous antibiotic treatment (amoxicillin, amoxicillin-clavulanic acid, extended-spectrum cephalosporins, and fluoroquinolones [$p = 1.00, 0.30, 0.12, 0.52$, respectively]) appeared to be risk factors for infection or colonization in our study, but the number of patients is too small to reach a conclusion. However, patients that were infected or colonized had greater functional impairment, especially incontinence and dementia. Nonambulatory status, decubitus ulcers, and feeding tubes were not risk factors for acquiring ESBL-producing *E. coli* in our study.

The outbreak has not been controlled: 13 patients have persistent digestive-tract colonization. Difficulties encountered in controlling such outbreaks may be explained by several factors. Patients cannot be easily isolated in long-term care facilities. Strict isolation and limitation of activity and mobility cannot always be applied because of their impact on social activities.

Acknowledgments

We thank Abel Naas for helping to collect the clinical information and Claudia Ferreira for helping to write this letter.

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Do Antiborrelial Antibodies Suggest Lyme Disease in Cuba?

To the Editor: Lyme disease is the most common vector-borne disease in the United States and parts of Eurasia (1). It represents a considerable emerging infectious disease threat because of its consequences to human health and the difficulties in preventing and controlling it (2,3).

In Cuba, Lyme borreliosis has never been reported. However, in the last 20 years ixodid ticks, mainly *Amblyomma cajennense*, have been found in the human population in the Cuban village of Las Terrazas, Pinar del R o. These ixodid bites were frequent and widespread, especially in children, many of whom were hospitalized without a confirmatory laboratory diagnosis. Affected persons had symptoms associated with Lyme disease such as erythematous macules or papules, fever, fatigue, malaise, headache, arthralgias, myalgias, meningitis, peripheral radiculoneuropathies, and myocarditis (4).

A Cuban researcher, a specialist in

ixodid ticks, was bitten several times by the ticks; dermatologic and neurologic symptoms compatible with Lyme disease (skin lesions, hyperesthesia with loss of reflexes, loss of muscular coordination, and fecal incontinence) developed. Borreliosis was not diagnosed at this stage; the diagnosis was either myeloradiculitis or Guillain-Barré syndrome. Three years later, a serologic diagnosis of Lyme disease was made by indirect immunofluorescence in a laboratory in the Czech Republic (5).

During 1998, serum samples from 14 persons who lived in the village Las Terrazas and had epidemiologic and clinical evidence of Lyme disease, were studied in our laboratory. We used an immunoglobulin (Ig) G and IgM–enzyme-linked immunosorbent assay (ELISA) kit (Enzygnost Borreliosis, Behring, Marburg, Germany), in which each strip contained wells coated with inactivated borrelian antigen (detergent extract from strain isolate PKo [*Borrelia afzelii*]), to detect specific antibodies to *B. burgdorferi* complex. The assays were performed according to the manufacturer's instructions. In our study, five serum samples had positive IgM titers and one near the cutoff value by IgM and IgG.

ELISA has been widely used to detect antibodies to *B. burgdorferi*; however, this assay is not standardized, which results in different levels of sensitivity and specificity. False-positive results may occur, especially when serum samples are obtained from persons with other illnesses (6).

To study possible cross-reactions with other infectious illnesses, different serologic tests were applied to the positive serum samples by using ELISA. One sample was weakly reactive to human leptospirosis (indirect hemagglutination assay with erythrocyte-sensitive substance antigen [Labiofam, Havana, Cuba]), but no samples were reactive to syphilis (rapid plasma reagin [Imefa, Havana,

Cuba] and hemagglutination of *Treponema pallidum* [Oxoid, Diagnostic Reagents, Basingstoke, UK]). No indication of other infectious diseases was found.

All serum samples positive by ELISA were also analyzed by IgG and IgM Western blotting in the spirochete laboratory at the University of Trieste, Italy. The Western blotting was performed with a protein profile from whole-cell strain PKo and by applying the criteria of positivity described by Hauser et al. (7). Two serum samples showed clear IgM antibody bands to 41- and 23-kDa proteins. No IgG bands were observed. This test reportedly is more sensitive than ELISA for IgM detection (6).

We investigated the clinical manifestations of the patients with positive Western blotting. We found that one of the patients had been bitten several times by ticks and had an erythematous rash around the different bite sites; the rashes reddened and expanded over the course of a few days, with partial central clearing. The patient also had fever, hepatosplenomegaly, adenopathies, joint pain, and some nonspecific symptoms. He was given erythromycin before the laboratory results were confirmed and had a satisfactory recovery. In similar situations, repeat testing would be highly advisable. This was the same patient with low levels of antibodies to *Leptospira*. Investigating the symptoms of the other patient was not possible.

The presence of IgM antibodies is frequently confirmed in the early stage of Lyme disease (6). The patient's history of being bitten by an *A. cajennenses* tick, clinical manifestations of Lyme borreliosis, and specific antibodies to *B. burgdorferi* complex suggest the diagnosis of Lyme disease.

A. cajennenses has not been reported as a vector for Lyme disease. However, it is very abundant and aggressive in Cuba, and bites from

this species are common. The genus *Ixodes*, the main vector of *B. burgdorferi* sensu lato, has not been reported in the area of the study. Several articles describe a new species in the United States, *B. lonestari*. *B. lonestari* in *A. americanum* has been confirmed in humans with erythema migrans (8,9).

No serologic test is available for antibodies to *B. lonestari*. That we found antiborrelial-complex antibodies may suggest the presence of a new species in this antigenic complex containing cross-reactive antigens, but many other studies are necessary to confirm it. This study represents the first serologic report of antiborrelial antibodies in Cuba. It suggests that Lyme borreliosis is present and that new cases can be expected in our country. Further laboratory studies are necessary for a more accurate diagnosis of this emerging infectious disease in Cuba.

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Human Herpesvirus 6 Encephalomyelitis

To the Editor: Denes et al. (1) reports successful treatment of human herpesvirus 6 (HHV-6) encephalomyelitis. The patient was an immunocompetent young woman whose symptoms were fever, urinary retention, blurred vision, quadriparesis, bilateral papillitis, and optic neuritis. Magnetic resonance imaging (MRI) showed multiple lesions on the spinal cord white matter and the left thalamus, and the cerebral spinal fluid (CSF) showed inflammation. The patient was treated with acyclovir for 3 days, high-dose methylprednisolone

for 5 days, cidofovir for 1 day, and ganciclovir for 15 days, starting on day 23 of hospitalization. By establishing a relationship between antiviral drug doses, serial determinations of HHV-6 DNA by polymerase chain reaction (PCR) in CSF, and neurologic improvement, Denes et al. concluded that antiherpesvirus drugs led to her recovery.

This case fits well in the spectrum of acute disseminated encephalomyelitis (ADEM), an inflammatory demyelinating disease of the central nervous systems of children and young adults, which occur in close temporal relationship with several infectious illnesses and immunizations (2–6). The disease has particular predilection to the optic nerves, spinal cord, brainstem, basal ganglia, and cerebral and cerebellar hemispheres. Maximal neurologic deficits are reached within several days, and resolution takes weeks or months. The condition is typically monophasic, but relapses have been reported (7). Histologic multifocal areas of inflammation and demyelination are found. In the pathogenesis of ADEM, an initial injury caused by an infectious agent, followed by a secondary autoimmune response, has been postulated, and animal models have provided experimental support; both CD4 and CD8 T cells have been implicated in a secondary autoimmune response (6). Despite the lack of controlled studies, corticosteroids are widely used to treat ADEM and high-dose methylprednisolone is the drug of choice (3,4). The largest series of ADEM in adults included 40 patients with a mean follow-up period of 38 months. The patients were given a standardized treatment regimen of methylprednisolone, 500 mg daily intravenously for 5 days, with no additional therapy if they recovered completely. In patients with persistent neurologic deficits, the initial intravenous therapy was followed by a regimen of oral methylprednisolone, which was

slowly tapered over 4 to 6 weeks. In patients with no response to this therapy, or whose condition had deteriorated during therapy, cyclophosphamide was given to seven patients, and immunoglobulin was given to one patient. Thirty-eight of 40 patients improved during the acute phase of the illness; in 7, symptoms completely resolved. One patient's condition remained unchanged and one patient died; no antiviral drugs were given (5).

The neurotropism of HHV-6 and that the CNS may be a site of viral persistence or latency are well recognized (8,9). On autopsy, evidence of fulminant encephalitis with HHV-6 DNA demonstrated by PCR, immunohistochemical staining, or nucleic acid hybridization, confirms that HHV-6 causes acute CNS disease (8). Nevertheless, whether evidence of HHV-6 DNA in CSF demonstrated by PCR can be solely relied on is debatable. HHV-6 DNA was detected in the CSF of 41 (28.9%) of 142 children with a history of HHV-6 infection (9). HHV-6-DNA was detected in the CSF of 47 (61%) of 77 children examined after primary HHV-6 infection. In the remaining 30 children (39%), HHV-6 DNA was detected in both peripheral blood mononuclear cells and CSF samples. HHV-6 variant A was detected more frequently in CSF than in specimens of other sites, which suggests that HHV-6A has greater neurotropism (10).

The role of HHV-6 in acute multifocal neurologic disease in immunocompetent adults requires additional observation, and its role in multiple sclerosis is in question. Much can be learned from careful study of patients (1).

I caution the casual reader who may conclude that using antiviral drugs against herpes viruses is recommended when acute multifocal neurologic disease clinically compatible with ADEM is indicated. High-dose IV methylprednisolone is the most utilized treatment, and the patient in