Case Reports

Is *Ixodes (Ixodiopsis) angustus* a Vector of Lyme Disease in Washington State?

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LYME DISEASE is a tick-borne spirochetosis that, since its initial recognition in the mid-1970s, has become a public health problem of increasing concern in the United States.[1-3] It is presently the most commonly reported arthropod-transmitted disease in this country. In the West, human cases have been recorded along the Pacific slope of the Sierra Nevada mountains (California) and in Nevada, Oregon, and Utah.[2] In California alone, 125 indigenously acquired cases were reported by state health authorities in 1986, and the official total for 1987 will be higher (R.A. Murray, PhD, oral communication, April 1988).

Two species of hard-bodied (ixodid) ticks serve as primary vectors of the etiologic agent, *Borrelia burgdorferi,* to humans—that is, *Ixodes (Ixodes) dammini* and *Ixodes (Ixodes) pacificus* in eastern and western United States, respectively.[4,5] A third species of ixodid tick, *Amblyomma americanum,* has been implicated as a secondary vector to humans in New Jersey.[6,7] Although the western black-legged tick, *I pacificus,* is assumed to be the usual source of human infection in the Far West,[5,8-11] other ixodid ticks that bite people less frequently also may transmit the infection to humans in this region. With this communication, we report a case of Lyme disease acquired in Washington State that was associated with the bite of an *Ixodes* tick in the subgenus *Ixodiopsis.* This tick, *Ixodes angustus,* has not previously been incriminated as a vector of *B burgdorferi.*

Report of a Case

The patient, a 3-year-old girl from Redmond, Washington, was referred to one of us (H.F.) in May 1987 for the removal of a tick that was embedded in the lash line of her right upper eyelid (Figure 1). Her parents noted the foreign body two days before the initial appointment and had tried unsuccessfully to remove it. They reported that their daughter had encountered the tick near their home in suburban Seattle. Examination showed an engorged, live tick embedded in the nasal aspect of the right superior eyelid, with bloody excrements on the skin immediately below the eye (Figure 1). A slight erythema had developed on the eyelid around the site of tick attachment. The tick was removed with forceps after the child had been sedated with chloral hydrate. A culture of the wound was taken and topical erythromycin ointment prescribed, to be applied three times a day for two weeks. During a follow-up appointment one week after removal of the tick, the wound was healing uneventfully, though a slight erythema remained (Figure 2). Treatment with topical erythromycin was continued even though the culture of the wound was negative for pathogens.

Three weeks after the tick removal, the patient presented with a notably erythematous skin lesion (Figure 3). Her parents reported that the lesion had been present for six days (beginning 23 days after the initial contact with the tick), with no associated signs or symptoms except that the area was tender to the touch. On biomicroscopic examination, there was a cherry red, swollen area in the upper lid measuring about 5 by 7 mm, extending in a semicircular area around the tick attachment site. The skin was intact and no residual foreign bodies were observed in the eyelid. A second culture of the area showed no growth within 48 hours. The topical antibiotic was switched to a combination ophthalmic ointment containing polymyxin B sulfate, bacitracin zinc, and neomycin sulfate (Neosporin), to be applied three times a day, and cephalexin oral suspension (Keflex), 125 mg three times a day, was prescribed for two weeks. Additionally, the parents were instructed to apply warm soaks four times a day to the affected area. Within the next three weeks, the lesion resolved slowly, and five months later only a slight erythema remained.

The initial clinical impression was that the delayed skin reaction was due either to a granulomatous response to retained mouthparts or to a secondary bacterial infection. (A subsequent examination of the tick by one of us [R.S.L.] showed that the distal portion of the hypostome had broken off; the hypostome is the central-most portion of the mouthparts that serves to anchor the tick to its host while feeding.) On the other hand, the time course and appearance of the skin lesion were also characteristic of erythema migrans (formerly erythema chronicum migrans), the hallmark of Lyme disease.[2,12] Serologic tests for Lyme disease six months after the tick bite showed that the patient had an indirect fluorescent antibody (IFA) titer of 1:256 and an optical density ratio by enzyme-linked immunosorbent assay of 0.27. An IFA titer of 1:256 or greater and an optical density ratio of greater than 0.20 are considered diagnostic of Lyme disease in a patient having compatible clinical features, such as erythema migrans. Serologic tests for syphilis proved to be negative. On receipt of the serologic findings, the patient was treated with a two-week course of oral penicillin V potassium, 125 mg four times a day. To date, she has shown no evidence of arthritis, carditis, neuritis, or other sequelae that may occur during late Lyme disease.[13]

Discussion

Lyme disease was not recognized as a distinct clinical disorder in Washington before 1986. Since then, 11 cases

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have been confirmed among residents of this state. In the West generally, and in California and Oregon specifically, the western black-legged tick, *Ixodes (Ixodes) pacificus,* is thought to be the primary vector of *B burgdorferi.*[5,8-11] This tick has been recorded from 14 of Washington's 39 counties so far by a passive surveillance system. The relation of *I pacificus,* however, and other ticks to the Lyme disease spirochete and its associated vertebrate hosts has not been studied in the Pacific Northwest.

In the northeastern United States, the primary vector of *B burgdorferi* to humans is *I (Ixodes) dammini,*[4] which has a broad host range similar to that of *I pacificus.* Two other tick species in the subgenus *Ixodes, Ixodes neotomae* and *Ixodes scapularis,* also have been found infected naturally with *B burgdorferi* in this country.[14,15] *I neotomae* feeds mainly on lagomorphs and wood rats,[16,17] whereas *I scapularis* attaches to various species of lizards, birds, and mammals (including humans).

The tick implicated in the case described here was determined to be an adult female *Ixodes (Ixodiopsis) angustus.* J. E. Keirans, PhD, of the Smithsonian Institution, United States National Museum, confirmed the specific identity of the tick. This tick has been recorded from at least 24 states, including all of those in the Far West.[16] Its preferred hosts are various species of small rodents, but it occurs sometimes on shrews, cats, dogs, and humans.[16] In Washington State, *I angustus* has been recorded from ten counties, all of which are located west of the Cascade Mountains.

Ixodes angustus is the first member of the subgenus *Ixodiopsis* to be incriminated as a vector of *B burgdorferi* in this country or elsewhere; the principal vector in Europe is *Ixodes (Ixodes) ricinus,*[18] and *Ixodes (Ixodes) persulcatus* has been implicated recently as a vector in Asia.[19] It is not surprising that a tick such as *I angustus* that bites rodents principally and humans accidentally may sometimes transmit the Lyme disease spirochete to people. The white-footed mouse *(Peromyscus leucopus)* has been shown to be the primary reservoir of *B burgdorferi* in the northeastern US,[20,21] and the deer mouse *(Peromyscus maniculatus)* likewise is susceptible to infection with this spirochete.[22,23] Moreover, *P maniculatus* is distributed throughout most of the continental US including all of the West,[24] and this rodent and several other *Peromyscus* species serve as hosts of *I angustus.*[17,25]

The association of the bite of an *I angustus* tick with the development of an erythema migrans-like lesion 23 days later and a significant serum antibody titer six months afterward strongly suggests that this particular tick had transmitted *B burgdorferi* infection to our patient. Further, the patient had not traveled to another Lyme disease-endemic area, nor was she known to have been bitten by another tick or other potential arthropod vectors during this period. Definitive proof, of course, that *I angustus* is a vector of *B burgdorferi* must await the recovery of this spirochete from a tick that had been removed from a person who subsequently became ill with Lyme disease.

Lyme disease vector surveillance has not existed to a great extent in Washington State. Field and laboratory studies are necessary to identify tick species in the area and to determine the proportions harboring Lyme disease spirochetes. Tick and spirochete surveys conducted in northern California and southwestern Oregon from 1982 to 1984 have shown that the prevalence of spirochetal infections in adult *I pacificus* has ranged from 0.9% to 4.3%.[5,9] The infection rates for *I dammini,* the main vector for *B burgdorferi* in the northeastern United States, are reported as high as 100% in certain localities.[26] Investigations are underway to determine the prevalence of *B burgdorferi* in *I angustus* and to define the role of this tick in the ecology of Lyme disease.

The association of a Lyme disease case with a species of tick not previously known to be a vector of the disease portends expanding public health problems. *Ixodes angustus* is

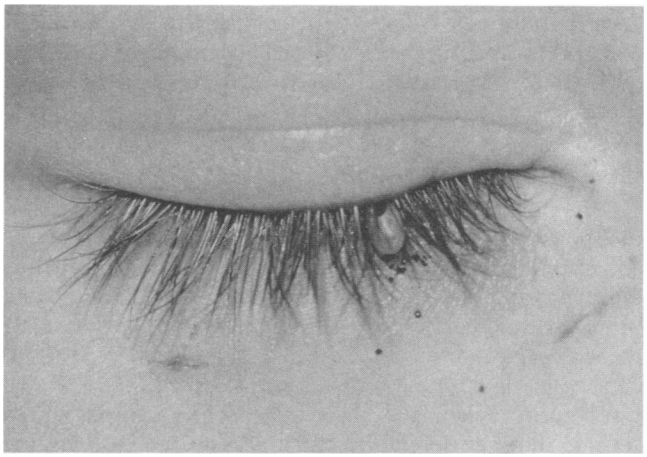

Figure 1.—A female *Ixodes angustus* is attached to the lash line of the right upper eyelid of the patient.

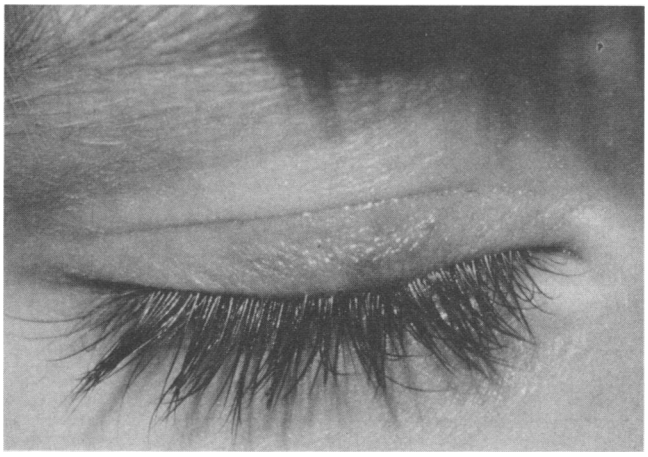

Figure 2.—The eyelid of the patient one week after removal of the tick shows slight erythema.

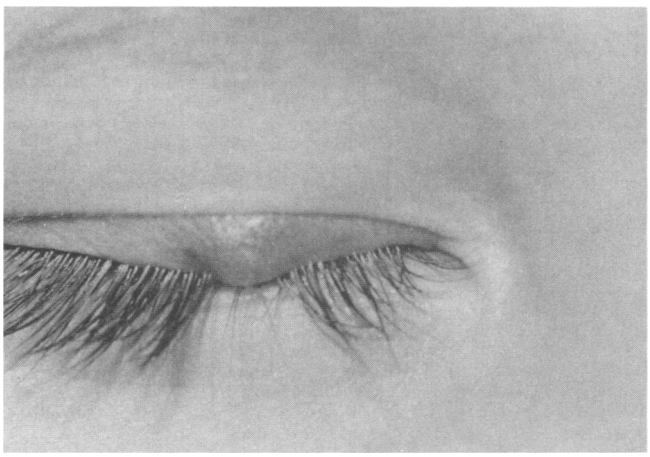

Figure 3.—An erythematous lesion is seen on the right upper eyelid three weeks after the tick was removed.

widely distributed in North America, occurring from California to Alaska on the Pacific Coast. Studies on the distribution of ticks in western Oregon have shown that *I angustus* is the most common tick found in certain coastal regions of this area.[27] Although generally considered to be a nest-specific parasite of rodents and insectivores, *I angustus* is known to bite humans.[17] The wide distribution and common occurrence of this tick species may have important implications for potential Lyme disease transmission outside known endemic areas and an increased incidence within established areas. Furthermore, as our rural population expands and participation in outdoor recreational activities increases, placing humans into areas formerly the domain of wild animals and their parasites, exposure to ticks and tick-borne diseases will increase.

The transmission of Lyme disease spirochetes to humans may be minimized by the prompt and proper removal of a tick vector. The lengthy feeding period (several days) is an important factor contributing to the risk of infection.[28] In addition to timely removal, it is important that the mouthparts of an embedded tick be removed completely to prevent the occurrence of secondary bacterial infections. The procedure recommended for removing an attached tick is to grasp it as close to the skin surface as possible with forceps or protected fingers and gently pull backward with steady, even pressure.[29]

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Adrenal Apoplexy Revisited

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DESPITE ITS INFREQUENCY, acute adrenocortical insufficiency is an important disorder to recognize because it is uniformly fatal unless promptly treated. Acute adrenocortical insufficiency is brought about by an intense stress occurring either in a patient with preexisting chronic adrenocortical insufficiency or in a patient with presumably normal adrenal glands that undergo massive hemorrhagic necrosis. This latter condition has been termed adrenal apoplexy. I describe the cases of two patients with adrenal apoplexy and discuss the cardinal aspects of its pathogenesis, clinical diagnosis, and management.

Report of Cases

Case 1

The patient, a 74-year-old woman, was admitted to the medical service because of fever, cough, shortness of breath, and chest pain for a week that had not responded to erythromycin therapy. She had had a resection of an adenocarcinoma of the sigmoid colon four years earlier without evidence of recurrence, a cerebrovascular accident six years earlier, and cutaneous lupus in the remote past at which time she was given glucocorticoids for three months. She was taking isosorbide dinitrate for mild angina and hydrochlorothiazide and triamterene for hypertension. On physical examination she was in moderate respiratory distress with a heart rate of 100 beats per minute, a blood pressure of 145/80 mm of mercury, a temperature of 38.3°C (101°F), a respiratory rate of 26 per minute, and signs of left lower lobe consolidation; the rest of the examination was unremarkable. A chest roentgenogram showed bilateral patchy infiltrates. Examination of a sputum specimen showed leukocytes and a mixture of bacteria that on culture yielded *Hemophilus influenzae* and oral flora. The blood leukocyte count was 8.1×10^9 per liter with 0.66 polymorphonuclear leukocytes and 0.17 band forms.

Treatment with intravenous fluids and cefamandole nafate was begun. On the fourth hospital day, the patient began

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