Lyme disease takes its name from a town in New England where an outbreak of chronic arthritis in children was studied and reported in 1977. Epidemiology suggested a tick borne infection, and in 1982 the causative organism, a previously unrecognised spirochaete, was found and named *Borrelia burgdorferi*. Since then the disease has become more widely recognised as a result of clinical awareness, better diagnostic tests, and an increase in deer stocking. It is now the commonest tick borne infection in the USA, with over 6000 reported cases in 1987. The tick is widely distributed, and variants of the disease have been described in Europe since early this century. A rise in cases is now being reported in Britain for similar reasons as in the USA.

*Borrelia burgdorferi* is transmitted by ixodes ticks. The tick's life cycle is not fully documented, but in New England it depends on the white footed mouse for its larval and nymphal stages and the white tailed deer as the preferred adult host, though West Coast ticks are apparently happy to substitute lizards for mice. The spirochaete lives in the gut of the tick and is injected into the unfortunate host through its saliva. Ixodes ticks are widely distributed throughout America, Europe, and Asia, and the spirochaete can be found in mosquitoes and deer flies as well, though the importance of these to human disease is unknown.

Lyme disease is a multisystem disease with major cutaneous, neurological, cardiac, and rheumatological manifestations. For a complete and authoritative review of the disease and all its ramifications readers should consult the review by Steere,' who described the original cases in 1977. The disease has similarities to syphilis, with stages of localised infection, dissemination, and late persistent infection. Ocular involvement is uncommon and occurs mainly in the second stage. Following inoculation of the spirochaete by a tick bite patients develop a characteristic skin lesion, erythema migrans (stage I), usually centred on the tick bite, and they may develop fever, lymphadenopathy, and constitutional symptoms. Conjunctivitis has been reported in 11% of patients. Dissemination (stage II) is by blood or lymphatic spread. Secondary annular skin lesions appear, and the patient may be quite ill with fever, malaise, and fleeting musculoskeletal pains. After several weeks or months 15–20% of patients in the USA have developed neurological symptoms, of which chronic lymphocytic meningitis, cranial polyneuropathy, or radiculitis are the commonest; 4–8% develop cardiac symptoms, with arrhythmias or myopericarditis. After six months about 60% of patients develop arthritis of major joints (stage III), particularly the knees. Apart from chronic arthritis a progressive encephalomyelitis and a skin disorder known as acrodermatitis chronica atrophicans can occur at this time. Congenital infection has been reported, usually with a fatal outcome for the fetus.

Ocular involvement can be seen as conjunctivitis in stage I but occurs more commonly in stage II; a keratitis may be seen in stage III. Ocular involvement appears to be relatively infrequent. The commonest findings are a uveitis or endophthalmitis, oculomotor palsy, or papilloedema from meningeal involvement.

Most descriptions of ocular disease concern sporadic case reports, but Kinward et al have recently reported a series of six cases with ocular involvement. The patients' ages ranged from 11 to 71 years; five of the six had visited endemic areas for Lyme disease but only two had a positive history of a tick bite. Five of the six patients had a bilateral vitritis, with similarities in one to pars planitis; the other patient had a fourth nerve palsy. Three of the six patients had a facial nerve palsy, the commonest cranial neuropathy found with Lyme disease. The most important lesson from these cases is that recognition of Lyme disease led to appropriate antibiotic treatment and a good visual outcome. Other ocular manifestations include ischaemic optic neuropathy, optic neuritis, and optic disc swelling with or without raised intracranial pressure, as well as oculomotor palsies. These are reviewed in the discussion of a patient with unilateral endophthalmitis from Lyme disease in this issue of the *BJO*.

The various manifestations appear to be related to direct infection with the spirochaete, which can be recovered histologically or cultured with varying degrees of success from involved organs. Most cases are, however, diagnosed serologically using an ELISA test. This becomes positive for IgM during the first few weeks of infection, during this time false negative results can occur. An IgG response occurs later. Seronegative cases can occur; in these the diagnosis was made by finding a specific T cell blastogenic response, and it has recently been shown that in these cases the antigen is sequestered in immune complexes.

In Britain Lyme disease usually occurs in people from a rural or agricultural background. In a study of serological titres in 101 patients from Dumfries chosen because of potential exposure 12 were found to give positive results from serological tests; all were farmers, six exclusively working with dairy cattle, the others working with cattle and sheep. No patient had typical features of Lyme disease, but 11 of the 12 had symptoms such as arthritis, which might have been relevant. No ocular Lyme disease has yet been reported in
Britain, though the gardener of a London ophthalmologist has apparently suffered cardiac manifestations. *Borrelia burgdorferi* is sensitive to tetracyclines and erythromycin. These appear to be the best antibiotics for the treatment of the systemic infection, as the spirochaete is apparently less sensitive than *Treponema pallidum* to penicillin, but the best treatment for intraocular disease is not yet known. The Bascom Palmer patients with early disease did well with doxycycline, and good results in the others were obtained with intravenous ceftriaxone. The disease may relapse after treatment, and in this series all three patients who did relapse had had prior treatment with oral steroids. The role of steroids in systemic infection is controversial, in one series predisposing patients to antibiotic failure. Systemic steroids are therefore probably at present best avoided in the management of patients with intraocular Lyme disease.

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