

## Lyme disease-ocular/oral scenario.

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### Editorial

Lyme disease is caused by the strains of spirochete "*Borrelia burgdorferi*" and commonly involves the skin, nervous system, heart, and joints. Though this disease is commonly found in the temperate regions, Ixodes(I.) ticks which transmit this disease are said to be present in the Himalayan region as well. Various species of the genus *Ixodes* reported from this region are *I. acutitarsus*, *I. granulatus*, *I. himalayensis*, *I. kashmericus*, and *I. ovatus* [1]. It is a multisystem disorder but the ocular/oral manifestations are the lesser known aspects and adequate knowledge is not shared on the subject. Hence, the examination of these two organ systems are often overlooked.

To observe ocular manifestations in Lyme disease, it is not necessary that the site of inoculation of the infection is close to the eyes. Ocular manifestations are in the form of conjunctivitis, episcleritis, scleritis, uveitis, macular oedema, vasculitis, venular occlusions, optic neuritis, cranial nerve palsies, diplopia, lack of accommodation, [2] periorbital edema, choroiditis, panophthalmitis, exudative retinal detachment, papilledema, motility abnormalities, and symblepharon. The infectious basis for Lyme disease was identified in 1982, which along with its various presenting features, requires a high index of suspicion [3]. Paralytic strabismus especially 6th cranial nerve palsy is among the most frequent manifestations of ocular Lyme disease. Most described cases of diplopia associated with Lyme borreliosis are secondary to abducens palsy complicating an intracranial hypertension [4].

The three most common early neurologic presentations of Lyme disease are cranial neuritis with 7th nerve palsy, painful radiculitis, and lymphocytic meningitis. Other cranial nerves may be involved leading to trigeminal neuralgia and Horner syndrome. Doxycycline (100 mg twice daily for 10 to 21 days) is the first choice for therapy for non-neurologic Lyme disease (relatively contraindicated in children younger than 8 and pregnant women). Neurologic disease is treated with intravenous ceftriaxone, 2 g daily for two to four weeks [5].

Although several types of keratitis have been associated with Lyme borreliosis, peripheral ulcerative keratitis with neovascularisation, seems characteristic [6]. Systemic disease, such as Grave's disease and blood disorders like lymphomas are the principle cause for orbital myositis. In these aetiologies, myositis is often bilateral. Infectious diseases, such as Staphylococcal septicaemia, may be another cause for unilateral myositis. Orbital myositis is an unusual manifestation of Lyme disease, although it is likely that the condition is underdiagnosed. Unexplained muscle swelling occurring in a patient who has had a rash or a recent history of a tick bite in an endemic area for Lyme disease should prompt consideration of this diagnosis [7]. Choroidal neovascular membrane (CNVM)

in Lyme disease has also been reported. Haematogenous spirochetal dissemination and the accompanying inflammatory process could lead to breaks in Bruch's membrane and CNVM development. Many of these clinical features of Lyme disease are also characteristic of syphilitic eye involvement [8] and hence a high degree of suspicion should be there when any patient presents with the signs and symptoms discussed above. Oral cavity involvement in the form of facial and dental pain, facial nerve palsy, headache, temporomandibular joint pain, and masticatory muscle pain has also been reported [9].

### References

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