Commentary

Leptospirosis is a zoonotic infection that is caused by *Leptospira interrogans*, a spirochete. The organism is motile, gram-negative aerobic and slow growing. Numerous animal populations can serve as carriers, but rodents, cattle, dogs and pigs appear to be the predominant animal hosts in many countries.[1,2] Humans are infected through direct contact with infected animals or an exposure to fresh water or soil, contaminated by urine of the carrier.[1,3]

The occurrence of leptospirosis is associated with socio-economic status, occupation, association with animals, recreational activity, climate and rainfall. Heavy rain and flooding will cause leaching of leptospira from soil into the water. Because of the occurrence of recent large outbreaks following the occurrence of severe floods, leptospirosis is a re-emerging infection.[1]

The pathogenesis of leptospirosis is not well-understood. The pathogenic mechanism can be classified into direct effect of the organism during bacteremic phase, and the host's response to an infection in the immunological phase. After entering the body, the organism invade the blood stream resulting in a bacteremia, disseminating into various organs such as the kidney, liver, lungs, heart and central nervous system. The organism disrupts the endothelial cell membranes of small vessels, leading to organ hemorrhage and ischemia.[2]

Fever, chills and rigor, myalgia, headache and proteinuria characterize an acute bacteremic phase of leptospirosis.[4] Hepatic involvement results in jaundice[2], and other systemic manifestations include pulmonary hemorrhage, acalculous cholecystitis, myocarditis and pancreatitis.[1] Symptom resolution may coincide with an immune phase and antibody production. Severe headache, meningism and lymphocytic meningitis may occur during this immune phase.[2]

Ocular involvement in leptospirosis may occur during both the systemic bacteremic and immunological phases. Ocular manifestations in an acute phase include conjunctival congestion without discharge, chemosis or subconjunctival hemorrhage.[5] Jaundice and limbal congestion is a pathognomonic of severe systemic leptospirosis.[2] Uveitis is an important complication of the late immunological phase, and hypopyon may occur when an inflammation is severe. Other ocular immunological manifestations include interstitial keratitis, hyperemic disc, membranous vitreous opacities, perivasculitis without vascular occlusion, retinal hemorrhage and neuroretinitis.[5]

Neuroretinitis is a type of optic neuropathy with which there is an inflammation of the retina and optic nerve, and classically characterized by the presence of optic disc edema accompanied by serous retinal detachment, extending to the macular area with formation of a partial or complete macular star. Neuroretinitis is a rare ocular manifestation of leptospirosis.[6] Infection like syphilis, tuberculosis, cat-scratch disease, lyme disease, toxoplasmosis, hepatitis B, mumps, measles and cycticercosis can be presented with neuroretinitis.[6]

Leptospirosis remains a diagnostic challenge since it often presents as non-specific febrile event. Ocular manifestation with neuroretinitis alone without systemic manifestation makes the diagnosis of leptospirosis more challenging.

The authors report a case of leptospirosis presenting as neuroretinitis alone without systemic manifestation of leptospirosis.[7] However, with the serological test, they were able to confirm the diagnosis and managed successfully.

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