Dengue-Associated Neuroretinitis: A Case Report

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ABSTRACT

Objective: To describe a case of dengue-associated bilateral neuroretinitis in a young female adult.

Methods: This is a case report.

Results: A 25-year-old female was referred for evaluation of bilateral blurring of vision during the convalescent stage of dengue fever. Visual acuity was 20/80 in each eye. Fundoscopy showed mild optic disc swelling, macular thickening, and hard exudates bilaterally. Dengue-associated neuroretinitis was considered. Intravenous methylprednisolone treatment for three days resulted in significant improvements in visual function and resolution of fundus abnormalities.

Conclusion: Dengue is a potential etiology of neuroretinitis in endemic areas, especially in those who develop visual symptoms during the convalescent phase. Prompt recognition and treatment may prevent long-term visual impairment.

Keywords: Dengue fever, neuroretinitis, methylprednisolone

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Dengue, an arboviral infection caused by one of four dengue virus serotypes (DENV-1 to DENV-4), is transmitted primarily by the *Aedes aegypti* mosquito and is endemic in many tropical and subtropical regions, including the Philippines.[1-3] The clinical spectrum of dengue ranges from an acute febrile illness to more severe forms such as dengue hemorrhagic fever and dengue shock syndrome. While systemic manifestations are well-documented, neurological complications are less common and include encephalitis, myelitis, and Guillain-Barré syndrome.[4]

Ocular complications of dengue are also rare but increasingly recognized, and may include retinal hemorrhages, maculopathy, and optic neuropathy. Optic neuritis, in particular, is an uncommon manifestation of dengue fever. It may present bilaterally and without the classical features associated with demyelinating optic neuritis, such as pain on eye movement. [5] Most reported cases occur during the convalescent phase of the illness, suggesting an immune-mediated pathogenesis rather than direct viral invasion.[5-6] Neuroretinitis is another rare ocular sequelae of dengue infection and visual recovery can be variable in affected individuals.[7-8] In this report, we describe a case of dengue-associated neuroretinitis in a young adult female, emphasizing the clinical findings, diagnostic workup, treatment response, and implications for early recognition and management.

CASE PRESENTATION

A previously healthy 25-year-old female was admitted to the Internal Medicine Service with complaints of fever, generalized weakness, and retro-orbital headache. Initial work-up revealed thrombocytopenia and a positive dengue NS1 antigen test, confirming dengue infection. On the eighth day of illness, coinciding with her first afebrile day, the patient developed painless blurring of vision in both eyes. At this time, platelet count was 66 x 10^9/L. The patient had no history of smoking, alcohol consumption, or sexual activity. Notably, she had a history of exposure to a domestic cat two to three weeks prior to her illness. She was subsequenlty referred to the Ophthalmology Department for further evaluation. On examination, uncorrected visual acuity was 20/150, which improved to 20/80 with pinhole, in each eye. Color vision was intact, with the patient identifying 16 out of 16 Ishihara plates in each eye. Pupils were equal and briskly reactive to light, with no evidence of a relative afferent pupillary defect. Intraocular pressures were 14 and 15 mmHg in the right and left, respectively. Anterior segment examination was unremarkable.

Fundoscopic examination revealed mild optic disc swelling, macular thickening, and hard exudates in both eyes (**Figure 1**). Optical coherence tomography (OCT) scan of the macula showed cystoid macular edema in the right eye and neurosensory macular detachment in the left eye with increased central subfield thickness in both eyes. OCT of the optic discs confirmed disc edema, showing thickened average retinal nerve fiber layer in both eyes (**Figure 2**). The Uveitis Service was consulted and systemic work-up, including erythrocyte sedimentation rate, C-reactive protein, chest radiography, and a tuberculin skin test, was performed, all of which were unremarkable. Dengue maculopathy was considered and oral prednisone was prescribed. However, initiation of the medication was delayed due to unavailability of the medication.

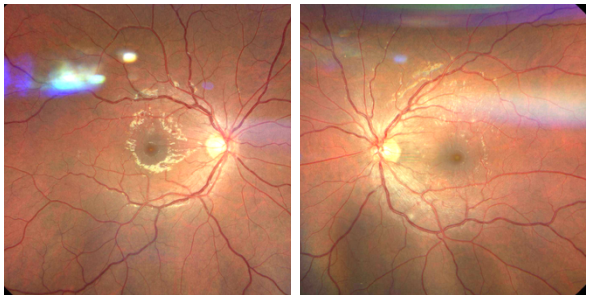

Figure 1. Baseline colored fundus photos showing bilateral mild optic disc swelling, thickened macula. and few scattered hard exudates that were more prominent in the left macula.

The patient's visual function continued to worsen over the next 3 days, with impairment of color vision. At this point, she was referred to the neuro-ophthalmology service. Contrast computed tomography (CT) of the brain was unremarkable. Bilateral neuroretinitis was considered and the patient was started with nepafenac eyedrops and intravenous methylprednisolone 1 gram per day was initiated for three days. On the third day of treatment, visual acuity had improved to 20/30 in both eyes, with normalization of color vision. Repeat OCT showed resolution of macular edema and optic disc edema (**Figure 3**). The patient was

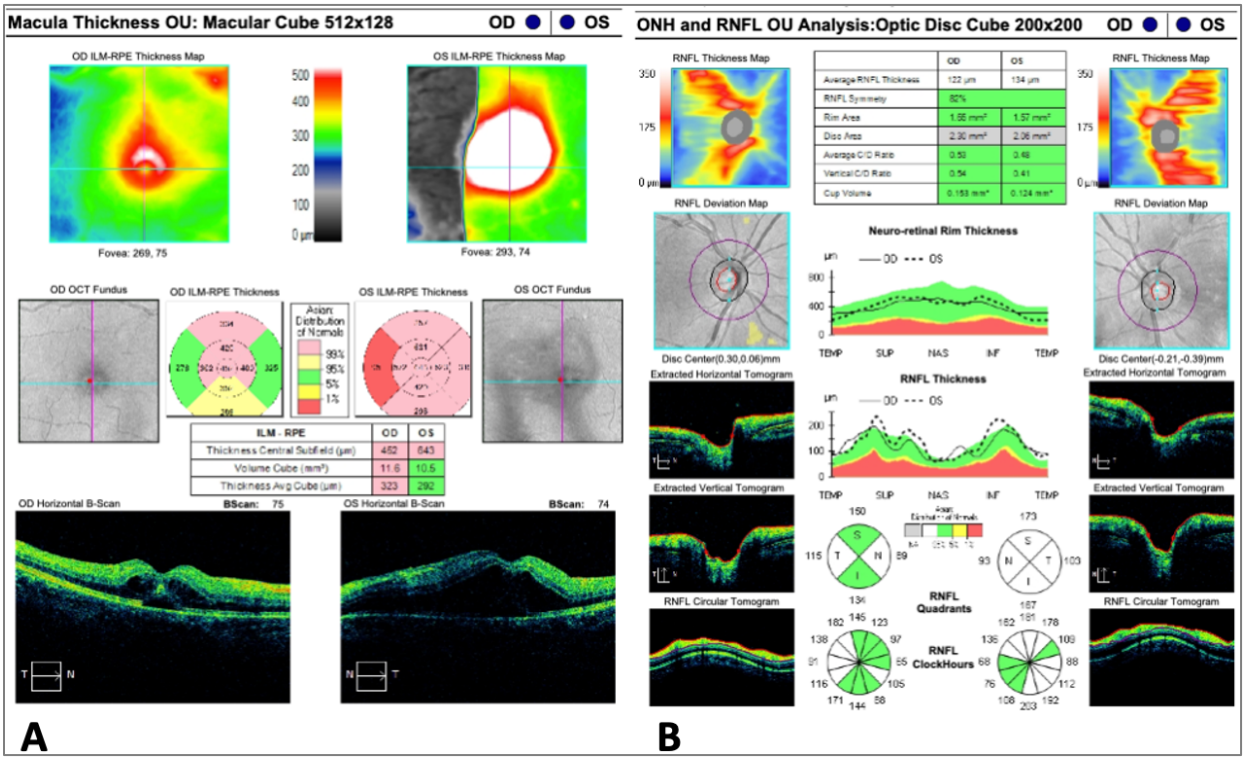

Figure 2 A. OCT of the macula showed a cystoid macular edema in the right eye, neurosensory retinal detachment in the left eye, with increased central subfield thickness in both eyes. **B.** OCT of the optic nerve head showed thickening of the average RNFL in both eyes.

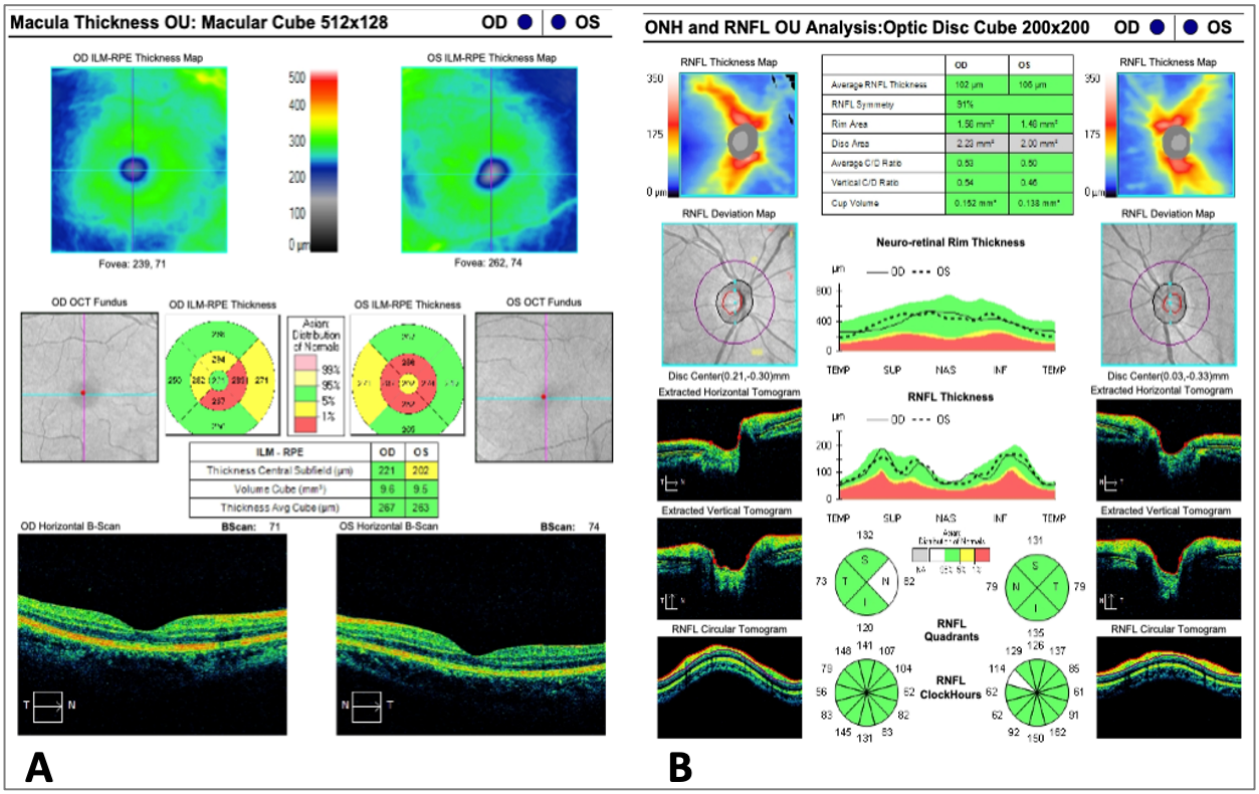

Figure 3. A. Repeat OCT measurements taken after three days of intravenous methylprednisolone pulse therapy showed normal foveal architecture with development of macular thinning in both eyes. **B.** Optic nerve head retinal nerve fiber layer thickness was also within normal limits.

discharged on a tapering dose of oral prednisone, omeprazole, vitamin B complex, and nepafenac eye drops.

Two weeks later, visual acuity had recovered to 20/20 in the right eye and 20/25 in the left eye. The rest of the ocular exam findings were unremarkable.

DISCUSSION

Ocular complications following dengue infection are rare and may include neuroretinitis, anterior uveitis, vascular sheathing, retinal hemorrhage, exudates, macular edema, retinochoroiditis, and optic disc edema.[9-12] In this report, we described the clinical presentation, ocular findings, clinical course, and response to treatment of a patient who developed bilateral neuroretinitis during the convalescent phase of dengue. The presence of mild optic disc swelling, macular edema, and hard exudates supported the diagnosis of neuroretinitis. An immune-mediated mechanism by immune complex formation, molecular mimicry, or autoimmune activation triggered by viral antigens is presumed to underlie the condition rather than direct viral neurotropism, and the response to corticosteroids is typically favorable.[10] This underscores the importance of early recognition and timely treatment. Similar reported cases have shown acute presentations, with most patients recovering spontaneously, although some may experience persistent visual field defects.[9,13-15]

Neuroretinitis has been reported in association with various infectious, inflammatory, vascular, and idiopathic etiologies. It is commonly seen in cat-scratch disease caused by *Bartonella henselae,* and less frequently in conditions such as ocular tuberculosis and syphilis.[16] Additionally, neuroretinitis has also been reported in systemic autoimmune diseases such as sarcoidosis, Behçet disease, systemic lupus erythematosus, and polyarteritis nodosa.[16] In this case, neuroretinitis was presumed to be secondary to dengue infection due to the temporal relation and negative, albeit limited, systemic work-up.

In summary, we reported a case of dengue-associated bilateral neuroretinitis in a young female adult during the convalescent phase of illness. Although rare, this ocular sequela should be considered in dengue-endemic regions in patients who present with isolated ocular symptoms, accompanied by optic disc swelling and macular edema. Prompt diagnosis and treatment with corticosteroids can lead to favorable visual outcomes. Clinicians should maintain a high index of suspicion to prevent long-term visual morbidity associated with delayed treatment.

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