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Case Report

Bilateral acute angle closure attack precipitated with dengue fever

Rajwinder Kaur[1], Mandeep Kaur[1], Priyanka Dahiya[1], Harleen Kaur[1]

[1]Department of Ophthalmology, Adesh Institute of Medical Sciences and Research, Bathinda, Punjab, India.

***Corresponding author:**
Rajwinder Kaur,
Department of Ophthalmology,
Adesh Institute of Medical
Sciences and Research,
Bathinda, Punjab, India.

rajujerry26@gmail.com

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ABSTRACT

Dengue is known to cause varied ocular problems from mild-to-severe sight threatening. We present the case of a 63-year-old female patient with bilateral shallow anterior chamber and acute angle closure attack precipitated during the convalescent phase of dengue infection. She arrived at the emergency department at midnight with a history of vomiting, headache, weakness, and pain in the left eye (LE). Her diagnosis of dengue fever was supported by specific serological testing. On ocular examination, the anterior chamber of both eyes was noticeably shallow and her LE felt stony hard on digital palpation. Intravenous mannitol 300 mL over 45 min were given stat followed by topical medications. In the morning, intraocular pressure measured was 10 mmHg (right eye [RE]) and 26 mmHg (LE). After thorough examination, she was diagnosed with occludable angle in RE and angle closure in LE after gonioscopy. Neodymium-doped Yttrium Aluminum Garnet laser peripheral iridotomy was done in both eyes. She was relieved of symptoms. The varied spectrum of possible ocular symptoms should prompt the ophthalmologist to suspect the potential involvement of eye in dengue fever. Detailed history taking is of utmost importance in early diagnosis of some rare presentation of dengue-related ophthalmic involvement. Increased awareness of dengue-related ophthalmic complications among clinicians involved in the care of patients with dengue would facilitate prompt ophthalmologic assessment and emergency medical care.

Keywords: Glaucoma, Dengue fever, Bilateral angle closure glaucoma

INTRODUCTION

The virus that causes dengue fever is spread through a mosquito bite. Dengue virus (DENV) is a single-stranded ribonucleic acid-enveloped virus. It is regarded as one of the most significant viral infections spread by mosquitos. There are four serotypes and it is a member of the family Flaviviridae and genus Flavivirus (DENV 1–4). When infected female mosquitoes bite people, the virus is spread to them. The principal vector of this virus is the *Aedes aegyptus* mosquito.[1]

The number of dengue cases reported to the World Health Organization increased over 8 fold over the last two decades, from 505,430 cases in 2000, to over 2.4 million in 2010, and 5.2 million in 2019.

The primary sign of dengue is fever, which lasts 3–7 days and is often greater than 38°C in most individuals. The complex multifunction DENV NS1 protein participates in vascular leakage, host immune system evasion, and capsid assembly. Increased vascular permeability is a sign of severe dengue.

Any of the four serotypes (DEN 1-4) of DENV can produce dengue fever, a self-limiting, influenza-like disease. The most severe presentation of the disease, dengue hemorrhagic fever, is marked by thrombocytopenia, increased vascular permeability and plasma leakage. There may also be myalgia, joint discomfort, retrobulbar pain, sore throat, and facial erythema. Ocular symptoms

include ocular pain and decreased vision. Common ocular findings include subconjunctival hemorrhages, macular edema, macular hemorrhage, blot hemorrhages, vasculitis, and optic neuropathy. Exudative retinal detachment, cotton wool spots, and anterior uveitis are seen less frequently.[2]

We describe a case of a bilateral acute attack of angle closure precipitated by dengue fever. In our opinion, the episode of severe dengue is known to cause vascular permeability thus affecting the uveal tissue and has precipitated this acute attack.

CASE REPORT

A 63-year-old woman reported with severe headache, vomiting, and extreme ocular discomfort in her left eye (LE). She complained of blurred vision, colored halos, watering, photophobia, and redness in both eyes for the past 2 days.

She was recovering from dengue fever at the time of presentation which was confirmed by a serological test of dengue NS1 antigen. According to her blood workup over the past 1 week, her platelet count showed a decreasing trend reaching the lowest level of 45000/cumm but started correcting from 3rd day of fever onward reaching normal counts at time of presentation.

She had a history of recurrent episodes of headache and pain in the LE in the past 1 week, which typically lasted for an hour and subsided with sleep or rest. She typically correlates these episodes with dengue. She had no such episodes before dengue.

Her physical examination revealed mild dehydration, a body temperature of 95°F, blood pressure (BP) of 110/70 mm Hg, and no signs of rash, jaundice, or stiffness of the neck. Blood tests revealed hemoglobin levels of 13.3 g%, platelet counts of 1.1 lakhs/cumm, white blood cells levels of 7730/cumm, serum glutamic oxaloacetic transaminase 60 IU/L, and serum glutamic pyruvic transaminase76 IU/L. Urine routine and microscopy were normal.

She arrived in emergency department at 3 A.M. at our institute. Ophthalmology consultation was done in view of her ocular symptoms. Bedside examination showed visual acuity of more than 20/100 in the right eye (RE) and 20/200 in the LE. Cornea was clear in the RE but mild haze was observed in LE. Pupillary reaction in the RE was normal but LE showed non-reacting, fixed, and mid dilated pupil. On digital tonometry, the LE was stony hard indicative of elevated intraocular pressure (IOP). On oblique torch illumination test, both eyes revealed a shallow anterior chamber (LE>RE), Eclipse sign was positive in both eyes left more than RE [Figures 1 and 2]. Bilateral circumcorneal congestion and immature cataracts were observed in both eyes. Fundus examination revealed small and crowded disc with cup disc ratio 0.1:1 in undilated pupil and mid dilated pupil in the right and LE, respectively.

A clinical diagnosis of bilateral acute angle closure attack was made. Detailed history was taken to rule out any ocular and systemic medications to rule out other causes of secondary angle closure glaucoma. Any allergy to drugs especially sulfa drugs was also asked. Two tablets of acetazolamide were given stat.

After checking the vitals (BP was 120/60) heart rate – 75 beats/min, intravenous mannitol 20% (5 mL/kg body weight), 200 mL was given over 45 min along with intravenous fluids 500 mL normal saline (0.9%) at the rate of 70 mL/h, and topical antiglaucoma medications (brimonidine 0.2% and timolol 0.5% eye drops) were started in the LE. After an hour of therapy, the patient had symptomatic relief. Patient refused

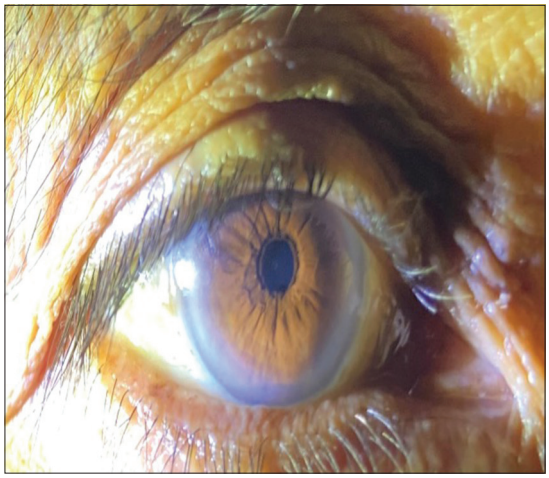

Figure 1: Right eye shows shallow anterior chamber with anterior chamber depth Van Harrick Grade 1.

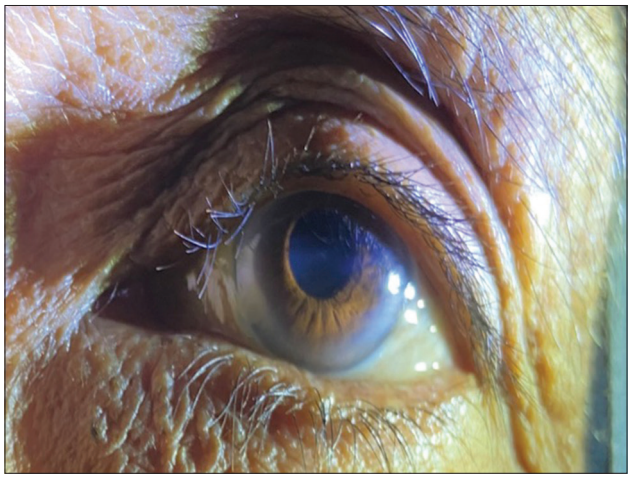

Figure 2: Left eye shows shallow anterior chamber with anterior chamber depth Van Harrick Grade 0.

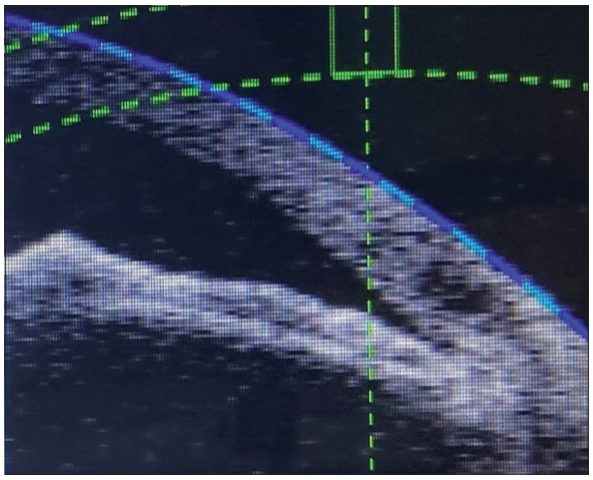

Figure 3: Anterior segment optical coherence tomography shows shallow anterior chamber.

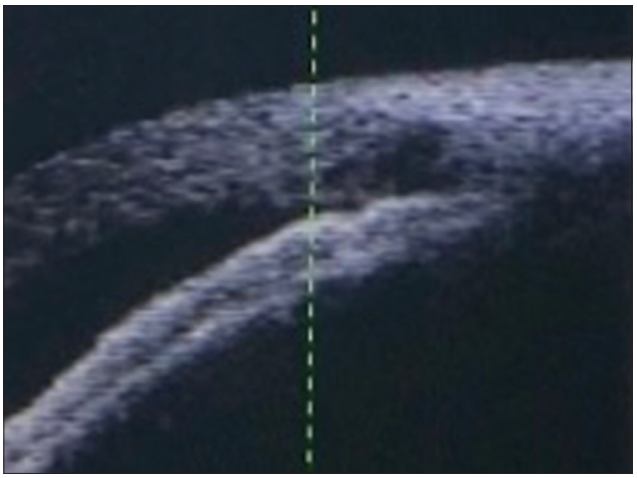

Figure 4: Anterior segment optical coherence tomography shows iridocorneal touch.

admission and was told to follow-up early next morning. Tablet acetazolamide was started qid and patient was asked to report in the morning.

Next day, the patient's uncorrected vision was 20/40 in the RE with no pinhole improvement, and 20/80 in LE, improving up to 20/40 with refractive correction of +0.75 DS R/E and no improvement with refractive error of +1.50 DS L/E Near add each eye was +2.25DS. Central corneal thickness (RE 489 μ, LE 528 μ measured with IOL MASTER 700) correction her corrected IOP measured by Goldmann applanation tonometry was found to be 16 mmHg in the RE and 25 mmHg in the LE on eye drops brimonidine and timolol at 10 AM. The pupillary reaction was normal in the RE and sluggish and mid dilated in the LE. On slit lamp examination, there was circumcorneal congestion in the LE. RE cornea was clear and LE showed mild microcystic edema. Anterior chamber depth was Van Harrick Grade1 in RE and Grade 0 in LE. Anterior chamber was quiet. Anterior lens capsule showed glaucomflecken with immature senile cataract nuclear sclerosis grade 2 in both eyes. Gonioscopy done with Goldmann 3 mirror revealed appositional closure in temporal and superior quadrant and 180° complete closure in RE. There was 360° complete angle closure in L/E. No angle structures could be seen even on indentation. The RE revealed only Schwalbe's line in all the angles.

On Carl Zeiss IOL master 700, the axial length was 22.81 mm in R/E and 22.12 mm in L/E with anterior chamber depth of RE 2.41 mm and LE 2.25 mm. Anterior segment optical coherence tomography showed iridocorneal touch [Figures 3 and 4].

A final diagnosis of bilateral acute angle closure attack was made. Bilateral Neodymium-doped Yttrium Aluminum Garnet peripheral laser iridotomy (PI) was performed in both eyes after IOP lowering. This was followed by eye drops brimonidine (0.2%), timolol (0.5%) twice daily, and prednisolone acetate (1%) 4 times a day in both eyes for 2 weeks.

Post-PI the IOP reduced to 16 and 20 mmHg in the right and LE, respectively. Five days later, patient was asymptomatic with best corrected visual acuity of 20/40 in both eyes. After 3 weeks, her post-PI IOP was 14 and 16 mmHg in the right and LE, respectively, without oral and topical treatment.

DISCUSSION

The pathogenesis of ocular complications in dengue fever is not fully understood. DENV can manifest clinically in the eye as either ocular inflammation (ciliary congestion, shallow AC, and anterior uveitis) or as hemorrhagic complications (subconjunctival hemorrhage and macular hemorrhage).[3] In addition, posterior segment maculopathy and dengue-related foveolitis is present, which is characterized by a disruption of the outer neurosensory retina in optical coherence tomography.[2]

Iritis, uveitis, glaucoma, and retrobulbar neuritis were listed as consequences in a 1929 paper by Bistis (1929, apud Richardson) about the dengue epidemic in Athens. During the same outbreak, anargyros (1929, apud Richardson) documented five cases with ocular sequelae. In one case report, glaucoma of the LE developed on the fourth and final day of the fever, concurrently with the onset of the exanthem and a less severe bout of glaucoma of the RE developed 2 days later. Iridectomy was done, first in the left, then in the RE.[4]

Our patient presented with bilateral acute angle closure attack during convalescent phase of dengue which was confirmed by serological tests.

Capillary leak syndrome occurs in patients infected with DENV and is the leading cause of morbidity. In this condition, the host's excessive inflammatory response

leads to a cascade of cytokine production that results in an increased vascular permeability with plasma extravasation, hemoconcentration, and in severe cases, shock. There are few reported cases of bilateral secondary angle closure glaucoma secondary to ciliary body swelling and anterior rotation in dengue capillary leak syndrome.

However, our case was an elderly female with immature cataract and hypermetropia, which are known predisposing risk factor for acute angle closure glaucoma. She developed bilateral acute attack of angle closure glaucoma precipitated by dengue fever during convalescent stage of disease.[5-8]

The possible mechanism of glaucoma in our case was iris swelling secondary to transudation of fluid or iritis post-dengue resulting in worsening of pupillary block. This initiated the attack of angle closure in both eyes, left more than right.

Limitation

Ultrasound biomicroscopy is gold standard for imaging of angles. However,due to its unavailability we have documented angle structures using IOL Master.

CONCLUSION

Detailed history taking is of utmost importance in early diagnosis of some rare presentation of dengue-related ophthalmic involvement. Increased awareness of dengue related ophthalmic complications among clinicians involved in the care of patients with dengue would facilitate prompt ophthalmologic assessment and emergency medical care.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent.

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Nil.

Conflicts of interest

There are no conflicts of interest.

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