Unusual presentation of severe dengue: Dengue maculopathy

Kee Huat Chuah, MRCP[1], Christina Wei Khee Ng, MBBS[2,3], Zabri Kamarudin, MMed(Ophth)[2], Chi Lun Wong, MMed(Ophth)[4]

[1]Department of Medicine, Hospital Kulim, [2]Department of Ophthalmology, Hospital Selayang, [3]Department of Ophthalmology, Universiti Kebangsaan Malaysia Medical Centre, [4]Department of Ophthalmology, Hospital Kulim

SUMMARY

Dengue infection is epidemic but ocular complications of dengue are less reported. We report a case of dengue-infected patient with disturbing ocular symptoms. He was admitted for severe dengue. Subsequently, he presented with symptoms of bilateral central scotoma with metamorphosia. Fundus examination revealed cotton wool spots and flame-shaped haemorrhages at the macula, with dull foveal light reflex. He was diagnosed with bilateral dengue maculopathy. The patient was managed conservatively with surveillance. One week later, his eye symptoms improved and were resolved six months later. Early recognition and close monitoring remains the key to successful management and interventions are rarely needed.

KEY WORDS:
Dengue ; maculopathy; eye disease

INTRODUCTION

Dengue infection is epidemic in this region and is associated with high morbidity and mortality. The disease can range from a mild self-remitting febrile illness requiring only outpatient treatment; to severe dengue with plasma leakage, bleeding complications or multiorgan dysfunction that require intensive care unit (ICU) management and can even lead to death. On the other hand, another less known complication, ocular manifestation of dengue, could be very disabling. Dengue eye disease may manifest as maculopathy, sub-conjunctival haemorrhage, vitreous and retinal haemorrhage, posterior uveitis or optic neuritis; with symptoms of blurring of vision, scotoma, metamorphosia or floaters.[1] Here we are reporting an interesting case of a severe dengue patient with hepatitis, who presented with the rare complication of dengue maculopathy.

CASE REPORT

Our patient is a 25-year-old gentleman, with no comorbid illnesses, who was admitted and treated for severe dengue with severe hepatitis. He initially presented with fever for two days with no warning signs. The diagnosis of dengue fever was confirmed with positive NS 1 antigen test. At admission, his total white cell count was 2.55 x 10^9/L, haematocrit 41.8%, platelet count 85 x 10^9/L, alanine transaminase (ALT) 42 IU/L and aspartate transaminase (AST) 40 IU/L. His condition worsened with warning signs and severe hepatitis on day five of illness, during the defervescence / critical phase, and was transferred to ICU. His lowest white cell count was 1.52 x 10^9/L and platelet count was 15 x 10^9/L. His highest haematocrit was 45.5%, ALT was 873 IU/L and AST was 1302 IU/L. He was treated with multiple fluid boluses and maintenance drip. He recovered well and was transferred out of ICU on day six.

At 40 hours of defervescence, on day seven of illness, the patient complained of bilateral scotoma and metamorphosia while attempting to read the newspaper. He was referred to the ophthalmology team. Ocular assessment revealed bilateral visual acuity of 6/6, normal visual field by confrontation test, light brightness of 80-90% in the right eye and 100% in the left eye. Fundus assessment showed multiple cotton wool spots at bilateral macula and flame-shaped haemorrhage on the left, with bilateral dull foveal light reflex (Figure 1). He was diagnosed with dengue maculopathy. He was managed conservatively with surveillance and reassurance was given.

One week later, his symptoms improved significantly with minimal residual scotoma in the right eye and complete recovery in the left. Optical coherence tomography and fundus fluorescein angiography were not done due to unavailability of the service. Besides that, the patient was not keen to have the tests done elsewhere since the symptoms were resolving spontaneously. In subsequent follow up visits, the patient's symptoms of central scotoma gradually cleared and were completely resolved by six months. Bilateral fundus examination revealed residual cotton wool spots. Previously seen flame-shaped haemorrhages had resolved completely.

DISCUSSION

In Singapore, prevalence of dengue maculopathy among hospitalised patients with dengue fever is 10.0%. However, only 24.1% of them reported visual symptoms.[2] Therefore, because of the lack of symptoms, ocular manifestations are perceived to be rare and the awareness among clinicians is low.

This article was accepted: 7 December 2016
Corresponding Author: Kee Huat Chuah, Department of Medicine, Hospital Kulim
Email: chuah319@yahoo.com

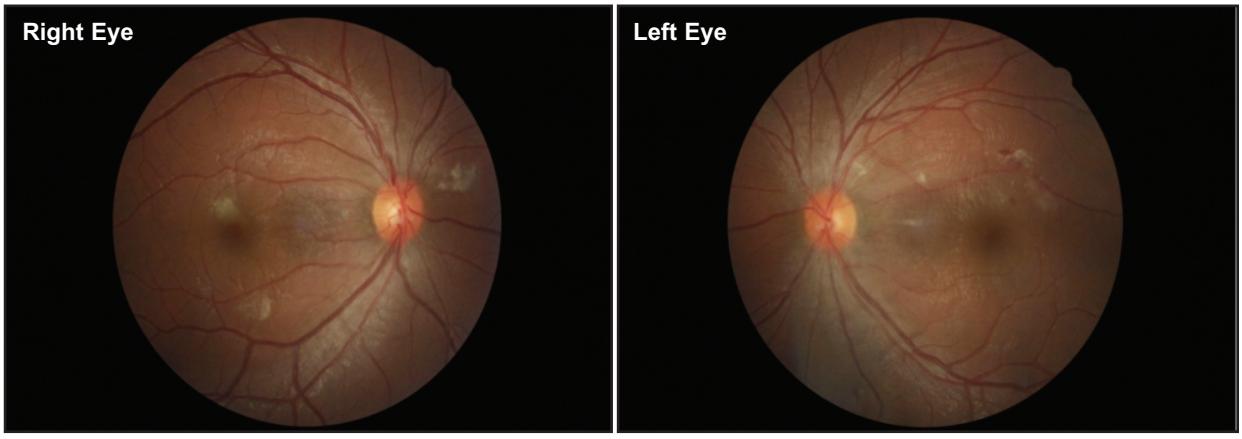

Fig. 1: Fundus photography of the patient with bilateral dengue maculopathy.

Dengue eye disease can affect either one or both eyes. It can occur as early as two days, and up to five months from the start of the fever. However, it usually happens within one day of the nadir of thrombocytopenia, which is estimated to be seven days after the onset of fever.[1] This is evident in our patient who presented with the symptoms on day seven while his platelet count was at its lowest on day six of the disease. Pathophysiology of the disease is not well understood. It is postulated to be due to multiple mechanisms, including haemorrhage due to thrombocytopenia and immune mediated reactions.[1]

Dengue maculopathy is the presence of macular swelling, haemorrhages, and yellow spots at the macula due to retinal or choroidal vasculopathy. The symptoms include blurred vision (51.2-87%), scotoma (29.1-63%), floaters (1%), and rarely, micropsia and metamorphopsia.[1] It can present as macula oedema (76.9%), macular haemorrhage (69.2%), foveolitis (28-33.7%), vasculitis or vascular occlusion.[1] Slit lamp biomicroscope, fundus camera and fundoscope are utilised to visualise the fundus while Amsler chart, Humphrey visual field analysis and microperimetry can be used to map the visual field defect or scotoma. As dengue maculopathy may not be evident clinically, several modalities are essential in diagnosis, assessing the severity and disease monitoring. These include optical coherence tomography to evaluate macula thickness and oedema; fundus fluorescein angiography and indocyanine green chorioangiography to look for vasculopathy. Unfortunately, in district hospitals with limited resources, diagnosis and monitoring are mainly by clinical assessment with the aid of slit lamp biomicroscope and fundus camera. Nevertheless, this should not refrain one from referring the cases to ophthalmologists. In severe or complicated cases, patients would be further referred to the Medical Retinal team for evaluation and opinion.

Prognosis of the eye disease ranges from complete spontaneous recovery to poor vision despite therapy.[1] Most cases of ocular involvement are self-limiting and resolve spontaneously without treatment.[3] To date, there is no randomised controlled trial, and there is no known effective treatment. However, any maculopathy or insult to the macula and the underlying retinal pigment epithelium may subject the patient to future development of choroidal neovascularisation. These abnormal sub-retinal new vessels can eventually bleed and lead to macula scar if left untreated. When this occurs, permanent blindness will ensue. Therefore, continuous monitoring is recommended until symptoms completely resolve. However, data on duration of follow up is still lacking.[5]

For cases of active maculopathy with persistent symptoms up to six months, corticosteroid therapy might be helpful.[1] As the underlying mechanism is likely to be immune mediated, corticosteroid therapy may have an effect on the recovery by preventing structural damage and permanent visual loss due to ocular inflammation. Cases with intra-retinal vascular or choroidal leakage, signs of active ocular inflammation, and foveal swelling are more likely to benefit from steroid therapy.[4] Larger studies are needed to validate and justify its usage as corticosteroid therapy do come with its own side effects. Mode of therapy, dosage and duration remain areas of concern.

In conclusion, most dengue maculopathy will improve spontaneously with time. However, close monitoring for ocular sequelae of dengue maculopathy such as choroidal neovascularisation and persistent macula oedema as a resultant of active disease is warranted to prevent irreversible visual damage. Larger epidemiological studies are needed to delineate the natural course of dengue eye disease. Physicians should be aware of this manifestation and be able to initiate adequate treatment, which leads to excellent functional and anatomical results.

REFERENCES

1. Ng AW, Teoh SC. Dengue eye disease. Surv Ophthalmol 2015; 60(2): 106-14.
2. Su DH-W, Bacsal K, Chee S-P, Flores JV, Lim WK, Cheng BC, et al. Prevalence of dengue maculopathy in patients hospitalized for dengue fever. Ophthalmology 2007; 114(9): 1743-7.
3. Teoh SC, Chan D, Nah G, Rajagopalan R, Laude A, Ang BS et al. A re-look at ocular complications in dengue fever and dengue haemorrhagic fever. Dengue bulletin 2006; 30: 184-90.
4. Bacsal KE, Chee SP, Cheng CL, Flores JV. Dengue-associated maculopathy. Arch Ophthalmol 2007; 125(4): 501-10.
5. Veloso CE, Schmidt-Erfurth U, Nehemy MB. Choroidal neovascularization induced byimmunogenic alteration of the retinal pigment epithelium in dengue Fever. Case Rep Ophthalmol 2015; 6(1): 18-23.