



Retinal maculopathy in an adult with yellow fever

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A previously healthy 53-year-old Brazilian man was admitted to hospital with fever, nausea, vomiting, severe hepatic dysfunction with jaundice, as well as thrombocytopenia and altered coagulation tests (19 000 platelets per μL ; partial thromboplastin time and prothrombin time was incoagulable; alanine aminotransferase 5204 units per L; and aspartate aminotransferase 6250 units per L). Serologies for toxoplasmosis, syphilis, leptospirosis, cytomegalovirus, and hepatitis A, B, and C viruses were all negative. Yellow fever was confirmed by two separate PCRs, on Feb 3, 2018, 7 days after symptom onset, and on Feb 5. An ELISA-IgM for yellow fever done on Feb 5 confirmed the diagnosis, whereas other arboviruses such as dengue, Zika, and Chikungunya were also excluded by PCR on the same day.

The patient reported discrete progressive decline in visual acuity in his left eye with the onset of yellow fever symptoms, with sudden loss of vision in this eye 19 days later, at which point an ophthalmologist was consulted (Feb 14, 2018). The loss of vision was not accompanied by hyperaemic conjunctivas or ocular pain. His best corrected visual acuity was 20/20 in the right eye and counting fingers in the left eye. Upon examination, the right eye was normal. The left eye had a normal anterior chamber and 3+/4+ cells in the anterior vitreous, with macular exudates and haemorrhages. In the left eye, optical coherence tomography showed retinal oedema and underlying subfoveal hyper-reflective deposits, and

fluorescein angiography showed capillaritis and late leakage in the perifoveal area (figure).

Correlating with the mechanisms of dengue maculopathy, which are mediated by the host's immune response in the late stages of infection and not directly by viral invasion, ocular manifestations in the convalescent stage of yellow fever infection point towards a similar immune-mediated mechanism. Hence, 1 mg/kg per day (60 mg) of oral prednisone was prescribed and maintained for 14 days and was subsequently tapered by 10 mg/day every week until the dose reached 5 mg/day. A month after steroid treatment was started, best corrected visual acuity in the left eye improved to 20/100, haemorrhages absorbed, and exudates reduced.

To our knowledge, this is the first report of a maculopathy that is presumably associated with yellow fever confirmed by positive serous PCR.

Contributors

OAZ contributed to the acquisition and interpretation of the data; drafting the work and revising it critically for important intellectual content; and final approval of the version to be published. FMCM, DSdC, and RB Jr contributed to the conceptualisation and design of the work, critical revision, and final approval of the version to be published. HSB was responsible for acquisition and interpretation of the data, critical revision, and final approval of the version to be published.

Declaration of interests

We declare no competing interests.

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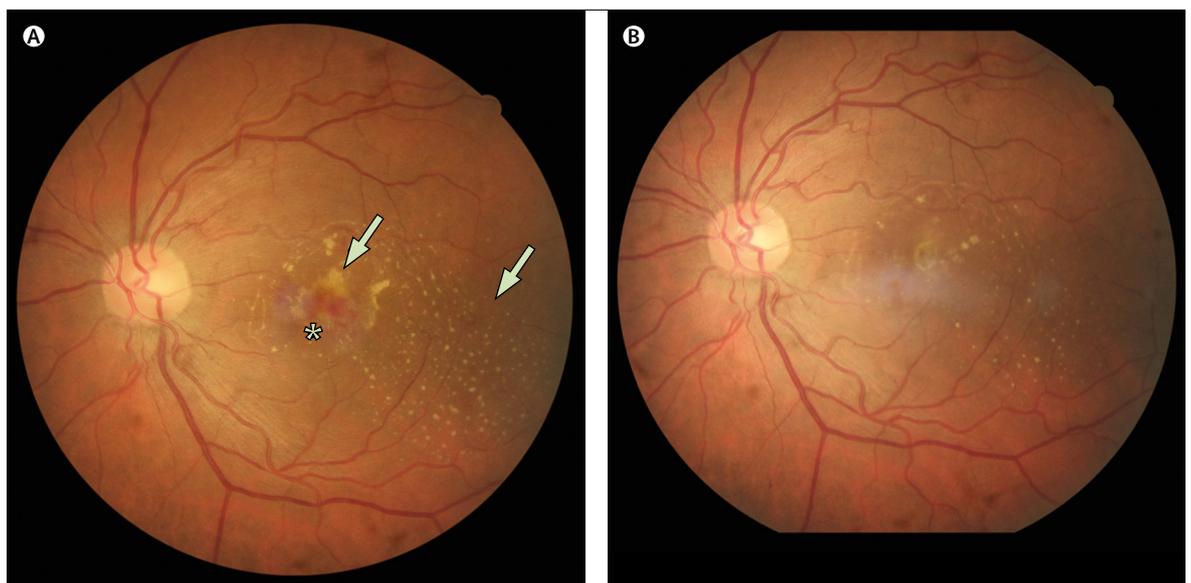


Figure: Retinal fundus image of the left eye

(A) Macular and extra-macular exudates (arrows) and macular haemorrhage (asterisk) are visible 19 days after the onset of yellow fever symptoms. (B) Haemorrhages were absorbed, and exudates were reduced 1 month after oral prednisone treatment was started.