PRESENTATION

A 17-year-old female patient with a history of malaria fever, caused by Plasmodium falciparum, presented with sudden decrease of vision in her left eye 10 days after the onset of fever to an Ophthalmologist elsewhere. The patient was referred to our center one month later. On examination, vision was 20/20 and 20/1200 in the right and left eyes, respectively. The ophthalmic examination of the right eye was unremarkable. The left eye had relative afferent papillary defect and fundus examination revealed retinal hemorrhages, superior branch retinal vein occlusion, and neovascularization. Fluorescein angiography (FA) revealed venous obliteration along the superior arcade, with large areas of capillary dropout in the superior and temporal periphery [Figure 1]. The areas of interest were intravascular micro-emboli (coiled structures) along the superior veins (arrows) on FA [Figure 2]. The resultant venous obstruction and capillary dropout areas were noted in the corresponding peripheral retina which was drained by these vessels, as observed on FA [Figure 3]. The superior vein obliteration was immediately distal to the micro-embolus (at the bifurcation).

DISCUSSION

Microvascular occlusion has been postulated to be the primary pathogenic mechanism in cerebral malaria and malaria retinopathy. Occlusion usually results from cohesiveness of infected and non-infected erythrocytes. Histologically, sequestration of infected and non-deformable erythrocytes has been observed in previous studies. The in vivo appearance of micro-emboli

Figure 1. Mid-phase fundus fluorescein angiography of the left eye shows venous obliteration along the superior arcade, with large areas of capillary dropout in the superior and temporal periphery. Neovascular fronds are seen along the superior arcade retinal veins; blocked fluorescence is noted along the inferior arcade due to subhyaloid hemorrhage.

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in the retinal vessels infected with malaria has not been reported before. FA images presented herein characteristically show intra-vascular coiled structures with resultant peripheral capillary dropout. We postulate that the coiled structures seen in retinal vessels may represent an aggregation of infected erythrocytes, which may get stained with FFA due to sluggish blood flow. Although the origin and significance of these coiled structures are unknown, their location corresponded well to the vessel obliteration, implying intravascular micro-emboli. The extensive new vessel formation early in the course of the disease appears to be due to the amount of ischemia related to extensive vascular occlusion due to multiple micro-emboli in the retinal circulation.

REFERENCES


Figure 2. The area of interest from Figure 1 shows intravascular micro-emboli (coiled structures) along the superior veins (yellow arrows) seen as hyperfluorescent intravascular lesions.

Figure 3. Superior and temporal quadrants of the left eye on late-phase fluorescein angiography show venous obstruction and capillary dropout areas in the corresponding peripheral retina drained by these vessels as shown in Figure 2. The superior vein obliteration is seen immediately distal to the micro-embolus (at the bifurcation, yellow arrows).

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