

The hallmark of pediatric cerebral malaria (CM) is sequestration of parasitized red blood cells in the cerebral microvasculature. Malawi-based research using 0.35 Tesla (T) magnetic resonance imaging (MRI) established that severe brain swelling is associated with fatal CM, but swelling etiology remains unclear. Autopsy and clinical studies suggest several potential etiologies, but limitations of 0.35 T MRI precluded optimal investigations into swelling pathophysiology. A 1.5 T MRI in Zambia allowed for further investigations including susceptibility-weighted imaging (SWI). SWI is an ideal sequence for identifying regions of sequestration and microhemorrhages given the ferromagnetic properties of hemozoin and blood. Using 1.5 T MRI, Zambian children with retinopathy-confirmed CM underwent imaging with SWI, T2, T1 preand post-gadolinium, diffusion-weighted imaging (DWI) with apparent diffusion coefficients and T2/fluid attenuated inversion recovery sequences. Sixteen children including two with moderate/severe edema were imaged; all survived. Gadolinium extravasation was not seen. DWI abnormalities spared the gray matter suggesting vasogenic edema with viable tissue rather than cytotoxic edema. SWI findings consistent with microhemorrhages and parasite sequestration cooccurred in white matter regions where DWI changes consistent with vascular congestion were seen. Imaging findings consistent with posterior reversible encephalopathy syndrome were seen in children who subsequently had a rapid clinical recovery. High field MRI indicates that vascular congestion associated with parasite sequestration, local inflammation from microhemorrhages and autoregulatory dysfunction likely contribute to brain swelling in CM. No gross radiological blood brain barrier breakdown or focal cortical DWI abnormalities were evident in these children with nonfatal CM.