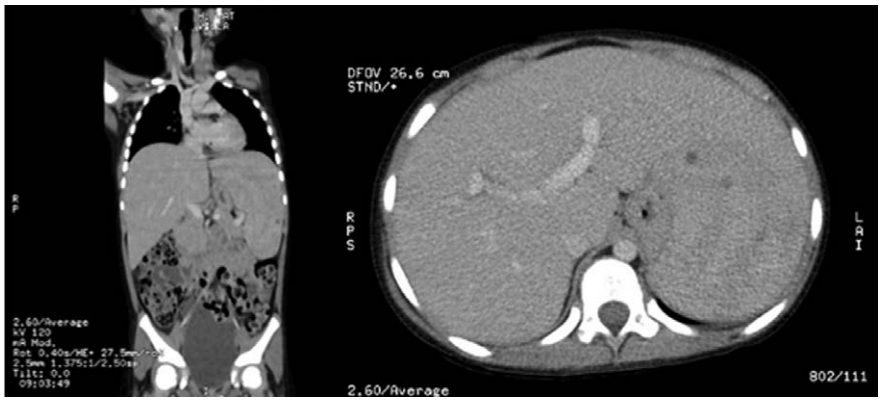

FIGURE 1. Axial computed tomography (CT) demonstrating splenomegaly in 13.8 cm with multiple hypodense images of 8 mm diameter.

a favorable response after 48 hours. On reassessment, the patient's mother referred consumption of unpasteurized dairy products. Rose Bengal was positive (>1:1,280), as well as confirmatory 2-Mercaptoethanol. Due to the persistent abdominal pain, a CT scan was performed, revealing multiple splenic abscesses of approximately 8 mm each. Treatment with doxycycline and gentamicin was initiated, showing a rapid and favorable response. After 10 days of incubation, initial blood cultures reported Gram-negative coccobacilli with nonhemolytic white colonies on Agar plates, and pan-susceptible *Brucella melitensis* was identified. The patient was discharged after 10 days with oral doxycycline and rifampin for 6 weeks. Serological testing of the household members was negative. After 6 months, the patient remained asymptomatic with no signs of relapse. Follow-up ultrasonography revealed complete resolution of the splenic abscesses and pericardial effusion (Fig. 1).

We present a case of Brucellosis with uncommon manifestations that initially resembled MIS-C. Brucellosis is caused by members of the genus Brucella, most commonly by *B. melitensis*. Common manifestations include fever, fatigue, lumbar pain and night sweats. *Brucella spp*. is transmitted by ingestion of infected animal products, most commonly unpasteurized dairy. Data suggests that splenomegaly and cardiac involvement are infrequent (1.8% and 0.7%, respectively).[1,2] Likewise, splenic abscesses are extremely rare, with only 4 available reports worldwide since 1995.[3–6] Although these manifestations are uncommon, they do not seem to confer higher mortality risk. Due to epidemiologic context and mimicking signs and symptoms, our patient was initially diagnosed with MIS-C. It is worth noting that, to diagnose MIS-C, a complete and adequate infectious assessment is required. Current trends suggest a global decrease in the incidence of MIS-C[7], which should be taken into consideration while evaluating differential diagnoses.

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Cerebral Malaria and Cytotoxic Lesions of the Corpus Callosum

To the Editors:

Malaria is a common cause of morbidity and mortality worldwide. According to the WHO, 229 million cases and 409,000 deaths were reported worldwide in 2020.[1] Children account for a high burden of malaria-related mortality and are at a higher risk of developing cerebral malaria (CM).[1]

A 12-year-old, previously healthy female from Guinea-Bissau was admitted 1 day after arriving in Portugal with a 2-day history of headache, generalized myalgia, and high-grade fever. Physical examination was unremarkable, except for pallor, icteric sclerae, and nontender hepatomegaly (4 cm). The patient was hemodynamically stable (BP, 101/52 mmHg; HR, 111 bpm), but shortly after admission, vomiting, lethargy, and impaired consciousness were observed (Glasgow coma score 11). She presented with anemia (hemoglobin, 10.0 g/dL; normal reference [NR]: 12.0–16.0), thrombocytopenia (78×10^9/L; NR: 150–450) and significant elevation of inflammatory markers (C-reactive protein, 301.0 mg/L; NR, <5; and procalcitonin, >100 ng/mL; NR, <0.1). No electrolyte disturbances, hypoglycemia, or acidosis. *Plasmodium falciparum* blood smear was positive, and severe malaria was diagnosed due to high parasitemia (28.5%/erythrocytes–11.872.000 parasites/μL) and central nervous system (CNS) involvement other infections were excluded.

The cerebrospinal fluid revealed pleocytosis (15 cells), generalized slowing on the electroencephalogram, and a lesion in the splenium of the corpus callosum with T2 hypersignal and restricted diffusion (Fig. 1), compatible with a cytotoxic lesion of the corpus callosum (CLOCCs) on brain magnetic resonance imaging (MRI). CM was diagnosed based on severely impaired consciousness, CLOCCs, and no other identifiable causes of coma.

The patient was treated with parenteral quinine and clindamycin, due to the immediate unavailability of artesunate, plus

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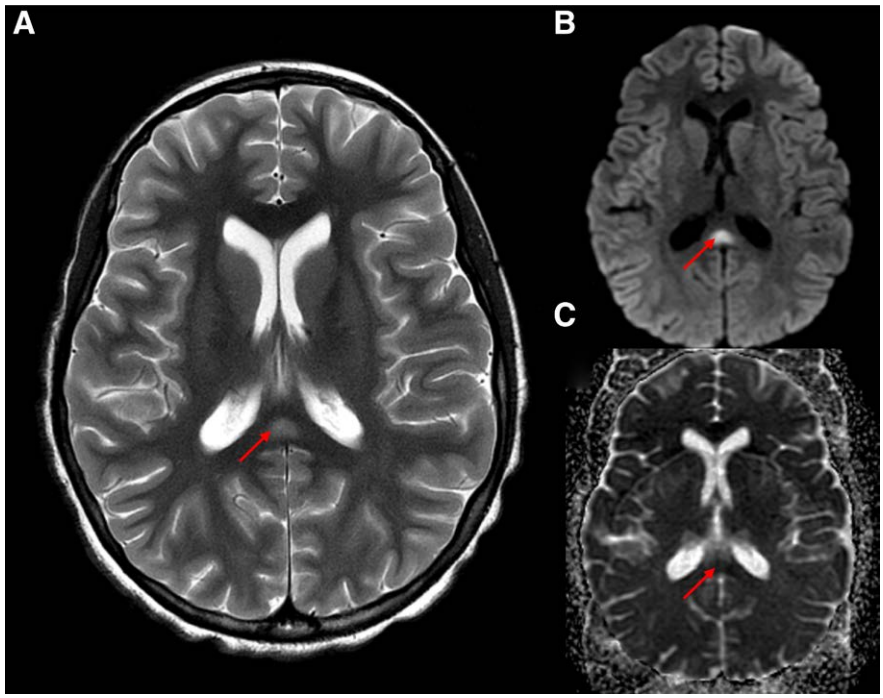

FIGURE 1. MRI axial images T2 weighted (A), DWI (B) and ADC map (C): oval lesion in the splenium of the corpus callosum (arrow) with T2 hypersignal and diffusion restriction with DWI hypersignal and ADC map hyposignal, corresponding to cytotoxic edema.

ceftriaxone. She became afebrile with progressive improvement in neurological status after 48 h and complete parasite clearance on day 3. Artemether-lumefantrine was initiated on day 5, and the patient was discharged on day 8.

CM is defined as a disturbance of consciousness in a patient with *Plasmodium* parasitemia and no other identifiable cause.[2] Despite the reported low mortality rate of pediatric imported malaria in Europe (<1%), CM entails a risk of life-threatening acute CNS effects with diffuse encephalopathy and often rapidly progressive coma, as observed in our patient.[3]

Distinctive brain MRI features involving both white and gray matter structures have been reported in a large cohort of Malawian children with CM. Increased volume, T2 hyperintensity, and Diffusion-weighted imaging (DWI) abnormalities affecting the cortical area (61.7%), subcortical and periventricular white matter (71.7%), basal ganglia (84.2%) and the corpus callosum (49.2%) were the most frequent features.[4] CLOCCS was a common MRI finding in CM, as suggested by DWI/Apparent diffusion coefficient mismatch with DWI increased signal and Apparent diffusion coefficient low signal,[4] as observed in our patient (Fig. 1). The corpus callosum, mostly the splenium, is a region vulnerable to inflammatory cytokinopathy, driven by malaria, seizures or other causes.[5] The higher density of cytokines and glutamate receptors in this area leads to high extracellular glutamate levels, inducing cytotoxic edema upon infection, which is frequently reversible.[5]

CM in children is the most severe complication of *P. falciparum* infection and needs a high suspicion index in nonendemic countries. Altered MRI and CLOCCs, although unspecific, are associated with CM.

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Treatment of Parapneumonic Effusion in Children

To the Editors:

We read the article by Lohuis et al[1] regarding conservative treatment of parapneumonic effusion in children. In their article, the authors stated that the greater amount of children with parapneumonic effusion (PPE) could be treated conservatively with antibiotics only, especially in the absence of mediastinal shift, pleural septations/pockets, pleural thickening or extensive effusions. In a cohort of 136 patients, 117 patients (86%) were treated conservatively and 19 (14%) underwent pleural drainage. They found patients undergoing pleural drainage had mediastinal shift more frequently compared with conservatively treated patients (58% vs. 3%, difference 55%; 95% confidence interval: 32%–77%). In the study patients treated conservatively, median

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