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Intracellular hemozoin pigments and merozoites in severe *Plasmodium falciparum* cerebral malaria

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Clinical image description

A 40-year-old Chinese male returning from Ethiopia presented to the emergency department with a two-week history of low-grade fever and generalized convulsions. This led to a sustained coma with a Glasgow Coma Scale (GCS) score of 3/15 and decerebrate rigidity. His initial laboratory examination results were as follows: total leukocytes, 7.53 × 10^9/L; hemoglobin concentration, 61 g/L; platelet count, 10 × 10^9/L; lactate dehydrogenase, 454 U/L (120-250); creatine kinase, 1457 U/l (55–170); alanine aminotransferase, 29 U/l (0–33); aspartate aminotransferase, 32 U/l (0–32). His coagulation profile showed a prothrombin time (PT) > 100 sec (reference range 9.2-15 sec), activated partial thromboplastin time (APTT) >200 sec (reference range 21-37 sec), fibrinogen <0.25 g/L (reference range 21-37 g/L), and D-Dimer > 40 µg/mL (reference range 0.00-0.55 µg/mL).

Because his laboratory findings were characterized by severe thrombocytopenia and anemia, we immediately proceeded with a smear review. The result of the smear review revealed severe thrombocytopenia and anemia originating from infection with *Plasmodium*. Wright-Giemsa-stained peripheral thin blood smears showed the presence of malarial pigment (hemozoin) within monocytes and neutrophils, neutrophils with phagocytosed merozoites, ring-infected RBCs, and schizonts (Figure 1A-M). Furthermore, Giemsa-stained thick blood smears showed the presence of malarial pigment (hemozoin) within monocytes and a large number of small trophozoites (Figure 1N-P). Several infected RBCs had 2-3 small trophozoites/cells. Brain computed tomography revealed diffuse cerebral edema. The patient was intubated immediately. Despite adequate treatment, his clinical condition rapidly deteriorated and he died within six hours. The remaining blood specimen was analyzed by pan-human Plasmodium polymerase chain reaction confirming *P. falciparum* infection. The most striking feature of this case is the presence of schizonts and intracytoplasmic hemozoin due to *P. falciparum* malaria infestation.

Hemoglobin is the major nutrient source for *Plasmodium* species during malarial blood infection. One of the by-products released from the breakdown of hemoglobin, free heme, is detrimental to the malaria parasite because it can generate reactive oxygen species (ROS) that induce oxidative stress leading

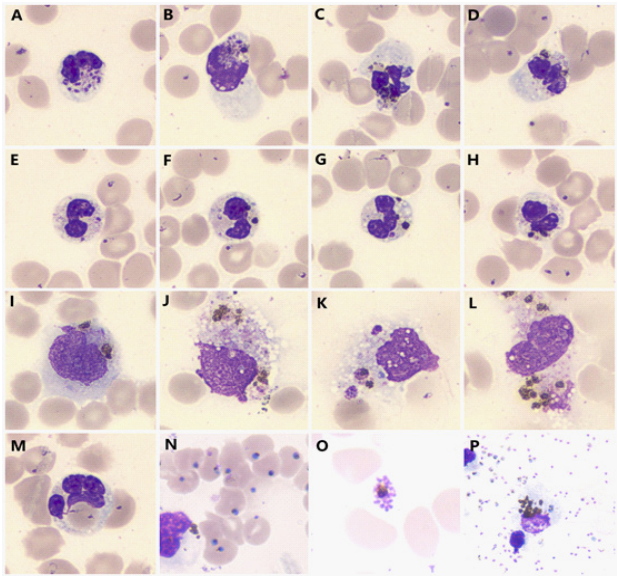

Figure 1: Peripheral thin blood smears showing phagocytosis of merozoites by neutrophils (Figure 1A-D), the presence of brown hemozoin pigments in neutrophils (Figure 1E-H), brown hemozoin pigments in monocytes (Figure 1I-L), and ring-infected RBCs in neutrophils (Figure 1M). Images were acquired using a CellaVision DI60 automated slide reader (Wright-Giemsa stain, 1000× magnification). [The examination of peripheral thin blood smears showed ring-infected RBCs and schizonts (Figure 1N-O, Giemsa stain, 1000× magnification). Examination of peripheral thick blood smears showed the phagocytosis of brown hemozoin pigments by monocytes and the presence of abundant small trophozoites (Figure 1P, Giemsa stain, 1000× magnification).

to cytolysis and parasite death [1]. To avoid the toxicity of free heme, the parasites catalyze it into insoluble, inert hemozoin crystals which are stored in digestive vacuoles as non-toxic molecules. Malaria is a complex, infectious, hemolytic disease in which intracellular components released during hemolysis, including cytoplasm, free heme, hemozoin, and parasite components, are engulfed by circulating neutrophils and monocytes. The extent to which hemozoin accumulates within these immune phagocytic cells reflects both the parasite load and the severity of infection [2].

Erythrocytes infected with schizonts and mature trophozoites are frequently sequestered in the microvasculature. This is one of the major pathological features of malaria caused by *P. falciparum* and is also the reason why they are infrequently observed in peripheral blood samples [2]. The presence of schizonts and hemozoin in >5% of phagocytes (neutrophils or monocytes) is predictive of a highly sequestered parasite load and poor prognosis [1].

References

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