inflammatory response appears to be a common response in both malaria and bacterial infections, with increased body temperature seen in both conditions. However, while studies in children are lacking, we and other investigators have noted a difference in oxygen consumption in adults with malaria, compared to those with sepsis [1, 2]. Using the same methods described in the article on pediatric malaria, we found that Indonesian adults with malaria had increased oxygen consumption, compared with both controls and individuals with sepsis [2]. Previously, Day et al, using a thermodilution method, also observed that Vietnamese adults with severe malaria admitted to the intensive care unit had increased oxygen consumption, compared with patients with nonmalarial sepsis [1]. So while an inflammatory response is common to both, the effect on oxygen metabolism is different in both malaria and sepsis. Increased nitric oxide with resulting mitochondrial dysfunction has been described in adults with sepsis [3]. Decreased nitric oxide bioavailability in severe malaria may result in increased mitochondrial oxygen consumption seen in pediatric and adults [2, 4], but there is also impaired endothelial function in sepsis [5].

The mechanism of increased lactate proposed by Eisenhut is plausible but does not fully account for our paradoxical observation that oxygen consumption is increased in the setting of decreased or impaired oxygen delivery due to microvascular obstruction. The hypothesis of cerebral vasospasm due to nitric oxide deficiency is also one that is consistent with our previous results of nitric oxide quenching due to the increase in cell-free hemoglobin due to hemolysis [6] and will need to be confirmed in future studies.

Notes

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