Authors' reply

Sir,

We thank M. D. Al-Mendalawi.[1] for their interest in our case report[2] and the insightful comments. We agree that association of malaria and hemolytic uremic syndrome-thrombotic thrombocytopenia purpura (HUS-TTP) may not be causal and only a few cases have been described hitherto. However, ADAMTS13 activity and antigen levels have been reported to be reduced in patients with falciparum as well as vivax malaria.[3] This evidence of endothelial injury in malarial infections is

intriguing and needs to be explored. Furthermore, given the vivax malaria increasingly being reported as a cause of severe malaria,[4] it will be interesting to study what proportion of them have HUS-TTP.

Thrombotic microangiopathy is a histologic description that is characteristic of several diverse disorders, such as malignant hypertension and scleroderma, as well as TTP and HUS.[5,6] When the patients with microangiopathic hemolytic anemia and thrombocytopenia are initially evaluated, the comprehensive term TTP-HUS can probably be the best one to describe the clinical entity seen in our patient.

We agree that a complete evaluation including assessment of ADAMTS13 activity and genetic and autoimmune tests to identify a complement-related defect is required, which could not be done in our case. In absence of such a detailed evaluation, there remains a possibility of presence of underlying complement disorder where HUS was triggered by malarial infection. Infections, including diarrhea, are known as a trigger to develop HUS in patients having complement dysregulation.[7,8]

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