Hemolytic uremic syndrome associated with *Plasmodium vivax* malaria successfully treated with plasma exchange

Sir,

The two recently published case reports,[1] have thrown the light on the association of *Plasmodium vivax* malaria with hemolytic uremic syndrome (HUS). This unusual association is novel and indicates potential virulence of the parasite. However, such association is debatable at best. The diagnosis of HUS in the report of Keskar *et al*.[2] was based on the presence of high serum lactate dehydrogenase, elevated reticulocyte count and presence of schistocytes on peripheral blood smear. Furthermore, the kidney biopsy showed features of thrombotic microangiopathy (TMA). I presume that the aforementioned constellation is actually pertained to a group of pathological conditions termed TMAs rather than merely HUS. It is well-known that TMAs might be associated with many infections, bone marrow transplantation, pregnancy, systemic vacuities and certain drugs. They include several phenotypes, most prominently are thrombotic thrombocytopenic purpura (TTP), Shiga toxin–producing *Escherichia coli* HUS and atypical hemolytic uremic syndrome (AHUS). The diagnostic criteria of TTP are based on the clinical features of neurologic and renal dysfunction along with anemia and thrombocytopenia, low a disintegrin and metalloproteinase with a thrombospondin type 1 motif, member 13 (ADAMTS13) activity and the presence of ultra-large von Willebrand factor. In contrast, AHUS might appear as a consequence of mutations related to the alternative pathway for the compliment system. It can be distinguished from TTP on the basis of an ADAMTS13 enzyme activity measurement.[3,4] It is essential that HUS, AHUS and TTP be differentiated quickly, as they require markedly divergent treatments. Plasmapheresis in HUS is not efficient. Alternatively, plasma therapy and in some cases dialysis are used. The prompt treatment for most cases of AHUS and TTP is with plasma exchange initially and monoclonal therapy (rituximab in TTP and eculizumab in AHUS) as the mainstay of therapy.[5] I presume that Keskar *et al*.,[2] didn't consider the appropriate diagnostic tools, probably due to technical difficulties and limited financial resources, to exactly determine which TMA variant was associated with *P. vivax* malaria in their studied patient and hence, to implement the appropriate therapy.

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