

Letter to the Editor

Spontaneous chronic subdural hematoma following *Plasmodium vivax* malaria: A rare association

Dear Editor,

Chronic subdural hematoma (traumatic or spontaneous) is one of the commonly encountered neurosurgical emergencies. However, development of the same following malaria is an uncommon event. Spontaneous subdural hematoma following *Plasmodium falciparum* malaria has been reported in literature¹⁻². But, to date, no case of *P. vivax* associated with chronic subdural hematoma has been reported. We herein report a 45-yr old male patient who developed spontaneous chronic subdural hematoma following *P. vivax* malaria.

A 45-yr old male presented to us with complaints of gradual reduction in level of consciousness, headache and weakness of left upper and lower limbs over 20 days duration. He had history of fever with chills and rigors for which he got treated in a tertiary care centre in northeastern part of India, endemic for malaria, approximately one month back. During his hospital stay he developed severe headache which gradually worsened and was associated with excessive drowsiness and weakness of left upper and lower limbs. On investigation he was found to be positive for *P. vivax* by OptiMAL test, [a rapid, 20-min malaria detection test utilizing a dipstick coated with monoclonal antibodies against the intracellular metabolic enzyme parasite lactate dehydrogenase (pLDH), Diamed, Cressier, Switzerland]. He was treated with artemether (dosages not known) for about 10 days and gradually improved to become afebrile. However, he developed headache and gradually progressive weakness. About 20 days following discharge he was brought to our emergency department in altered sensorium and glasgow coma scale (GCS) score of E2M5V1 (8/15) with recurrence of fever. Pupils were unequal and not reacting to light and fundi showed bilateral papilledema. His reflexes were brisk with bilaterally upgoing plantars. There were no signs of meningeal irritation. NCCT scan of brain showed bilateral chronic subdural hematoma (left > right) with mass effect and midline shift of 5 mm towards the right side. His coagulation profile was normal [platelet count: 2.6 lakhs/mm³, bleeding time: 3 min and clotting time: 5 min, prothrombin time: 12 sec (control: 13 sec), and activated partial thromboplastin time: 34 sec (con-

trol: 32 sec)]. He was managed by emergency burr hole placement in bilateral frontal and parietal region with evacuation of altered subdural blood, irrigation with ringer lactate solution and placement of subdural drainage tubes, which were connected to closed drainage system and were removed after 72 h. He improved in his sensorium in the immediate postoperative period to a GCS of 15/15. In view of fever, the fluid was subjected to culture and sensitivity (aerobic— no growth after five days incubation).

Further, the patient was investigated for causes of spontaneous subdural hematoma including repeating his prothrombin time, activated partial thrombin time, and DIC profile (thrombin time: 10 sec; normal: 12–14 sec, fibrinogen level: 716 mg/dl; normal: 200–400 mg/dl, FDP— not detectable, Ivy's bleeding time: 4 min; normal: 2–7 min, clot solubility test: normal, D-dimer: 0.5 µg/ml; normal: <0.5 µg/ml). Liver function tests, collagen vascular studies [CRP: 1.2 mg/l; IgM RF: 10 IU/ml; ANA: 0.5; Acl Ab IgG: 04 GPL units; Acl Ab IgM: 05 MPL units; anti MPO: 0.1 IU/ml] and immunological studies Weil-Felix and Scrub (*O. tsutsugamushi*) typhus titers were negative; ANCA by immunofluorescence was negative; anti-myeloperoxidase antibodies and anti-proteinase-3 antibodies were negative] were normal. Peripheral smear was negative for malarial parasite for three consecutive days. The patient was afebrile from postoperative Day 1 and was discharged on Day 7. There was no recurrence till six months follow-up.

Acute SDH without traumatic brain injury is a rare pathological entity and accounts for fewer than 5% of SDH³. There are various reports of spontaneous SDH in healthy young adults who had risk factors like hypertension, vascular malformations, neoplasia such as hematological malignancies causing thrombocytopenia, solid tumour dural metastases, infection, hypervitaminosis, coagulopathy and alcoholism⁴. Spontaneous SDH has also reported in patients with sudden increase in intravenous pressure during coughing, defecation, trumpet blowing, and heavy weight lifting. It is also reported in patients following systemic hypotension causing intracranial hypotension resulting spontaneous SDH⁵. All these risk factors were absent in our patient.

In our case the patient had neurological symptoms which

started following a febrile episode due to malaria. There are reports of subdural hemorrhage following *P. falciparum* malaria². There are reports of *P. vivax* malaria causing cerebral malaria and other neurological deficits like bilateral facial nerve palsy⁶⁻⁷. *Plasmodium vivax* is seen with several complications in its severe form of malaria, either sequestration related, such as cerebral malaria, renal dysfunction, hepatic dysfunction, circulatory collapse, hemoglobinuria, abnormal bleeding and ARDS, or non-sequestration related, such as anemia and thrombocytopenia⁸⁻⁹. The cause of the bleeding remains unclear in the absence of definitive laboratory evidence and literary back up.

However, this may be the index case wherein an association (causal or non-causal) has been demonstrated for *P. vivax* malaria and chronic subdural hematoma.

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CORRIGENDUM

JVBD Vol. 50 No. 4 (December 2013) Page No. 322, text line number 10, reference number 4 shall be read as 5 and in line number 11, the reference number 5 shall be read as 6.