Concurrent meningitis and vivax malaria

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ABSTRACT

Malaria is an endemic infectious disease in India. It is often associated with other infective conditions but concomitant infection of malaria and meningitis are uncommon. We present a case of meningitis with vivax malaria infection in a 24-year-old lady. This case emphasizes the importance of high index of clinical suspicion to detect other infective conditions like meningitis when fever does not improve even after anti-malarial treatment in a patient of malaria before switching therapy suspecting drug resistance, which is quite common in this part of world.

Keywords: Anti-malarial, meningitis, vivax malaria

Introduction

Malaria is a protozoan disease transmitted by the bite of infected Anopheles mosquitoes. It is caused by parasite Plasmodium which is of mainly four species – Plasmodium falciparum, Plasmodium vivax, Plasmodium ovale and Plasmodium malariae. Vivax and falciparum malaria are responsible for most of the cases. Approximately up to 40% of the world’s population is at risk for malaria infection and 100 countries are in endemic areas for malaria transmission.[¹] It is responsible for nearly 216 million episodes and 66,5000 malaria deaths cases as reported in 2010.[²,³]

Malaria is sometimes associated with other infective conditions which if not diagnosed may be fatal.[⁴‑¹²] Here we present a case of vivax malaria associated with meningitis which was initially not diagnosed and later when the patient did not respond properly to antimalarial therapy was thought to have chloroquine-resistant vivax malaria. Clinicians need to rule out co-existent conditions and need to look out actively for red flags of meningitis in all cases of malaria.

Case Report

A 24-year-old female admitted with complain of fever, headache, vomiting and neck pain for 1 week. Previously before admission she was diagnosed to have vivax malaria by both microscopic examination and antigenic testing in some local hospital and she had received a course of chloroquine (600 mg stat dose followed by 600 mg after 24 hours and 300 mg after next 24 hours) with intravenous ceftriaxone 2 g per day for 4 days. But her fever improved marginally and she was having a persistent headache, nausea, vomiting and neck pain. History, clinical examination and investigations (HIV testing was negative) during admission were not suggestive of any condition that might lead to immune-compromise in the patient. We suspected it to be a case of chloroquine-resistant vivax malaria and sent the blood sample for examination of malaria parasite. However, malaria parasite examination was found to be negative on both microscopic examination and antigenic testing. On thorough clinical examination she was found to have neck rigidity, a positive Kernig's sign and a positive Brudzinski's sign and mild splenomegaly. The patient was started on empirical antibiotic (intravenous ceftriaxone 4 g per day) as associated meningitis was suspected. Simultaneously cerebrospinal fluid (CSF) and other routine laboratory tests were sent for examination. Her hemoglobin was 11 g/dl, total leukocyte count was 10,190/mm³ (41% neutrophil, 50% lymphocyte, 8% monocyte and 1% eosinophil) and ESR (erythrocyte sedimentation rate) was 42 mm on first hour. Her CSF examination showed a cell count of 170/mm³ with 10% neutrophil and 90% lymphocytes. CSF protein was 121 mg/dl, sugar 53 mg/dl (with a random blood sugar of 90 mg/dl at the time of sending CSF sample) and adenosine deaminase (ADA) was 4.8 U/L. Gram stain, Ziehl Neelsen (ZN) stain, India ink

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preparation and culture of CSF revealed no organism. Her blood urea, creatinine, sodium, potassium and liver function test were within normal limit. She responded well to therapy and her symptoms gradually disappeared within 4-5 days. She was discharged after completing a course of ceftriaxone 4 g per day for 10 days.

Written informed consent has been taken from the patient.

Discussion

Vivax malaria is a common infection in India and is usually chloroquine sensitive but chloroquine-resistant vivax malaria is also increasing. Failure of subsidence of fever after chloroquine treatment usually raises suspicion of chloroquine resistance. However, malaria is sometimes associated with other infective conditions,[4-12] which may also lead to failure of subsidence of symptoms as occurred in our case. The presence of neck pain and positive meningeal signs raised the possibility of having associated meningitis in this patient which was confirmed by CSF examination. The CSF report showed lymphocytic pleocytosis, raised protein level and a marginally low sugar level which was corroborative with partially treated bacterial meningitis as patient was already on ceftriaxone before admission. However, the possibility of viral meningitis cannot be ruled out as we do not have such investigation facilities. It is important to note that in viral meningitis CSF sugar level remains normal which in this case was marginally low which increases the possibility of partially treated bacterial meningitis.

Association of meningitis with malaria had very few previous case reports[9-12] and in those reports meningitis was found to be associated with falciparum malaria. In a study falciparum malaria was found to be present in 16.7% of children (less than 15 years of age) having meningitis.[11] To the best of our knowledge, this is the first reported case of an association of vivax malaria with meningitis published from South Asian Region. There is only another such reported case from Papua New Guinea to the best of our knowledge.[13] Failure to identify this condition may lead to misdiagnosis of drug-resistant malaria thereby altering the course of treatment and increased morbidity and mortality.

Conclusion

Possibility of associated infective conditions like meningitis should be kept in mind whenever a patient of malaria does not respond to antimalarial treatment along with the possibility of drug resistance. A detailed clinical examination and judicious use of laboratory investigations will help in proper diagnosis.

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