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COMPLICATED MIXED MALARIAL INFECTION PRESENTING AS SPLENIC INFARCTION IN INTRAVENOUS DRUG ABUSER: CASE REPORT

Haematology

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ABSTRACT

Introduction- Malaria is one of the most common tropical infection in India. It is a vector-born infection caused by Plasmodium species via Anopheles mosquito. Malarial infection presenting as splenic infarction is rare and only few cases are reported till now. We report a case of malaria due to mixed infection with Plasmodium falciparum and vivax, presented with splenic infarction. **Case Report-** A 27 year old male,chronic intravenous drug abuser presented with high grade intermittent fever with chills and rigor for 10 days, pain in left upper abdomen and headache for 7 days. Patient had pallor and tender splenomegaly. His serology for Human Immunodeficiency virus, Hepatitis B and Hepatitis C virus was positive. Peripheral smear showed ring forms of Plasmodium vivax and gametocytes of Plasmodium falciparum along with rapid diagnostic test positivity for both. Ultrasound abdomen was suggestive of hepatosplenomegaly and a hypoechoic area with ?inflammatory/ infarct changes in the upper pole of spleen. CECT abdomen showed hepatosplenomegaly with segmental infarct at upper pole of spleen with splenic rupture leading to small subcapsular/perisplenic collection. **Discussion-** Malaria was confirmed in this case by demonstration of ring forms of Plasmodium vivax and gametocytes of Plasmodium falciparum. Rapid diagnostic tests (RDT) for malaria are useful adjuncts for diagnosis in the field environment. Most common symptom in patients with splenic infarct is left hypochondriacal pain. CT scan of the abdomen is the most sensitive tool for diagnosis of splenic infarction. The treatment of splenic infarct is conservative with good outcome, as documented in our patient also. **Conclusion-** Splenic infarction, although an uncommon presentation of malaria, should be suspected in presence of left hypochondrium pain. Splenic infarction may be may be complicated by splenic rupture or splenic abscess which may require surgical intervention. The treatment of an uncomplicated splenic infarct is conservative with good outcome, as is documented in our patient.

KEYWORDS

INTRODUCTION

Malaria is one of the most common infectious diseases throughout India. It is a vector-born infection caused by *Plasmodium* species, which is transmitted through Anopheles mosquitoes, and it accounts for the majority of cases of illness and fever among travellers [1].It is caused by five different *Plasmodium* spp., *P. falciparum*, *P. vivax*, *P. ovale*, *P. malariae*, and the new species *P. knowlesi*. Molecular methods have demonstrated the existence of two distinct species of *P. ovale*: *P.ovale curtisi* and *P. ovale wallikeri*. Malaria can manifest with various life-threatening complications in which splenic involvement is frequently observed. This involvement may vary from simple asymptomatic splenic enlargement to serious complication such as hematoma, rupture, or infarction[1,2]. Splenic infarction is rare and only a few cases have been reported in the literature[1-5] A case of malaria due to mixed infection with Plasmodium falciparum and Plasmodium vivax who presented with splenic infarction and rupture is reported here. Most of the reported cases of splenic infarct in malaria have been due to infection with *P. falciparum* and is less common with *P. vivax*. We report this case for its rarity.

CASE PRESENTATION

A 27 yr old male,chronic intravenous drug abuser,chronic smoker and alcoholic presented with high grade intermittent fever with chills and rigor for 10 days and pain in left upper abdomen with headache for 7 days. At the time of presentation, temperature was 102 ºF, pulse 122 beats/min,and blood pressure(BP) of 100/80mmHg. On physical examination, patient appeared pale and having tender splenomegaly. He also had black coloured lesion over the tip of left index finger and left feet.

Lab investigation revealed hemoglobin of 4.8gm/dl, total leukocyte count of 7000/cumm, platelet count of 2.8lac/mm3, hematocrit of 13.7%, serum sodium of 126mmol/l and serum creatinine of 1.5mg/dl. Liver function test showed SGOT of 64 IU/L, SGPT of 36 IU/L, ALP of 91 IU/L,total bilirubin of 0.9 mg/dl(Direct bilirubin-0.3mg/dl), albumin of 2.6 g/dl, globulin of 4.7 g/dl. Viral markers were done in view of chronic IV drug abuse which came out to be reactive for HIV, hepatitis B & hepatitis C virus.

Patient was also evaluated for severe anemia(Hb-4.8gm/dl) which showed low serum vitamin B12 levels(144.9pg/ml), low Total Iron Binding Capacity(TIBC-177ug/dl) and transferrin levels(98.6mg/dl) for which vitamin B12 supplementation & two units of PRBCs were given during the course of illness.

Peripheral smear showed ring forms of Plasmodium vivax and gametocytes of Plasmodium falciparum. Rapid diagnostic test was also done which showed positivity for both for which antimalarial treatment was started.

Ultrasound whole abdomen was suggestive of hepatosplenomegaly. A hypoechoic area with cystic like changes of size 26mm x 20mm seen at upper pole ?inflammatory / infarct changes. Piperacillin/ Tazobactum(4.5gm) was started empirically in view of suspicion of splenic abscess. For confirmation, CT abdomen with contrast was done which showed mild hepatomegaly with homogeneous CT attenuation and splenomegaly(approx 15 cm) with segmental infarct measuring appx 7 x 5.7 x 3 cm (TR x AP x CC) at upper pole of spleen with splenic rupture leading to a small subcapsular/perisplenic collection of 16ml approximately. No evidence of any major arterial or venous occlusion seen. General Surgery opinion was taken for the same, advised to manage the patient conservatively.

MRI brain was done outside in view of headache and dizziness which was suspicious of cerebral venous thrombosis. To rule out, MR venography was performed in our institution which came out to be normal.

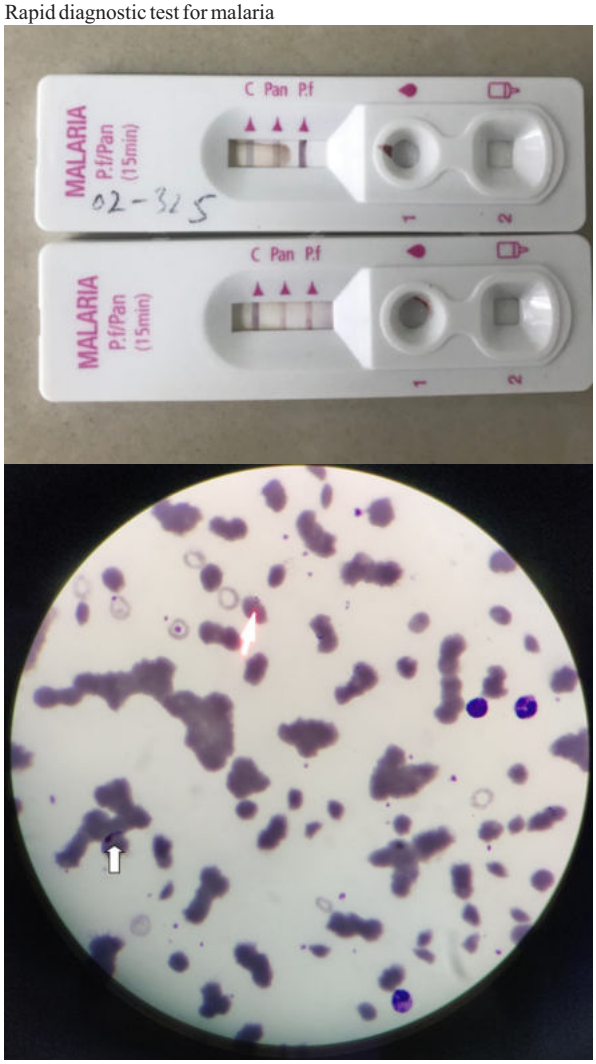
The patient was admitted in intensive care unit for monitoring and was treated with intravenous Artesunate 120mg at 0,12,24hrs then once daily which was followed by Tablet Artesunate/Lumefantrine (480/80mg) once daily for 3 days. Tab Doxycycline 100mg twice daily was also given for 7 days. Subsequently he was given Primaquine 15mg twice daily for 2 weeks after excluding G6 PD deficiency.

Psychiatrist opinion was taken in view of chronic drug abuse,advised for lorazepam 1mg tablet twice daily and to review the patient on outpatient basis after stabilization from the current illness.

Dermatologist consultation was also sought for hyperpigmented lesions in finger and feet which were secondary to chronic intravenous drug abuse for which no active intervention was advised.

In addition to the antimalarial treatment, he was also given supportive and symptomatic treatment with paracetamol, ondansetron, pantoprazole, probiotic & iv fluids to which he responded well and was discharged after recovery from the current illness with follow up advise.

Rapid diagnostic test for malaria



Peripheral smear showing ring forms of Plasmodium vivax(*red arrow*) and gametocytes of Plasmodium falciparum (*white arrow*)

DISCUSSION

Malaria is a vector-born parasitic infection caused by *Plasmodium* species, which is transmitted through Anopheles mosquitoes and it results in high mortality rates. There are four species responsible for malaria, with a newly discovered fifth species known as *P. knowlesi. P. falciparum*, and *P. vivax* has the widest distribution. Severe malaria is usually caused by *P. falciparum* . *P. falciparum* can cause both uncomplicated and complicated malaria. The symptoms of malaria vary, but it generally starts as lethargy, anorexia, nausea and vomiting with a headache, accompanied by recurrent intermittent fever and chills with sweating [6]. *Plasmodium* spp. are diagnosed based on microscopic features of a PBS. Malaria was confirmed in this case by demonstration of ring forms of *Plasmodium vivax* and gametocytes of *Plasmodium falciparum* Rapid diagnostic tests (RDT) for malaria are useful adjuncts for diagnosis in the field environment. Most common symptom in patients with splenic infarct is left hypochondriacal pain. CT scan of the abdomen is the most sensitive tool for diagnosis of splenic infarction. Splenic infarction can be focal or global and can result due to arterial or venous occlusion. Within the spleen, arterial supply is segmental and occlusion of a secondary branch results in a wedge-shaped infarct. Spontaneous recovery occurs in most of the patients with anti–malarial and other conservative treatment only.

Spontaneous splenic rupture is a known rare complication of *P. vivax*, which occurs during acute infection and during the primary attack. Chronically enlarged spleens are less vulnerable to rupture. Other splenic complications reported in malaria are splenic hemorrhage, splenic abscess and splenic torsion. Therefore, clinicians should consider splenic complications like splenic infarct, spontaneous splenic rupture and hemorrhage in the differential diagnosis of left upper quadrant pain in a patient with malaria. It is possible that many cases of splenic infarcts in malaria are missed because ultrasound and CT of the abdomen are not routinely done in patients of malaria. The treatment of splenic infarct is conservative with good outcome, as documented in our patient also. The clinician should be vigilant for the possible complications of splenic infarct with splenic abscess, rupture and hemorrhage which may require surgical intervention.

CONCLUSION

- Splenic infarction, although an uncommon presentation of malaria, should be suspected in presence of left hypochondrium pain.
- CT scan of the abdomen is the most sensitive tool for diagnosis of splenic infarction.
- The treatment of an uncomplicated splenic infarct is conservative with good outcome, as is documented in our patient.
- The clinician should be vigilant for the possible complications of splenic infarct with splenic abscess, rupture and hemorrhage, which may require surgical intervention.

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