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Algid Malaria (*P. falciparum*) in a Living Donor Liver Transplantation Recipient

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Abstract

Liver transplantation is the gold standard for end stage liver disease. Infections continue to be the commonest cause of post transplantation morbidities. Infections can be classified into bacterial, fungal, viral and parasitic. Parasitic infections can be further classified into protozoal and helminthic. Malaria is one of the protozoal infections. Very few cases of malaria after liver transplantation have been reported in the literature so far. Post transplantation malaria may be either acquired primarily or reactivation following immunosuppression or transmitted through organ or blood products. We describe a case of *P. falciparum* malaria acquired primarily post-transplant in a patient with ABO incompatible transplantation. Patient presented with acute febrile illness and shock along with mild graft dysfunction. After a diagnostic delay he was successfully treated with Artemisinin Containing Treatment regimen.

Keywords: *Plasmodium falciparum* malaria; Algid malaria; Living donor liver transplantation; ABO incompatible liver transplantation; Malaria endemic ethnicity

Introduction

Liver transplantation is the gold standard for end stage liver disease. Infections are the commonest complications after liver transplantation [1-4]. Transplant infections are either preexisting infections in the patient which flares up after immunosuppression, those transmitted by transplanted organs and transfused blood and blood products and those acquired post transplantation. These infections can be protozoal, bacterial, viral or fungal infections. Post-transplant patients are prone to infections lifelong although incidence is high in the perioperative period. Post-transplant malaria is one of the rare infective complications [5]. There are few case reports. We report one case of *Plasmodium falciparum* malaria in a ABO incompatible liver transplantation patient in the immediate post-transplant period managed successfully with Artemisinin containing treatment regimen [6].

Case Presentation

A 54-year-old gentleman (blood group B positive) from Nigeria was symptomatic for the past 3 years with abdominal distension and pedal oedema. He was diagnosed with ethanoic CLD (chronic liver disease) and managed medically with diuretics and hepato protective medications. 3 months ago he developed diuretic resistant ascites and encephalopathy. He had h/o multiple large volume paracenteses. No h/o jaundice, gastrointestinal bleed or hepato-renal syndrome. He was worked up for liver transplant and considered for ABO incompatible transplantation (Donor-Son, Blood group A-positive). Pre-transplant preparation was done with Rituximab + plasmapheresis regimen. He underwent Living donor liver transplantation (LDLT) with Modified right lobe graft. He tolerated the procedure well and was shifted to intensive care unit for elective ventilation. Acidosis corrected overnight and inotropic support was weaned off. He was extubated on post-operative day (POD)-1. Graft function improved satisfactorily. Peak bilirubin was 5.1 on POD-1 and peak INR was 3.4 on POD-2. Immunosuppression was started on POD-1 with standard regimen of tacrolimus, mycophenolate and steroids. On POD-4 he had Tacrolimus toxicity which was managed by withholding of tacrolimus till the levels reduced to normal levels, adequate hydration and liberal sedation to control the central nervous system symptoms. On POD-9 there was increase in antibody titre along with increase in enzymes. Suspecting antibody mediated rejection pulse therapy was given and he responded well. On POD-11 he developed episode of fever with hypotension and shock. He was appropriately resuscitated with inotropic support Empirical antibiotics were given and body fluid cultures were sent as per protocol. However, he continued to have spikes of fever along with deranged liver enzymes. In view of his malarial endemicity nativity wet smear for malaria was sent. The report was positive for *Plasmodium falciparum* malaria. He was appropriately managed with artesunate based regimen. He recovered well. CT liver angiography done on POD-13 (14/11/16) showed patent vascular anatomy of the graft. Drains were removed on POD-13 and POD-4. His Anti A titres were in satisfactory range throughout postoperative course. At 6 months post transplantation he is having good graft function and there is no recidivism.

Discussion

We are presenting this case for its rarity. Although few cases of post liver transplantation malaria have been reported in the literature [7], this is the first case of *Plasmodium falciparum* malaria in an ABOi- LDLT. Possible routes of malarial infection were Reactivation malaria, transmission through transplanted liver and transfused blood products and primarily acquired infections in areas of malarial endemicity [8]. In our case since the species isolated was *Plasmodium falciparum* reactivation was ruled out. Chronic malaria is common with *P. Vivax* and *P. Ovale*. Since the donor was negative for malarial infection (both acute and chronic) Trans-hepatic transfer was also ruled out. Being a Joint Commission International (JCI) accredited hospital all the blood and blood products in our hospital are irradiated and preserved. Hence the transfer through blood products was also unlikely. Since Delhi is endemic for malaria and our patient developed symptoms on the 11th POD (*P. falciparum* has incubation period of 7 to 14 days), primarily acquired malaria seems to be of high possibility. The High grade fever was also associated with elevation in liver enzymes. Malarial infection is known to produce graft dysfunction [9]. Other probabilities of graft dysfunction like rejection (in view of ABOi) and biliary complications (in view of 3 ducts in the graft) Bacterial infections (in view of recent pulse therapy) were also of equally high propabilities in this patient. But laboratory diagnosis combined with response to treatment proved beyond doubt that it is a case of post transplantation acquired *P. falciparum* malaria producing graft dysfunction.

Conclusion

In acute febrile illness, in endemic areas malarial infection can produce acute graft dysfunction.

This is the first case of *Plasmodium falciparum* infection post ABOi LDLT.

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