Case Report

A CASE OF PLASMODIUM FALCIPARUM MALARIA WITH COMPLICATIONS PRESENTED IN FAROOQ HOSPITAL, LAHORE.

Omair Farooq[1], Alia Waheed[2], Atiqa Arshad[3]

INTRODUCTION

Malaria is a serious parasitic disease that is affecting more than 200 million people in the world and accounting for about half a million deaths every year.[1,2] According to World Health Organization (WHO), around 229 million cases all over the world and 405,000 deaths were reported in 2019.[2,3]

Malarial infections are not common in the United States. About 1500–2000 malaria cases have been reported annually in the United States; almost all of them were among the people who travel recently and arriving from endemic countries.[4]

Cerebral malaria is a harmful form of malarial infection caused by Plasmodium falciparum and mostly affecting children, pregnant women, or adults with malaria-limited immunity.[5] Despite proper management and advancements in healthcare, mortality rates remain very high in cerebral malaria, ranging between 15% to 25%.[5,6] Poor outcomes are especially common among patients who present late, those who develop signs of cerebral edema and in the immunocompromised patients.[6]

We present a case of a young man who suffered a severe form of Cerebral Malaria and successfully recovered despite a poor initial prognosis.

CASE REPORT

A 43-years-old male patient presented to Farooq Hospital, West Wood, Lahore, with complaints of severe abdominal pain and loose stools, 10-15 in frequency, watery inconsistency, but having no blood in it. At the time of admission, his blood pressure was 80/40mmHg, and the patient was immediately resuscitated in the Emergency Department and was then shifted to ICU, where he was administered 3 liters of Normal Saline. However, despite this initial therapy, his blood pressure failed to show any signs of improvement. On initial examination of the patient, he was found dehydrated, deeply jaundiced, and unconscious with a GCS of 9/15. He was put on oxygen 6-7 Liters to maintain a saturation of 92%.

A provisional diagnosis of hypovolemic shock was made and it was decided to shift the regimen; the CVP line was passed and the patient was administered with an additional 1 liter of IV Normal Saline infusion with nor-epinephrine. This started to improve his blood pressure and finally stabilized at 100/60 mmHg.

His initial investigations revealed that he had a hemoglobin of only around 8g/dl, while bilirubin was 7mg/dl and Renal Function Tests (Creatinine) was raised around 2.1mg/dl; so initial suspicion and differentials also indicated that he might have Hemolytic Uremic Syndrome.

But, the peripheral smear of the patient showed Malarial Parasite, Ring Forms, Gametocytes and Trophozoites of Plasmodium Falciparum, so his history was taken again, which revealed that the patient had recently returned from South Africa and as labs also revealed thrombocytopenia, so there was a suspicion of Malarial/Viral Hemorrhagic Fever. However, Peripheral Smear revealed more than 50% Parasitemia along with Gametocytes of Plasmodium Falciparum; he had severe complicated falciparum malaria along with Black Water Fever, Hemolysis, and complication of Cerebral Malaria. His brain CT scan was clear, so, a possible diagnosis of Cerebral

[1]Senior Registrar Medicine, Farooq Hospital, Westwood Branch, Lahore.
[2]Associate Professor Pathology, AMDC, Lahore
[3]Assistant Professor Pathology, AMDC, Lahore.

Malaria with complications was made.

Immediately, the patient was started on injectable antimalarial therapy; due to potentially severe adverse effects of Quinine, he was started on intramuscular Artemether 160mg stat, followed by 80mg i/m daily, which is comparatively more effective and safer. In addition to that, the patient was started on broad-spectrum antibiotics including iv injection Meronem 500mg, TDS which was adjusted, and iv injection Flagyl 500mg, BD. While the patient was being managed, he developed Severe Metabolic Acidosis; his bilirubin kept on increasing and went up to 16mg/dl, and his Renal Function Test (Creatinine), went up to 2.8mg/ dl. He was transfused with 4 PCV's and 2 Whole Blood but his hemoglobin remained around 8g/dl. The patient was also a known case of Chronic Hepatitis B and the liver showed coarse architecture on ultrasound examination. The patient was also started on Tab Rifaximin 550mg, BID. On Day 3, the Patient developed constipation so he was given Duphalac Enema and oral Duphalac 30ml daily. Along with that, the patient was also started on Cap Usro, 250mg BD. On day 4, the patient showed signs of improvement, Chest x-ray showed infiltrates which were suspicious of Hospital-Acquired Pneumonia. HRCT was done 2 days later, which showed Subsegmental Consolidations and showed a picture of Viral Pneumonia. Covid Antibody Test was negative. Dengue serology and leptospiral antibodies were also negative. Blood Cultures revealed no growth in 5 days. The patient was well managed and on the 8th day of admission, he started showing signs of improvement. His hemoglobin improved to around 8g/dl, bilirubin dropped to 6mg/dl, and creatinine improved to1.5mg/dl. Parasitemia gradually decreased from 50% to <2% and then the patient was discharged.

This was the case of complicated falciparum malaria which has a mortality of almost 50% but he recovered well.

DISCUSSION

Malaria is a common parasitic infection infecting a large number of populations around the world. Our patient had a history of travel from the malaria-endemic area and came with the symptoms of high-grade fever, chills and presented in the emergency with shock, dehydration, impaired consciousness level. The lab reports confirmed the diagnosis of malaria. These findings are inconstant with the findings of a large number of studies conducted nationally and internationally.[7,8]

The patient had raised urea and creatinine and raised liver enzymes. Released Hemoglobin causes renal damage, which causes Acute Renal Failure. If no treatment is given to the patient, Anemia and broken Hemoglobin Products lead to Coma or Death in some cases.[9,10]

Patient presentation is mostly Acute Hepato-Nephritis within 24 to 48 hours after the administration of the antimalarial drug. Severe anemia even in the beginning and intravascular hemolysis due to severe malaria can lead to Oliguria, Dark Colored Urine, Abdominal Pain, Jaundice, Hepatic Splenomegaly, Vomiting, and Renal Failure.[10] The life-threatening situation seen initially has been significantly reduced now in the hospitals.[11]

Presently, morbidity data reported by most of the authors is between 23 to 26%.[11,12] Acute kidney damage pathogenesis and other organ dysfunction in plasmodium falciparum malaria understood properly.[12] Contributing factors may be volume depletion, hemolysis, disseminated intravascular coagulation, and sepsis.[12,13] Thrombotic microangiopathy has been seen in malaria.[13] Falciparum malaria-causing endothelial dysfunction may have a role in organ failure and not frequently seen in the course of vivax malaria.[13,14]

The time to coma recovery, which is about 7 days, is much better with Artemether than Quinine and that was observed in our case of P. falciparum malaria patient with recent traveling to the malaria-endemic area. It is therefore pointed out that the treating physicians should be aware of the chances of plasmodium falciparum infections in the patients who have travel history or have been in contact with travelers who returned from the malaria-endemic area recently (luggage,

airport, local transmission). Physician and pathologist communication is needed for the early diagnosis and effective management of such cases to prevent mortality.

AUTHOR'S CONTRIBUTION

OF: Conception of idea
AW: Drafting article
AA: Data analysis, editing

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