Case Report

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The most lethal human protozoan parasite is plasmodium falciparum: severe malaria-associated acute renal failure – a case report

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Introduction and importance: Malaria continues to be a significant global public health problem, particularly in endemic nations. The most common cause of acute renal failure is a *Plasmodium falciparum* infection.

Case presentation: A 28-year-old male was brought into the emergency room with significant complaints of fatigue, chills, fever, and a lack of appetite. The patient had no prior history of malaria. He was not given any antimalarial medication as prophylaxis while traveling to his workplace. As a result of laboratory investigations to identify malarial parasites in peripheral blood using thin and thick smears, malaria parasites were found in the patient's blood. At the border of the colitis, the liver was palpable. Both the chest radiograph and abdominal ultrasonography were clear. His level of consciousness assessment indicated a Glasgow coma scale reading of 10 out of 15. He received 1000 ml of normal saline solution with 40% glucose solution as part of his supportive care. He received intravenous artesunate 60 mg (2.4 mg/kg) when he was admitted to an ICU, and then every 12 and 24 h for the next 3 days (a total of three doses, 540 mg).

Clinical discussion: A typical symptom of severe malaria is acute kidney injury, which also carries its own risk of death. In regions with active transmission, *Plasmodium falciparum* is recognized as a significant contributor to acute renal damage.

Conclusion: The mechanism proposed for kidney injury by severe malaria is hemodynamic dysfunction, followed by inflammation and immunological dysregulation in the patient in this study. He had reduced serum sodium levels within the red blood cells, which led to calcium influx into the cell, altering the red blood cell's deformability.

Keywords: acute renal failure, antimalarial medication, artesunate, case report, plasmodium falciparum

Introduction

Malaria is an Italian word composed of 'mala' and 'aria', derived from mal (bad) and aria (air), which means bad air. It is a disease brought on by a protozoan parasite of the genus *Plasmodium*, namely *P. falciparum, P. vivax, P. malariae, and P. ovale*[1]. Travelers from northern countries returning from malaria-endemic areas are still at high risk of contracting *Plasmodium falciparum* malaria[2]. In Ethiopia, where 68% of the land mass provides conducive conditions for malaria transmission and 60% of the population is at risk of contracting the disease, malaria is a serious public health issue. Due to its appearance at the height of agricultural activity, malaria in Ethiopia currently also poses a serious threat to the health and socioeconomic growth of the nation[3]. *Plasmodium falciparum* is often linked to acute renal failure, but *Plasmodium malariae* has been linked to chronic progressive renal failure[4]. *Plasmodium falciparum* infection increases the risk of developing severe malaria, which can cause clinical consequences such as coma, renal failure, respiratory distress, or even death[5]. If the infection is not detected and treated in a timely manner, travelers returning from endemic areas run the risk of developing severe malaria[6]. According to the WHO, artesunate is the first-line therapy for both adults and children with severe malaria in endemic countries[4]. In this case report, a middle-aged patient's acute renal failure due to severe malaria is discussed, along with how severe malaria causes acute renal failure. This case report was submitted in accordance with the surgical case report (SCARE) criteria[7].

Case presentation

A 28-year-old black male was brought into the emergency room with significant complaints of fatigue, chills, fever, and a lack of appetite. The patient was hospitalized after experiencing joint discomfort, a dry cough, and epigastric burning for 3 days prior to admission. The patient complained of deteriorating symptoms, such as fatigue and an epigastric burning sensation, on the day of admission. He had some sense of direction, was awake, and could speak well. The patient had no history of chronic diseases such as

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chronic renal failure, diabetes, hypertension, or heart failure. He did not know his family's history of any medical problems. One year ago, the patient visited a malarious area for work. The patient had no prior history of malaria. He was not given any antimalarial medication while traveling to his workplace. His vital signs revealed a 38.1°C body temperature, a blood pressure of 121/77 mmHg, and a heart rate of 101 beats per minute. While inhaling room air, the patient's oxygen saturation was 96%.

His laboratory investigations revealed a hemoglobin level of 13.7 g/dl, a hematocrit of 38.4%, blood urea nitrogen of 43 mg/dl, fasting blood glucose of 117 mg/dl, an aspartate aminotransferase level of 47 units/l, an alanine aminotransferase level of 40 units/l, an erythrocyte sedimentation rate of 13 mm/h, an estimated glomerular filtration rate of 92 ml/min, a white blood cell count of 4850/ml, a platelet count of 91 400/mm^3, a serum creatinine level of 2.7 mg/dl, neutrophils of 87%, lymphocytes of 7%, and monocytes of 1%. Other serum electrolytes, including serum liver enzymes, except serum potassium levels of 5.9 mmol/l and sodium levels of 129 mmol/l, were found to be within the normal range. As a result of laboratory investigations to identify malarial parasites in peripheral blood using thin and thick smears, malaria parasites were found in the patient's blood. P. falciparum was detected in blood smear samples under a microscope (185 parasites/μl). Cerebrospinal fluid testing and urine analyses both came up negative. The amount of urine sediment was typical. Hemolytic-uremic syndrome (HUS) peripheral blood smear with no schistosomes, rare RBC fragments, and very few platelets indicative of thrombotic thrombocytopenic purpura (TTP) Disseminated intravascular coagulation (DIC) laboratory results show normal hemoglobin and a decline in platelets. He was sent to an ICU after being diagnosed with severe malaria after a peripheral thin blood film revealed P. falciparum trophozoites.

Cardiac and pulmonary sounds were normal. Examination of the head, eyes, ears, nose, and throat revealed a swollen face with pink conjunctiva. Cardiovascular testing confirmed that S1 and S2 were audible. At the border of the Costa, the liver was palpable. Both the chest radiograph and abdominal ultrasonography were clear. His level of consciousness assessment indicated a Glasgow coma scale reading of 10 out of 15. His ECG revealed sinus tachycardia with an ST-segment of 0.07 s and a heart rate of 101 beats per minute. He had a big, round, nontender belly that was dull but not organomegalic.

He received 1000 ml of normal saline solution with 40% glucose solution as supportive care for his condition. When he was admitted to an ICU and at intervals of 12 and 24 h for the next 3 days, he received intravenous artesunate 60 mg (2.4 mg/kg). The patient was shifted to oral dosages of 20 mg artemether and 120 mg lumefantrine, four tablets twice a day for 3 days following the final dose of artesunate (a total of three doses, 540 mg). After 4 days in the hospital, he made a partial recovery and was eventually released after clinical and laboratory tests revealed a noticeable improvement. He gave advice, saying he would visit a nearby clinic in a month.

Discussion

Plasmodium falciparum is the parasite that causes severe malaria, which presents as severe anemia, renal failure, acute respiratory failure, hypoglycemia, shock, and/or involvement of the central nervous system[8]. Indicators of *Plasmodium falciparum* malaria presentation symptoms and death patterns include geographic distribution, the level of parasite transmission, and host immunity to the parasite[9]. In this study, the patient traveled to a region with the greatest frequency of malaria since he had a job there, where *Plasmodium falciparum* is a frequent infection that can have serious health effects.

A female Anopheles mosquito that has been infected spreads the parasite. The disease remains the leading cause of morbidity and mortality in many poor tropical mains the leading cause of morbidity and mortality in many poor tropical countries, where it is primarily caused by *Plasmodium falciparum*[10]. The study participant was not given antimalarial prophylaxis when he was traveling to his workplace. He had been bitten by *Plasmodium falciparum*-infected mosquitoes, placing him at high risk for developing severe malaria and its consequence, acute kidney damage. In this study, the patient contracted *Plasmodium falciparum* in three stages. The first stage is infecting the liver cells, then the blood cells, and finally, producing gametes that might be transmitted by mosquitoes.

Malaria has a variety of clinical manifestations, and the development of these manifestations is influenced by host, parasite, and social and geographic variables. Asymptomatic infections, acute febrile illnesses, severe malaria, and fatal cases are all on the clinical spectrum[11]. The patient in this study had a wide range of malarial signs and symptoms, such as joint pain, nausea, vomiting of ingested material, chills, flank pain, fever, tiredness, and nausea.

In regions with active transmission, *Plasmodium falciparum* is noted as a significant contributor to acute renal damage[12]. The mechanism proposed for kidney injury by severe malaria is hemodynamic dysfunction, followed by inflammation and immunological dysregulation in the patient in this study. He had reduced serum sodium levels within the red blood cells, which led to calcium influx into the cell, altering the red blood cell's deformability. Acute tubular necrosis is what leads to AKI in cases of severe falciparum malaria. Other potential causes of acute renal injury caused by severe malaria include volume depletion, immune-mediated glomerular injury, and renal microcirculation blockage caused by parasite erythrocyte sequestration[3].

Recent studies estimated a high prevalence of AKI in patients with malaria of 20–40% in adults and up to 59% in children[13], according to the Kidney Disease Improving Global Outcomes (KDIGO) criteria. When renal replacement therapy is not started in a timely manner, death from AKI can reach up to 75% in areas where malaria is prevalent. AKI is at risk of developing in the majority of malaria patients who have volume depletion, hypoalbuminemia, male sex, prior AKI, concurrent bacterial sepsis, blackwater fever, or comorbidities, including diabetes mellitus.

Four phases make up AKI. Phase 1 (onset phase): kidney damage occurs. Significant blood loss, burns, fluid loss, and diabetes insipidus are frequent triggers. It can happen for hours or days. Phase 2 (oliguric (anuric)): decreased urine production due to renal tubule injury. Blood urea nitrogen and serum creatinine levels are rising. There are acidosis, electrolyte abnormalities, and fluid overload. It happens for at least 8 to 14 days. Phase 3 (diuretic phase): The kidneys attempt to recover, increasing urine production, although tubule scarring and damage take place. It happens when the cause of AKI is fixed. The excretion of more

water and the osmotic effects of high blood urea nitrogen may result in electrolyte deficiency. It happens between 7 and 14 days. Phase 4 (recovery phase): Renal function improves and tubular edema goes away during this phase of recovery. It happens for a few months to a whole year[14].

Through microscopy, the parasite can be detected inside erythrocytes in thin or thick blood films, allowing for a diagnosis. The examination of a drop of the patient's blood that has been spread out as a 'blood smear' on a microscope slide can reveal any malaria parasites present. Thrombotic thrombocytopenic purpura, hemolytic-uremic syndrome, and disseminated intravascular coagulation are the differential diagnoses for acute kidney injury in severe malaria[15].

Treatment for malaria-associated kidney disease includes the proper antimalarial medications in addition to other necessary supportive treatments for AKI (hydroelectrolytic disturbance adjustments, fluid replacement, and dialysis). Hemodialysis is a more effective form of renal replacement therapy to consider when treating AKI[16]. Dialysis is necessary in 46–76% of patients, and complete restoration of renal function is said to take place in about 64% of P. falciparum and P. vivax malaria-related AKI cases[17]. As the first-line treatment for severe and complex malaria, parenteral artesunate is recommended[18]. When artesunate is contraindicated or when the only reason for parenteral medication is intolerance to oral therapy, parenteral quinine is the alternative treatment[19]. In this study, the patient received 60 mg (2.4 mg/kg) of artesunate intravenously at the time of admission to an ICU and then every 12 and 24 h for 3 days. The patient was shifted to oral dosages of 20 mg artemether and 120 mg lumefantrine, four tablets twice a day for 3 days following the final dose of artesunate (a total of three doses of 540 mg).

Conclusion

Malaria becomes severe when catastrophic organ failures, abnormalities in the patient's blood, or abnormalities in their metabolism combine with infectious conditions. The most common cause of AKI and the most severe form of malaria is falciparum. Adults with severe malaria frequently experience acute renal damage, which can impact up to 40% of patients. A typical symptom of severe malaria is acute kidney damage, which also carries its own risk of mortality. The drug of choice for treating severe malaria caused by *Plasmodium falciparum* is parenteral artesunate.

Ethical approval

This case report did not require review by ethics committee.

Consent

Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written informed consent is available for review by the Editor-in-Chief of this journal on request.

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Author contribution

G.B.: contributes to the preparation of the proposal, participated in preparing the first draft of the manuscript, and edits of the manuscript. The author checked and confirmed the final version of the manuscript.

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Data availability statement

All data that support the findings of this study are included in this article and its online supplementary material files. Further inquiries can be directed to the corresponding author.

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