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Acute Kidney Injury in a Young Patient with Severe Malaria: A Case Report

Şiddetli Sıtması Olan Genç Bir Hastada Akut Böbrek Hasarı: Olgu Sunumu

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Abstract

Acute kidney injury is a complication of severe malaria that is relatively uncommon. It has clinical importance associated with substantial morbidity and mortality in malaria-endemic regions. We report a young patient who developed acute renal injury as a result of severe malaria. In this case, malaria diagnosis was confirmed using the malaria rapid screening test and microscopic inspection of blood smears. Fever, nausea and vomiting, loss of appetite and jaundice were the clinical presentations. Treatment with artemether injection, volume expansion and antipyretics was given. He recovered well without needing hemodialysis. This is not always the case, and the prognosis is dependent on the time of presentation, the diagnosis, and the treatment received.

Keywords: Malaria, Acute kidney injury, Artemether, Somalia.

Özet

Akut böbrek hasarı, ciddi sıtmanın nispeten nadir görülen bir komplikasyonudur. Sıtmanın endemik olduğu bölgelerde morbidite ve mortalite ile ilişkili klinik önemi vardır. Şiddetli sıtma nedeniyle akut böbrek hasarı gelişen genç bir hasta olgusunu sunuyoruz. Bu olguda, sıtma tanısı sıtma hızlı tarama testi ve kan yaymalarının mikroskobik incelemesi ile doğrulandı. Klinik prezentasyonlar ateş, bulantı ve kusma, iştahsızlık ve sarılıktı. Artemeter enjeksiyon ile tedavi edilen hastaya volüm desteği ve antipiretik verildi. Hasta hemodiyaliz gereksinimi olmaksızın iyileşti. Olgularda durum her zaman böyle değildir ve prognoz başvuru zamanına, tanıya ve alınan tedaviye bağlıdır.

Anahtar Kelimeler: Sıtma, Akut böbrek hasarı, Artemeter, Somali.

Introduction

The bites of infected female *Anopheles* mosquitoes spreads malaria, which is caused by *Plasmodium* parasites [1]. Human malaria is caused by six parasitic species, two of which– *Plasmodium falciparum* and *Plasmodium vivax*– are the most dangerous species [2]. The malaria parasite *P. falciparum* is the deadliest and most common on the African continent as well as Somalia [1,3]. Malaria is preventable and treatable, and the world's goal is to reduce morbidity and mortality while chasing the long-term goal of eradicating malaria [3,4]. *P. falciparum* infection frequently results in life-

threatening complications such as cerebral malaria, severe anemia, acidosis, jaundice, acute kidney injury, acute respiratory distress syndrome, pulmonary edema, and disseminated intravascular coagulation [5,6]. An estimated 600,000 people die from malaria in worldwide annually, with the vast majority of these deaths occurring in Sub-Saharan African countries [3]. In this report, we present a case of severe malaria associated with acute kidney injury.

Case

A 28-year-old male patient who was born and raised in Somalia, a malaria-endemic country, complained of a high fever accompanied by chills and myalgia as well as vomiting, nausea, loss of appetite, and jaundice. On admission, the patient had a flu-like illness (fever, headache, and arthralgia) for 4 days. He gives no history of any chronic disease. Clinical examination on admission, the patient was oriented, febrile, and jaundice. He did not have any rashes, edema, or palpable lymph nodes. The patient had a blood pressure of 100/60 mmHg, tachycardia, tachypnea (30 breaths/min), and a fever of 39.5°C. Malaria was detected through a rapid diagnostic test (CareStart Malaria Pf/PAN, AccessBio, USA) and microscopic examination of blood smears, both of which were positive for *P. falciparum*. Laboratory investigations showed the following results: white blood cells, 11.5 × 10^3 mm^3; hemoglobin, 12.2 g/dL; mean corpuscular volume, 79 fL; platelets 46 × 10^3 mm3; aspartate amino-transferase, 133 IU/L; alanine aminotransferase, 60 IU/L; total bilirubin, 13.78 mg/dL; direct bilirubin, 10.72 mg/dL; albumin 2.4 g/L and random blood glucose, 81mg/dL; sodium 124 mEq/L; potassium 4.22mEq/L. Brucella, Hepatitis B and C were also negative. Abdomen ultrasound showed no organomegaly. Patient had a urine output, and chest x-ray showed no signs of pulmonary edema. Based on these findings, the patient was diagnosed as having severe malaria with acute kidney injury.

We admitted the patient to the ward, volume expansion and antipyretics is given. Artemether injection 80 mg in the first 24 hours (2 doses), followed by 40 mg per day is started. Despite his renal function deteriorated during early admission, we continued to give enough intravenous fluids and our patient didn't show any indication for hemodialysis and finally our patient improved without needing dialysis (see Table 1 for follow up laboratory reports).

Table 1: Laboratory investigation during admission and discharge

	at admission	at discharge	reference range
Urea	257 mg/dL	74 mg/dL	(10-45)
Creatinine	6.57 mg/dL	1.8 mg/dL	(0.5-1.35)
AST	133 U/L	45 U/L	(0-35)
ALT	60 U/L	39 U/L	(0-45)
Total Bilirubin	13.78 mg/dL	1.83 mg/dL	(0.30-1.10)
Direct Bilirubin	10.72 mg/dL	1.66 mg/dL	(0.01-0.4)
CRP	219 mg/L	39 mg/L	(0-10)
Sodium	124 mEq/L	142 mEq/L	(135-150)
Potassium	4.22 mEq/dL	5.12 mEq/L	(3.5-5.5)
WBC	11.5 × 10^3	9.56 × 10^3	(4-10)
Hemoglobin	12.5 g/dL	10 g/dL	(13-18)
Platelets	46 × 10^3	230 × 10^3	(100-430)

Abbreviations: AST; ALT; CRP; WBC

Discussion

This observation is a result of severe malaria, specifically *P. falciparum*, occurring in locations with high transmission. Malaria-related acute kidney injury occurs at a rate of 1–4% worldwide and may reach 60% in nonimmune adults from nonendemic regions who travel to malaria-endemic regions [7]. The exact cause of acute kidney injury in malaria is still an unknown to scientists. Blockage of the renal microcirculation as a result of infected erythrocyte sequestration, immune-mediated glomerular damage, and

volume depletion are some of the explanations proposed [8]. Clinical evidence (anuria) and biochemical characteristics have been used to establish the diagnosis of acute kidney injury (azotemia, hyponatremia, and hypokalemia) [9]. Malaria-induced kidney injury occurs via a variety of mechanisms. Anoxic-ischemic lesions may be detected in association with the attachment of *P. falciparum* trophozoite - and schizont - infected erythrocytes to the endothelium of renal capillaries in cases of high parasitemia [10]. Malarial acute kidney injury can develop as a solitary complication or as part of a multiorgan disorder [11]. In the majority of cases, malarial acute kidney injury shows clinically and pathologically as acute tubular necrosis and are associated with jaundice in around 75% of cases, as observed in our patient [12]. Jaundice is typically "biphasic," with high levels of conjugated and nonconjugated bilirubin caused by cholestasis and hemolysis, respectively [13].

It has been demonstrated elsewhere that initiating hemodialysis early in the care of a patient with malarial acute kidney injury improves the recovery of renal function [14]. But our patient didn't undergo hemodialysis and responded well with parenteral artemether injection and volume expansion by intravenous hydration.

Conclusion

Although acute kidney injury as a result of severe malaria is uncommon, it should be kept in mind specifically in Malaria endemic areas like Somalia. Early diagnosis and management results in good prognosis.

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