

CASE REPORT

Acute renal failure in a Caucasian traveler with severe malaria: a case report

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Introduction

Malaria is an Italian word composed of “mala” and “aria”, derived from malus (bad) and aeris (air). It is a disease caused by a protozoan parasite of the genus *Plasmodium*, namely, *P. falciparum*, *P. vivax*, *P. malariae*, and *P. ovale*, while *P. Knowles* commonly affects nonhuman primates [1]. The parasite is transmitted by an infected female Anopheles mosquito. The disease remains to be the major cause of morbidity and mortality in many tropical developing countries where it is mostly caused by *Plasmodium falciparum* [2]. It is estimated that the latter causes around 600,000 deaths annually and the vast majority of such deaths occur in sub-Saharan African countries [3]. The high mortality rates caused by *P. falciparum* are to a great extent attributed to the parasite’s ability to induce severe malaria associated with life-threatening complications such as cerebral malaria (CM), acute renal failure (ARF), severe anemia, acidosis, respiratory distress, jaundice, and acute respiratory distress syndrome (ARDS) [2].

Plasmodium falciparum malaria presenting symptoms and mortality pattern vary considerably according to geo-

Key Clinical Message

Acute renal failure (ARF) secondary to severe malaria is uncommon. We report a case of a patient visiting Africa for the first time presenting with malaria and ARF. There was complete recovery after hemodialysis. Early initiation of hemodialysis proves to be useful in restoration of renal function.

Keywords

Acute renal failure, falciparum malaria, hemodialysis, nonimmune.

graphical distribution, parasite’s transmission intensity, and host’s immunity to the parasite [3]. In areas with high and stable malaria transmission, for instance, severe malaria is common in children under 5 years of age and commonly presents as severe anemia, while adults with acquired immunity to the parasite do not usually suffer severe malaria. In areas with moderate malaria transmission intensity, however, severe malaria commonly presents as CM in young children. Likewise, in low unstable malaria transmission intensity, severe malaria occurs in all age groups and can manifest as, CM, renal failure, severe jaundice, and/or pulmonary edema [3, 4]. However, ARF has been reported to be one of the most common complications of falciparum malaria in nonimmune adults [2, 4, 5]. Malarial Acute Renal Failure (MARF) can occur as an isolated complication or as a component of multiorgan involvement. Indeed, an association of ARF and CM has been reported and found to cause relatively higher mortality rates than CM alone [2]. MARF should be suspected when urine output falls to less than 400 mL/24 h or 20 mL/h despite adequate rehydration and the diagnosis is confirmed when serum creatinine exceeds

3 mg/dl (260 μ mol/L) [2]. The availability and provision of renal replacement therapy (RRT) as well as appropriate antimalarial chemotherapy has been shown to reduce MARF associated mortalities and enhance the restoration of renal function [4].

Case Report

We are reporting a case of a 55-year-old male Caucasian referred from Morogoro Regional Referral Hospital to the University of Dodoma Haemodialysis Unit, with a 3-days history of acute onset of high-grade fever associated with episodes of nonprojectile vomiting with the vomitus being mostly comprised of recently eaten food. The fever was also associated with chills, myalgia, and loss of consciousness, with episodes of generalized tonic-clonic seizures. Few hours following the fever onset, the patient developed shortness of breath with no cough. On the second day, while in the ward, he developed anuria (decrease in urine output), with 50 mL collected over 24 h that was soon followed by hiccups. The patient was visiting Morogoro, Tanzania for the first time and he had never been to any malaria endemic area before. Likewise, he had never used any malaria chemoprophylaxis prior to his visit. He denied having chest pain, palpitations, diarrhea, bleeding from any site, and declared that he had not been exposed to sick contacts. At the Morogoro Regional Referral Hospital, he was given two doses of 600 mg Quinine in 600 mL of 5% dextrose infusion. He was referred to the University of Dodoma Haemodialysis unit due to his nonimproving renal functioning (anuria). His past medical history was scrutinized and found to be rather uneventful. He had smoked one pack of cigarette per day for 30 years and he drank alcohol occasionally. However, he denied illicit or any kind of drug use, and he had no known allergies. His family history indicated presence of diabetes mellitus and ischemic heart disease.

Physical examination revealed that he was obtunded (GCS = 9/15), jaundiced, dyspneic, pale. He had no rashes, no edema, and no palpable lymph nodes. Vitals: his blood pressure was 90/60 mm Hg with no postural hypotension, his heart rate was 102 beats per minute. The respiratory rate was 32 breaths/min, and his temperature was 37.8°C. He had an oxygen saturation of 88% while breathing in room air. Urine output was less than 50 mL/24 h and random blood glucose was 12.2 mmol/L (219.9 mg/dL). Pertinent findings on chest examination included fine crackles at the lung bases, with decreased vocal fremitus. Auscultation of the rest of the chest revealed no abnormalities. His cardiovascular examination showed normal first and second heart sounds, with no jugular venous distention, murmurs, rubs, or gallops. Examination of the head and neck was unremarkable.

Abdominal examination revealed normal bowel sounds without distension, tenderness to palpation, or organomegaly. Rectal examination showed normal rectal tone, heme-negative stool, and no masses. His extremities were warm and his peripheral pulses were palpable. The rest of our examination on the patient revealed no significant findings. Additionally, an electrocardiogram was performed and results were remarkable for sinus tachycardia. Furthermore, laboratory analyses including complete blood cell count (CBC) with differential, complete metabolic panel, finger-stick blood glucose, blood slide (BS) for malaria parasites (MPS), malaria rapid diagnostic test (MRDT), bilirubin levels, urine analysis, and lumbar puncture for Cerebral spinal fluid (CSF) were carried out. The CBC revealed a normal white blood cell (WBC) count without a left shift. He was found to be anemic, with a hemoglobin level of 11.9 g/dL, mean corpuscular volume of 87 fL, and an elevated erythrocyte sedimentation rate (ESR) of 35 mm/h. The initial renal workup revealed serum creatinine of 1.33 mg/dL (117.24 μ mol/L), high serum urea of 30.66 mmol/L, serum potassium of 5.1 mEq/L, and serum sodium of 140 mEq/L. Likewise, his levels of ASAT and ALAT were 64 and 53 IU/L, respectively. His total bilirubin was 46 μ mol/L, while direct bilirubin was 30 μ mol/L and random blood glucose was 8.8 mmol/L. Microscopic examination of BS for MPS showed 1200 trophozoites/200 WBC (4800 MPS per μ L) and MRDT was positive for *P.falciparum*. Regarding urine analysis, sedimentation revealed muddy brown granular casts, specific gravity of 1.010, and sodium content of 42 (mmol/d). Bacteriological analysis revealed that urine was negative for such organisms. CSF analysis revealed normal findings and abdominal ultrasound showed normal-sized kidneys with no features of chronic kidney disease. Finally, a chest x-ray was performed and confirmed pulmonary edema. On the basis of the foregoing findings, the case was diagnosed as complicated malaria with ARF, pulmonary edema, CM and anemia, and the patient was scheduled for emergency dialysis.

Double lumen catheter was inserted on the right femoral vein of the patient and hemodialysis was started and ran for 4 h on day one. The process was facilitated by pump pressure of 100 and no fluid removal from the body was allowed. The patient was also subjected to intramuscular (I/M) injection of Artemether 160 mg bolus then 80 mg OD \times 4/7, I/V Frusemide 80 mg BID \times 3/7, I/V normal saline alternating with ringers lactate 6 L/12 h, and PO Paracetamol 1 g TID \times 5/7. After three sessions of consecutive hemodialysis of 4 h per day, the patient was conscious, able to sit upright without support, and had urine output of 2400 mL/24 h. His serum creatinine, urea, and potassium were 0.43 mg/dL (37.8 μ mol/L), 4.72 mmol/L, and 3.5 mEq/L, respectively. He was

discharged from the hospital 3 days later in good health. He was closely followed up after 2 weeks and 1 month and found to have normal and stable renal function.

Discussion

MARF is almost always caused by *P.falciparum* infection although renal involvement has also been reported in *P. malariae*, and recently in *P. vivax* infections [2, 6, 7]. *P. malariae* has been associated with chronic progressive renal failure, while *P. falciparum* is usually associated with ARF as in our patient [7]. The Incidence of MARF worldwide is 1–4% and may be as high as 60% among nonimmune adults from nonendemic regions visiting endemic regions of the world [8] as it is the case in our patient.

In severe malaria, ARF has been shown to be an important cause of mortality [6]. In most cases, MARF manifests clinically and pathologically as acute tubular necrosis as is reported in this case. Approximately 75%, MARFs are associated with jaundice as observed in our patient [9, 10]. The jaundice is usually “biphasic” with both conjugated and nonconjugated bilirubin elevated due to cholestasis and hemolysis, respectively [11]. It has been shown elsewhere that early initiation of hemodialysis speeds recovery of renal function in the management of a patient with MARF, [10, 12]. Our patient was subjected to hemodialysis 3 days after the onset of symptoms. Hemodialysis has been shown to be more effective than peritoneal dialysis which is often readily available in Sub-Saharan Africa [12]. Several cycles of dialysis are recommended to counteract the hypercatabolic state. Hemodialysis is considered adequate when there is postdialysis decrease of blood urea nitrogen (BUN) and serum creatinine by 50% from baseline [12, 13]. Our patient underwent three consecutive cycles and demonstrated complete recovery of renal function and postdialysis BUN and serum creatinine more than 50% less than baseline values.

Conclusion

The foregoing report conclusively outlines the importance of rapid recognition, proper case assessment, and early initiation of repeated sessions of hemodialysis in addition to fluid replacement and appropriate antimalarial therapy for early recovery of renal function in MARF.

Consent

Written informed consent for publication of the clinical details was obtained from the patient. The UDOM ethics review board provided the approval to publish this case report. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

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Conflict of Interest

None declared.

References

1. Thanachartwet, V., V. Desakorn, D. Sahassananda, K. Y. K. Win, and T. Supaporn. 2013. Acute renal failure in patients with severe falciparum malaria: using the WHO 2006 and RIFLE Criteria. *Int. J. Nephrol.* 2013:1–6.
2. Manan, J. A., H. Ali, and M. Lal. 2006. Acute renal failure Associated with malaria. *Med. Coll. Abbottabad* 18:47–52.
3. Das, B. S. 2008. Renal failure in malaria. *J. Vector Borne Dis.* 45:83–97.
4. White, N. J. 2009. Malaria. Pp. 1204–1205 in G. C. Cook and A. I. Zumla, eds. *Manson's Tropical Diseases*, 22nd ed. Saunders Elsevier, London.
5. Abdallah, T. M., M. T. Abdeen, I. S. Ahmed, H. Z. Hamdan, M. Magzoub, and I. Adam. 2013. Severe *Plasmodium falciparum* and *Plasmodium vivax* malaria among adults at Kassala Hospital, eastern Sudan. *Malar. J.* 2013:148.
6. Mohanty, S., S. K. Mishra, S. S. Pati, J. Pattnaik, and B. S. Das. 2003. Complications and mortality patterns due to *Plasmodium falciparum* malaria in hospitalized adults and children, Rourkela, Orissa, India. *Trans. R. Soc. Trop. Med. Hyg.* 97:69–70.
7. Stone, W. J., J. E. Hanchett, and J. H. Kneppshield. 1972. Acute renal insufficiency due to falciparum malaria. Review of 42 cases. *Arch. Intern. Med.* 129:620–628.
8. Tangpukdee, N., S. B. O. Elshiekh, W. Phumratanaprapin, S. Krudsood, and P. Wilairatana. 2011. Factors associated with acute renal failure in falciparum malaria infected patients. *Southeast Asian J. Trop. Med. Public Health* 42:1305–1312.
9. Barsoum, R. S. 2000. Malarial acute renal failure. *J. Am. Soc. Nephrol.* 11:2147–2154.
10. Wilairatana, P., S. Looareesuwan, and P. Charoenlarp. 1994. Liver profile changes and complications in jaundiced patients with falciparum malaria. *Trop. Med. Parasitol.* 45:298–302.
11. Gowda, S., P. B. Desai, S. J. Shetty, V. S. Kagwad, and M. B. Ilakal. 2011. Malarial hepatitis and renal failure: a study of two cases. *IJPH* 8:1, 19–1 21.

12. Mishra, S. K., and B. S. Das. 2008. Malaria and acute kidney injury. *Semin. Nephrol.* 28:395–408.
13. Vannaphan, S., N. Walters, T. Saengnedasawang, N. Tangpukdee, P. Kham-In, M. Klubprasit, et al. 2010.

Factors associated with acute renal failure in severe falciparum malaria patients. *Southeast Asian J. Trop. Med. Public Health* 41:1042–1047.