Rupture of spleen in a mechanically ventilated patient with falciparum malaria admitted with pulmonary edema

Wanis H. Ibrahim, MRCP(UK), Ahmed Kamha, MRCP(UK), CABM, Wisam Ghadban, MRCP(UK), Abdulatif Alkhal, MD.

ABSTRACT

Rupture of the spleen in malaria may constitute a diagnostic challenge to many clinicians particularly in non-endemic areas where experience with malaria is limited. Our aim is to increase the awareness among clinicians from non-endemic areas of serious malarial complications. We present a young American military man who was admitted to Hamad General Hospital and had 2 serious malarial complications, namely, acute pulmonary edema and rupture of the spleen. He was unusual compared with what was published previously in 4 main points: 1. The rupture of spleen occurred while the patient on mechanical ventilation and under the effect of sedation, which constituted a diagnostic challenge. 2. The 2 complications occurred in a patient with a low parasitemia. 3. The causative species for splenic rupture is *Plasmodium falciparum*, and 4. The first sample of peripheral blood smear for malarial parasite was negative. We treated him successfully and discharged home in a good condition.

Saudi Med J 2006; Vol. 27 (1): 98-100

Malaria is becoming an expanding health problem, not only in endemic areas of the world, but also in non-endemic parts due to increasing numbers of international travelers and migrants and reduced efficacy of prophylactic agents due to increasing malarial resistance.[1,2] Plasmodium falciparum malaria remains a major public health problem in endemic areas with approximately 1.5-2.7 million deaths each year. Tropical African countries are estimated to contribute up to 90% of the total malaria incidence and the great majority of malarial deaths especially in children aged <5 years.[3,4] Malaria in non-endemic areas such as Europe and the United States, usually diagnosed in travelers and immigrants from endemic countries. Military personnel are particularly at considerable risk. Approximately 16,000 diagnoses are made each year in Europe.[3] Hyper-parasitemia (>5%) was a minor World Health Organization (WHO) definition criterion for severe malaria in 1990 and was changed to a major criterion in 2000 showing that malarial complications are usually associated with hyper-parasitemia; however, although this finding is frequent in complicated malaria, nonetheless it is not invariable.[3,5]

Case Report. A 21-year-old American marine with no history of chronic illnesses was referred from a ship hospital accompanied by his physician following endotracheal intubation due to severe progressive shortness of breath for 3 days. The history was obtained from his physician and started with fever and diarrhea for 8 days prior to admission, and progressive shortness of breath for 3 days. He received ciprofloxacin for 8 days and metronidazole

From the Intensive Care Unit (Ibrahim, Kamha), Department of Medicine (Ghadban) and Infectious Disease (Alkhal), Hamad General Hospital, Hamad Medical Corporation, *State of Qatar.*

Received on 31st May 2005. Accepted for publication in final form 17th September 2005.

Address correspondence and reprint request to: Dr. Wanis H. Ibrahim, Specialist Intensive Care Unit, Hamad General Hospital, PO Box 3050, Doha, *State of Qatar*. E-mail: wanisian@yahoo.com

for 3 days prior to arrival to our hospital with no improvement. Rather, his condition deteriorated rapidly and he developed progressive shortness of breath. There was no history of abdominal pain, chest pain or hemoptysis. He is married since 8 months and there was no history of extra-marital sexual relationship. He was in southern Iraq for few months and then recently moved to Djibouti. His smoking and alcohol history was unknown. Upon arrival to our intensive care unit, he was having endotracheal tube in situ, with left subclavian central line, under effect of sedation, febrile of 38-39.5˚C with profuse perspiration. His chest examination revealed bilateral fine end-inspiratory crackles, and his abdominal examination showed soft and lax abdomen with hepatosplenomegaly approximately 2-3 fingers below costal margins. His cardiovascular examination was unremarkable. There was no neck stiffness, skin rash, or joint swelling. His pupils were equal, and reacting and there was no gross motor abnormality. Laboratory examination found a hemoglobin of 11.9 g/dL, white blood cells $2.9x10^3$/UL, and platelet count $29.5x10^3$/UL. Neutrophils 56%, lymphocytes 25% and eosinophils 0.5%. Peripheral blood smear showed bi-cytopenia, leukopenia and thrombocytopenia. Erythrocyte sedimentation rate of 11 mm/lh and brucella melitensis antibody titre was negative. Blood glucose 6.9 mmol/L, blood urea nitrogen 11.3 mmol/L, serum creatinine 124 μmol/L, serum albumin 25 g/L, Calcium 2.2 mmol/L, total bilirubin 41 umol/L, serum sodium 138 mmol/L, Potassium 4.3 mmol/L, Chloride 107 mmol/L, HCO_3 20 mmol/L, creatinine kinase 134 U/L, troponin T <0.01, serum amylase 101 U/L, lactic acid 1.9 mmol/L and prothrombin time 21.4 seconds. International normalized ratio 1.8, activated partial thromboplastin time 56 seconds, D-dimer level was $\geq$ 8 ug/L. Fibrinogen level was 3.75 g/L. Chest radiograph showed bilateral, multiple widespread heterogeneous fluffy shadowing in both lung fields at hilar and perihilar areas, in keeping with adult respiratory distress syndrome or pulmonary edema (**Figure 1**). Two bottles of blood cultured were negative. Echocardiogram was normal with an ejection fraction 60%. A computed tomographic scan of the chest showed ill-defined soft tissue density areas in lung fields bilaterally more in the medial lung fields involving the middle and upper lobes, no mass lesion. The bilateral pleural effusion was noted. Ultrasound of the abdomen showed hepatosplenomegaly. The patient was connected to mechanical ventilator, and was commenced on intravenous third generation cephalosporin with a Macrolide. Legionella and Mycoplasma titre were requested along with HIV status which was normal. Three samples of thick and thin blood smear were sent for malarial parasites. The first sample was negative, However, the second and third samples were positive for *Plasmodium falciparum* with low parasitemia.

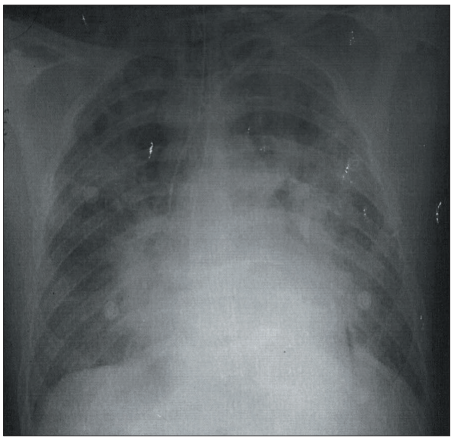

Figure 1 - Chest x-ray of the patient showing acute pulmonary edema.

On the fourth day post admission, the patient was irritable, fighting with the ventilator despite sedation, his oxygen saturation dropped down to 73% and required 100% oxygen to maintain adequate saturation. Physical examination revealed tense abdomen with marked tenderness all over. His hemoglobin dropped to 6.4 g/dl. Rectal examination was negative for melena or fresh blood. The possibility of splenic rupture was raised, and emergency ultrasonography followed by CT scan of the abdomen revealed splenomegaly with multiple areas of contrast enhancement within the spleen, and considerable amount of free fluid was seen around the spleen, liver and both paracolic gutters with CT densities suggestive of recent blood. Needle aspiration from peritoneal cavity confirmed the presence of fresh blood. Blood transfusion was started and urgent referral to the surgeon on call was made and surgical exploration of the abdomen was performed, which confirmed the diagnosis of splenic rupture. Splenectomy was then carried out.

The patient had uneventful postoperative period, his fever subsided and he was discharged home in good condition. Follow-up peripheral smear for malarial parasite prior to discharge was negative. Final diagnosis on the patient was *Plasmodium falciparum* malaria with low parasitemia complicated by non-cardiogenic pulmonary edema and splenic rupture.

Discussion. Splenomegaly occurs in 95-100% of individuals with acute malaria, however, clinically palpable spleen is seen only in 50-90% of cases.[1,6] On

gross examination, the spleen in malaria is usually hyperemic and congested with its capsule usually being thin and friable.[1] The majority of cases of malarial splenic rupture occurred in non-immune patients.[1,3] Why individuals who lack immunity against malaria are more likely to develop splenic rupture, is most likely due to the fact that these patients are prone to rapid progression of the disease and therefore, to severe splenic changes, and greater likelihood of splenic rupture.[1,7,8] The mechanism of splenic rupture appears to be related to the rapidly increasing size of the spleen that results in stretching of the capsule. Trauma that may or may not be noticed by the patient seems to have a major role in precipitating splenic rupture in patients with malaria.[7,9] This trauma might occur merely in form of increased intra-abdominal pressure such as in case of repeated vomiting and rigors that are commonly encountered in patients with malaria. A great caution must be taken when palpating a spleen of malarial patients, and it may be wise to advice medical students not to be harsh when examining malaria case. The malarial species, most often encountered in case of splenic rupture, is *Plasmodium vivax*, less often is *Plasmodium falciparum* and *Plasmodium malariae*.[1] The reason why splenic rupture is more encountered in *Plasmodium vivax* is not yet fully understood. We observed the splenic rupture in predominantly acute cases of malaria. The ruptured spleen in cases of malaria may constitute a diagnostic challenge to clinicians particularly in areas where malaria is rarely encountered. The high index of suspicion is crucial. This may be of a paramount importance when the patient is under effect of sedation or with impaired level of consciousness that may be encountered in cerebral malaria. Another factor that may add to the challenge is the fact that malaria itself may present with abdominal symptoms, which may mislead the clinicians in the incident that rupture occurs. Abdominal pain, fever, tachycardia, guarding, and tense abdomen with a drop in hemoglobin are commonly seen. Kehr's sign is a radiated pain felt on the left shoulder occurs as a result of irritation of the diaphragm by the ruptured spleen. A useful bedside diagnostic procedure is to insert a small needle into the peritoneal cavity (keep away from the spleen) and aspirate, if blood comes, it is highly suggestive of splenic rupture. Confirmation of the diagnosis may be achieved with radiological investigations like ultrasonography or CT scan. Resuscitation of such patients with blood and fluids is crucial, with continuous monitoring of hemodynamic status and hemoglobin. Splenectomy remained the treatment of choice for many decades, and many surgeons especially from non-endemic areas still consider it.[10] In recent years, there is growing support by many clinicians to the conservative and non-operative management.[11,12] This type of management may be of particular importance in patients living in endemic areas, due to the fear of severe future episodes of malaria in splenectomized patients. Nonetheless, conservative approach requires appropriate patient selection, availability of unlimited quantities of blood and high quality of clinical monitoring, while splenectomy is rapid and effective way of handling a life-threatening complication.[10]

References

1. Zingman BS, Viner BL. Splenic complications in malaria: a case report and review**.** ***Clinical Infectious Disease*** 1993; 16: 223-232.
2. Shultz MG. Malaria in migrants and travelers. ***Trans R Soc Trop Med Hyg*** 1989; 83 (Suppl): 31-34.
3. Bruneel F, Hocqueloux L, Alberti C, Wolff M, Chevret S, Bédos J. The clinical spectrum of severe imported falciparum malaria in the intensive care unit. Report of 188 cases in adults. ***Am J Resp Crit Med*** 2003; 167: 684-689.
4. Muentener P, Schlagenhauf P, Steffen R. Imported malaria (1985–95): trends and perspectives. ***Bull World Health Organ*** 1999; 77: 560-566.
5. Cosgriff TM. Pulmonary edema in falciparum malaria. Slaying the dragon of volume overload. ***Chest*** 1990; 98: 10-12.
6. Heineman HS. The clinical syndrome of malaria in the united states. ***Arch Intern Med*** 1972; 129: 607-616.
7. Covell G. Spontaneous rupture of the spleen. ***Trop Dis Bull*** 1955; 52: 705-723.
8. Gibney EJ. Surgical aspects of malaria. ***Br J Surg*** 1990; 77: 964-967.
9. Lubitz JM. Pathology of the ruptured spleen in acute vivax malaria. ***Blood*** 1949; 4: 1168-1176.
10. John BV, Ganesh A, Aggarawal S, Clement E. Persistent hypotension and splenic rupture in a patient with Plasmodium vivax and falciparum co-infection. ***J Postgrad Med*** 2004; 50: 80-81.
11. Davies GR, Venkatesan P. Successful conservative management of splenic rupture in vivax malaria. ***Trans R Soc Trop Med Hyg*** 2002; 96: 149-150.
12. Hamel CT, Blum J, Harder F, Kocher T. Non-operative treatment of splenic rupture in malaria tropica: Review of literature and case report. ***Acta Trop*** 2002; 82: 1-5.