

CASE REPORT

PLEURAL EFFUSION IN CHILDHOOD FALCIPARUM MALARIA

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Abstract. Pulmonary complication is a rare manifestation of childhood malaria and isolated pleural effusion without pulmonary edema has never been reported in children. We report here an 11-year-old boy who suffered from cerebral malaria and massive right pleural effusion. The patient was treated with intravenous artesunate, albumin, and other supportive treatments. He recovered completely after eight days. The clinical and laboratory courses suggested that the plasma leakage played a role in the pathogenesis of pleural effusion.

Malaria is one of the most important infectious diseases in tropical countries. With few exceptions, *Plasmodium falciparum* is the main species causing severe or complicated malaria. Acute pulmonary edema or acute respiratory distress syndrome is the main, frequently fatal pulmonary complication in adult patients. This condition is rare in children (White, 1996). Pleural effusion usually occurred in association with acute pulmonary edema (Punyagupta *et al*, 1974). Isolated pleural effusion without pulmonary edema has been reported in only few adults (Al-Ibrahim *et al*, 1975; Cayea *et al*, 1981) but has never been reported in children. We report here a case of childhood cerebral malaria with pleural effusion.

An 11-year-old previously healthy boy was referred to Bangkok Hospital for Tropical Diseases because of fever and convulsion. Two weeks before this illness the patient had traveled to Kanchanaburi Province which is an endemic area of malaria in Thailand. The illness began with gradually increasing fever, pallor, and weakness. He was admitted to a private hospital since the fourth day of illness. No specific etiology of the illness could be detected in the early days and the patient was treated symptomatically. Three days later, he developed three episodes of generalized tonic-clonic convulsion and severe anemia. *Plasmodium falciparum* was detected in his peripheral blood smear and the diagnosis of cerebral malaria

was made. Blood transfusion had been started before the patient was referred to our hospital.

On physical examination, he was unconscious with decorticate rigidity. His body temperature was 38°C, pulse rate was 92/minute, and respiration rate was 36/minute. There was decreased breath sound over his right lung. His liver was palpable 5 cm below right costal margin and spleen 3 cm below left costal margin. Edema was not present. Initial laboratory investigation showed hemoglobin of 10.7 g/dl, hematocrit 33%, leukocyte count 6,500/ μ l with 58% neutrophils, 37% lymphocytes, 5% monocytes, and a platelet count of 31,000/ μ l. His glucose-6-phosphate dehydrogenase status was normal. A random plasma glucose level was 110 mg/dl, blood urea nitrogen 15.5 mg/dl, creatinine 0.75 mg/dl, total bilirubin 2.7 mg/dl, direct bilirubin 1.0 mg/dl, aspartate dehydrogenase 103 U/l, alanine dehydrogenase 99 U/l, albumin 2.8 g/dl, and globulin 3.0 g/dl. Serum electrolyte was within normal limit. Peripheral blood smear revealed *Plasmodium falciparum* in density of 183,770/ μ l. Supine portable chest radiograph showed massive right pleural effusion (Fig 1).

First day treatment in Bangkok Hospital for Tropical Diseases consisted of intravenous artesunate, phenobarbital and diazepam to control convulsions, and endotracheal intubation with assisted ventilation because of inadequate spontaneous ventilation. Twenty g of albumin was intravenously infused followed by 40 mg of furosemide. After administration of furosemide, the pleural effusion decreased (Fig 2) and serum albumin rose from 2.8 to 3.7 g/dl. On the second day of admission,

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Fig 1—Supine chest radiograph on the first day of treatment.



Fig 2—Supine chest radiograph after administration of albumin and furosemide.



Fig 3—Supine chest radiograph on the third day of treatment.

however, he showed no remarkable improvement and serum albumin level decreased to 3.3 g/dl. On the third day of admission, he regained consciousness and was able to extubate on the fourth day of admission. Chest film showed minimal right pleural effusion on the third day (Fig 3) and showed no effusion on the fourth day of admission. The malaria trophozoites disappeared from peripheral blood 53 hours after initiation of treatment but the patient still had low-grade fever until the seventh day of admission. Repeated hematologic investigations on the fifth day of admission showed normal findings except mild anemia (hemoglobin 8.5 g/dl, hematocrit 26%). Blood chemistry evaluations on the same day showed decreased blood urea nitrogen (8.0 mg/dl) and creatinine (0.7 mg/dl), increased albumin (3.9 g/dl), and normal liver function. He was discharged after full recovery on the eighth day of admission.

Cerebral malaria and pulmonary edema are well known complications of falciparum malaria and many patients suffered from combination of these complications (Brooks *et al*, 1968; Punyagupta

et al, 1974; WHO, 1990). The exact causes of these complications are unknown but evidence suggests that sequestration, some immunological processes, disseminated intravascular coagulation, and increased vascular permeability play important roles in the pathogenesis (White, 1996; Punyagupta *et al*, 1974). Fluid overload may play an additional role in pulmonary edema. We do not know yet whether pleural effusion is the same but mild clinical entity as pulmonary edema or it is a separate clinical entity.

In this case, the patient had hypoalbuminemia and pleural effusion, which improved after administration of albumin. There was further decreasing of serum albumin level, which indicated further loss of albumin from blood circulation. Finally the albumin level returned to normal after clinical improvement from malaria. These findings indicated that there was plasma leakage during acute phase of malaria infection and subsequently caused pleural effusion. This event may be similar to plasma leakage in dengue hemorrhagic fever (Nimmannitya, 1987). Although we did not investigate for other concomitant infections that could cause pleural effusion, the clinical course suggested that falciparum malaria should be the sole infection in this patient. When malarial parasite disappeared from blood circulation, the cerebral and pulmonary manifestations disappeared more or less the same time and the patient recovered completely. Other causes of pleural effusion, *ie* intravenous fluid overload is unlikely since there was evidence of pre-renal insufficiency (BUN 15.5 mg/dl, Cr 0.75 mg/dl, BUN:Cr ratio 20) during the presence of pleural effusion.

The plasma leakage may also play some roles in pulmonary edema as the study by Punyagupta *et al* (1974) found that all nine malaria patients with pulmonary edema whose serum albumin was measured had hypoalbuminemia. Although some of these patients had liver function abnormality, acute liver dysfunction should not markedly affect

serum albumin level because albumin has half-life of about 20 days. We therefore suggest that plasma leakage, not only water and electrolyte leakage from increased vascular permeability, may play a role in pathogenesis of pulmonary manifestations of falciparum malaria.

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