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Malaria with Pulmonary Complications

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Introduction

Malaria is primarily transmitted by the bite of an infected female *Anopheles* mosquito to infect humans [1]. Diffuse interstitial edema, pulmonary edema, pleural effusion, and lobar consolidation are presented in severe falciparum malaria [2]. Sanklecha and colleagues reported three cases of childhood falciparum malaria in a family and revealed that two cases demonstrated bilaterally fluffy pulmonary infiltrates whereas the remaining case showed normal chest roentgenogram [3]. All reported patients with sickle cell anemia in their study demonstrated bilaterally pulmonary infiltrates [4]. Chest roentgenographic presentations are usually nonspecific, but they should be recognized in high endemic areas of malaria [4]. A patient with *Plasmodium vivax* malaria demonstrated diffuse bilateral alveolar opacities which indicated acute respiratory distress syndrome [5]. In India, three cases of ARDS with *Plasmodium vivax* malaria were also reported and demonstrated one case with bilateral parahilar infiltrates, one case with bilateral diffuse extensive opacities, and another case with bilateral basal ground glass opacities [6]. Pulmonary edema is an universal finding at autopsy [7]. The alveoli are filled parasite-red blood cells, non-parasited red blood cells, neutrophils and pigment-laden macrophages, and laminated periodic acid-schiff (PAS) positive membrane which finally destroys and incorporated the alveolar wall within it in many severe cases [7]. This is associated with abundant edematous fluid, pulmonary vasodilatation, and may have a marked inflammatory infiltrate [7]. Particularly in falciparum malaria, there is hyaline membrane formation in the alveoli that indicates leakage of proteinaceous fluid [7]. In the lungs of severe cases, the majority of blood vessels showed parasite-red blood cell sequestration in the septal capillaries and small blood vessels [8]. Mononuclear cell-pigment laden macrophage were seen admix with parasite-red blood cells in the microvessels of alveolar septa [8]. Platelet- activating- factor receptor activation was significant in the pathogenesis of pulmonary damage associated with *Plasmodium berghei* ANKA strain infection in a mice model, demonstrated in a recent study [9]. Approximately 60% of these infected mice had hypoxemia, dyspnea, pleural effusion, airway obstruction, pulmonary edema, and pulmonary hemorrhage [9]. There is little knowledge about the pathogenesis of malaria-associated acute lung injury and adult respiratory distress syndrome (ARDS) [10]. Increased endothelial permeability and inflammatory mediators may play a significant role, while parasite sequestration may take a minor role that supported by elevation of level of vascular endothelial growth factor found in mice model [10]. *Plasmodium falciparum* merozoite proteins could increase pulmonary endothelial permeability, while *Plasmodium falciparum* infected-red blood cells did not reveal the same properties indicating that the effects of the malaria parasites on the pulmonary endothelium are probably mediated the activity of Src-family kinases [10]. In infected mice lungs, increased water content was demonstrated, and contributed to the development of pulmonary edema [10]. DBA2 mice infected with *Plasmodium berghei* K173 showed proteins and inflammatory cells mainly CD4+ and CD8+ lymphocytes, monocytes and neutrophils accumulated in the lungs of infected mice [10]. Levels of cytokines and chemokines associated with ARDS were measured by Van den Steen and colleagues and revealed an expression of tumor-necrosis-factor-α, interferon-γ, CXCL10 and CXCL11, as well as neutrophills and monocyte chemo-attractant chemokines (CCL2, KC) in the lungs [10]. Subclinical impairment of lung function, such as impaired alveolar ventilation, reduced gas exchange, and increased pulmonary phagocytic activity may be found in uncomplicated malaria [10]. ARDS patients with *Plasmodium vivax, Plasmodium ovale*, and *Plasmodium knowlesi* were also reported despite it is the most common complication in *Plasmodium falciparum* malaria [10]. Two cases of *Plasmodium ovale* with ARDS were recently reported by Lau and colleagues [11]. *Plasmodium falciparum* causes the greatest severity and frequency of ARDS and could be partially attributed the resetting an sequestration of parasite-infected red blood cells in the pulmonary microcirculation [10]. The majority of the patients with ARDS have parasitemia [12] in up to 25% in adults, particularly in pregnant women that can up to 29% [13]. In children, ARDS may develop in up to 40% [13]. The association between the heavy parasitemia, white blood cell agglutinates and ARDS in patients with *Plasmodium vivax* malaria and could be due principally to dysregulation of cytokine production [10]. In patients infected with *Plasmodium knowlesi*, increased parasitemia in patients infected with *Plasmodium knowlesi* indicates parasite-specific effects that increase pulmonary capillary permeability, but could contribute to hypoxemia and metabolic acidosis [10]. Disappearance of the K76T mutation in PfCRT is related to chloroquine susceptibility [14], while a point mutation in the *Plasmodium falciparum* chloroqiune-resistance transporter (PfCRT) gene is responsible for chloroqiune-resistant falciparum malaria [15]. The World Health Organization (WHO) recommends oral treatment of piperaquine plus dihydroartemisinin as soon as the patient is able to take oral medication but not before a minimum of 24 hours of parenteral treatment [16]. The WHO recommended that intravenous artesunate can be administered preferentially over quinine for the treatment of severe malaria caused by any *Plasmodium* species in both children and adults [17], which also suggested by Taylor and colleagues [13]. In uncomplicated malaria caused by all *Plasmodium* species and chloroquine-resistant *Plasmodium vivax* in both children and adults, oral artemisinin-based combination therapies have also demonstrated equivalent (if not better) [17]. The best way to prevent malaria is insecticide-treated bed-nets in which insecticide is incorporated into the net fibers [18]. RTS,

S/ASO2, a vaccine has demonstrated promising results in endemic areas [18].

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