



AN INTERESTING CASE OF VIVAX INDUCED ARDS

General Medicine

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ABSTRACT

Though *Plasmodium vivax* is often considered to be innocuous, awareness of its potentially fatal side effects is growing. *Plasmodium vivax* malaria aggravated by ARDS is the subject of an intriguing case report. Prior to starting a Sulfadoxin Pyrimethamin combo treatment, patients were given artesunate. Rarely, *Plasmodium vivax* patients will present with ARDS or develop it while receiving treatment.

KEYWORDS

INTRODUCTION

India has a large area where malaria is prevalent, and the disease caused by the malaria parasite may manifest itself in a variety of ways. More than half of all cases of malaria in Asia are due to *Plasmodium vivax*(1). The most common parasite that causes severe malaria is *Plasmodium falciparum*, and it often manifests as organ involvement, such as kidney, lung, or CNS. Pulmonary consequences in *Plasmodium vivax* malaria are extremely uncommon and often manifest as a benign acute febrile disease(2). We report the case of a patient who had acute respiratory distress syndrome (ARDS) as a result of *P. vivax*, and who was successfully treated with artesunate, then a combination of sulfadoxin and pyrimethamine.

CASE REPORT

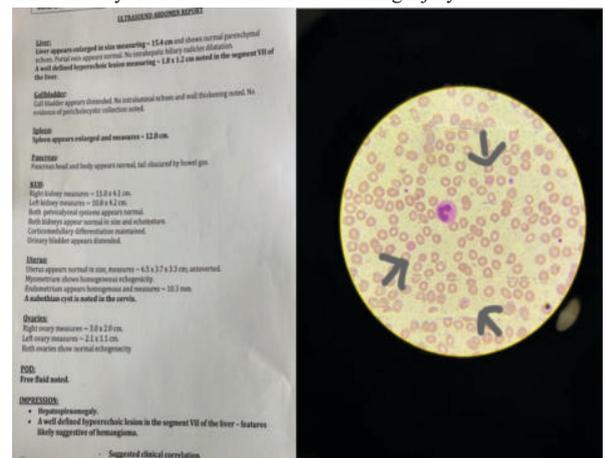
A 38-year-old woman who had been experiencing fever, myalgia, and coughing for seven days showed in. One day prior to admission, he also started having breathing problems. She had received chloroquine before being diagnosed with malaria and taken to a nearby hospital. Patient was sent to our hospital due to his deteriorating overall health. Her temperature, pulse, respiration rate, and blood pressure were all 38°C, 110/min, 50/min, and 90/70 mm Hg at the time of admission. She looked alert and well-focused. On inspection, the legs had purpura. On auscultation, there were bilateral crackles in the infraaxillary and infascapular areas. The liver was palpable and located 4 cm below the subcostal border. When inhaling room air, oxygen saturation was 78%. Analyses of the arterial blood gases revealed a hypoxemia PaO₂/FiO₂ ratio of 100.

By looking at peripheral film and using the histidine-rich protein 2 kit assay (best), falciparum infection was ruled out. Leptospirosis and dengue serology were negative. Blood and urine cultures came back negative. Bilateral nonhomogenous opacities were visible on the chest X-ray (Figure 1). The echocardiogram was normal with ejection fraction of 65% and no RWMA. The patient was transferred to the critical care unit and started on an artesunate and amoxicillin clavulanic acid combination. She kept receiving non-intrusive ventilation. But because of her ongoing hypoxia, she needed to be ventilated and intubated. Although the patient's oxygenation improved, the PO₂/FiO₂ ratio fluctuated. After receiving Sulfadoxin Pyrimethamin combo on the ninth day of hospitalisation, the patient's PO₂ significantly improved. Successful extubation and hospital release for the patient. Following release for four weeks, the patient was in good health.

DISCUSSION

The patient had histories of fever and shortness of breath, which is not how malaria typically manifests. The patient's malaria was discovered

outside hospital. Severe malaria symptoms in a *P. vivax*-infected patient are assumed to be caused by a mixed infection, however this isn't always the case. Acute respiratory distress syndrome (ARDS) as a consequence of vivax malaria has only been previously recorded in 22 instances, and pulmonary involvement is seldom observed 3. Uncertainty surrounds the pathophysiologic mechanism behind this condition, which may resemble ARDS seen in *P falciparum* malaria. It is not entirely clear how Plasmodia causes lung injury.



It was previously believed that *Plasmodium vivax* infection lacked this mechanism, despite new studies suggesting that *Plasmodium vivax*-infected red blood cells may also cytoadhere to endothelial cell ligand chondroitin sulphate A 4. However, this notion is also debatable since the majority of comparable earlier instances had minimal parasitemia, as in our case, and often the patient exhibits respiratory failure signs after beginning therapy when clinical improvement is occurring. When compared to ARDS brought on by *Plasmodium falciparum* 3, the reported mortality rate for malaria brought on by *Plasmodium vivax* is much lower. Lung damage in *P vivax* malaria may be explained by *P vivax* sequestration in the lungs and an inflammatory reaction after treatment. In previously documented cases, treatment approaches included the use of chloroquines, artesunate, primaquine, invasive ventilation, and noninvasive ventilation. Sulfadoxin Pyrimethamin is frequently thought to be ineffective against *Plasmodium vivax*, yet it quickly improved the clinical conditions of our patient. This was a brand-new finding about lung damage caused by *Plasmodium vivax*. Antifolates are efficient against *P vivax* malaria in South Asia, according to a randomised controlled study using an open-label design 5. Additional research is needed to better understand

the mechanism at play and the therapy of this malaria complication.

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