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Immunogenetics: Open Access

Short Communication

Note on Malaria Management in Immunogenetics

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DESCRIPTION

Malaria is an enormous public health problem with an estimated mortality of more than a million annually [1]. Global travel and migration have contributed to a steady occurrence of the disease in no endemic areas [2,3]. In 2006, the Center for Disease Control (CDC) received reports of 1564 cases of malaria among persons in the United States, an increase of 2.4% from 2005 all species of Plasmodium (P. falciparum, P. vivax, P malaria) present with an acute febrile illness. Most malaria-associated complications have been described with P. falciparum infection; recently, similar complications have also been reported with P. vivax. a primary attack in a nonimmune individual may be brief, severe, and may result in hemodynamic instability. In the no endemic countries, most patients with falciparum malaria present within 3 months of return from the endemic zones [4]. The pathophysiology of severe malaria has best been described with P. falciparum infection about cerebral malaria. Virulence of P. falciparum has been attributed to its propensity to produce heavy parasitemia and its ability to cytoadhere to the vascular endothelium, leading to sequestration of parasitized red blood cells (PRBCs) in the microvasculature of vital organs. Ring surface proteins 1 and 2, P. falciparum erythrocyte membrane protein 1(PfEMP 1) are parasitic proteins expressed on the surface of parasitized RBCs8 and promote adherence to vascular endothelium using several endothelial cell receptors: CD36, thrombospondin (TSP), and intercellular adhesion molecule 1 (ICAM-1)9 or chondroitin sulfate A in placenta.10 Parasitized RBCs also adhere to uninfected RBCs forming rosettes; a well-documented association exists between severe malaria and rosette formation.11 A role of complement receptor 1(CR1) in RBC rosette formation has been postulated as individuals with polymorphisms in the CR1 gene, who express low levels of CR1, show greatly reduced rosetting, and are protected against severe disease. The presence of parasites within the RBCs also makes them less deformable, the most common infections in patients with severe malaria are aspiration pneumonia and primary gram-negative bacteremia. It has been suggested that the infections early in the course of the disease are due to splanchnic ischemia and transmigration of enteric organisms and later may occur due to nosocomial infections. The term "algid malaria" is used for severe malaria complicated with hypovolemic shock and septicemia. In the endemic areas, Salmonella bacteremia has been associated specifically with P. falciparum infections. Aspiration pneumonia following seizures is an important cause of death in cerebral malaria. The other complications of a comatose patient, namely catheter-induced urinary tract infection and nosocomial respiratory infection may aggravate the clinical condition. Microscopy of stained blood smear remains the gold standard test for diagnosing malaria. A thick smear is more sensitive in detecting malarial parasites. Thin smear allows species identification and staging of parasite differentiation. Thin smear also allows quantification of the percentage of parasitized red cells, an important prognostic indicator, and marker of therapeutic response. The sensitivity of microscopy can be excellent to the tune of detection of malaria parasite density of 5 to 10 parasites/mL of blood in a thick smear (approximately 0.0001% parasitemia53; nearly all patients with clinical malaria, either partially immune ones from endemic areas or naive patients such as travelers, have parasitemia above this detection level 54-56). Microscopy is, however, operator-dependent, labor-intensive, time-consuming, and multiple meticulous smear examinations may be needed to detect malaria parasites and mixed plasmodial infection. Acquisition and retention of skill in microscopic diagnosis is difficult in a low prevalence country where malaria is seen infrequently in an individual center, with a potential to cause a delay in diagnosis and therapy and wrong identification of species. Rapid malaria diagnostic tests (MRDT) have their own appeal in hastening the diagnostic work-up in critically ill patients and the simplicity of their application.58 Current MRDTs, all use lateral flow immunochromatographic technology, using monoclonal antibodies, for the detection of malaria antigens either specific to a Plasmodium species or common across all human malaria. P falciparum-specific tests use either histidine-rich protein 2 (HRP-2) or P. falciparum lactate dehydrogenase; pan-plasmodium antigens used include Plasmodium lactate dehydrogenase (PLDH) and aldolase enzymes; P. vivax-specific tests have undergone only limited evaluation.

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Received: 07- Jan-2022, Manuscript No. IGOA-22-15513; **Editor assigned:** 10- Jan-2022, PreQC No. IGOA-22-15513 (PQ); **Reviewed:** 24- Jan-2022, QC No. IGOA-22-15513; **Revised:** 28- Jan-2022, Manuscript No. IGOA-22-15513 (R); **Published:** 10- Feb-2022, DOI: 10.35248/ IGOA.22.7.160.

Citation: Sreenivas T, El-Bahnasawy MM (2022) Note on Malaria Management in Immunogenetics. Immunogenet Open Access. 7:160

CONCLUSION

Severe malaria is associated with significant morbidity and mortality. The management includes admission to the intensive care unit, prompt administration of appropriate parenteral antimalarial agents, and early recognition and treatment of the complications. In children, the complications include metabolic acidosis (often caused by hypovolemia), hypoglycemia, hyperlactiacidemia, severe anemia, seizures, and raised intracranial pressure. In adults, renal failure and acute respiratory failure are more common causes of death. Clinicians should have a high index of suspicion, especially with travelers returning from endemic areas.

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