Perspective
Volume 11:4, 2022

Journal of Malaria Control and Elimination

ISSN: 2470-6965 Open Access

Identification of Lymphocyte in Lymphatic Vessels and Spleen Sections in Fatal Neonatal Malaria

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Introduction

Secondary lymphoid tissues are important in the human immune response to *Plasmodium falciparum* infection. Previous research has linked acute *falciparum* malaria to significant changes in the cellular immune system, including a decrease in the frequency and absolute number of circulating T cell subsets. Two proposed explanations for this observation are temporary relocation of T cells, possibly by infiltration into secondary lymphoid tissue, or permanent loss through apoptosis. The current study was designed to determine the phenotype of lymphocyte subsets that accumulate in the lymph node and spleen during the acute stages of *falciparum* malaria infection in Malawian children, as well as to test the hypothesis that lymphocytes are relocated to lymphoid tissues during acute infection.

Despite significant progress in reducing case numbers and mortality over the last decade, the global malaria burden remains high, with *Plasmodium falciparum* malaria expected to account for 241 million clinical cases and 627,000 deaths in 2020. *Falciparum* malaria can appear as an uncomplicated (UM) or severe infection. The latter includes cerebral malaria (CM), severe malarial anaemia (SMA), and other complications, with some overlap. CM has the worst outcome, contributing to the greatest number of deaths and a wide range of neurological sequelae in survivors.

Description

Although the pathogenesis of the various clinical types of severe malaria is unknown, they are caused by a combination of host and parasite factors, including high inflammation and sequestration of infected red blood cells (iRBCs) onto the vascular endothelium, followed by an inflammatory response, resulting in end-organ damage [1-3]. Even in high transmission areas and despite repeated parasite exposure, natural protective immunity against malaria takes years to develop. This immunity is stage-specific as well as parasite-specific, and it is influenced by age, genetics, pregnancy, nutritional status, and co-infections. Reduced exposure can cause this acquired immunity to wane quickly, potentially increasing the risk of developing more severe forms of the disease.

Vaccines have been shown to be the most reliable, cost-effective, and efficient method of controlling the burden and spread of many infectious diseases, but this has not been the case with malaria. RTS, S/AS01, the most promising malaria vaccine candidate to date, was approved for pilot implementation in three African countries in 2015, Malawi being one of them. The search for a robust and effective malaria vaccine continues, and a better understanding of naturally acquired immune responses to the various stages of the parasite, including transmissible stages, could be critical in informing effective vaccine designs.

During the course of a malaria infection, studies in murine models of malaria showed a depletion of T and B cells from the spleen's marginal zone, with a corresponding increase of the same cells in the red pulp. A postmortem study of adult Vietnamese patients who died from *P. falciparum* malaria confirmed this observation. The architecture of the spleen in this cohort was altered, with a distinct dissolution of the splenic marginal zones and a significant loss of B cells. T and B cells migrate to different segments of the lymph nodes, where they interact with antigen-carrying APCs and undergo clonal expansion, which is important in the host immune response to malaria [4,5].

Conclusion

Previously, our group and others demonstrated that acute *falciparum* malaria is associated with significant disruptions of the cellular immune system, as evidenced by lower frequencies and absolute numbers of T cells in the peripheral circulation. The two leading explanations for this observation are temporary relocation of T cells, possibly via infiltration into secondary lymphoid tissue, and permanent loss of these cells via apoptosis.

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Date of Submission: 07 July, 2022, Manuscript No. mcce-22-80950; **Editor assigned:** 09 July, 2022, Pre QC No. P-80950; **Reviewed:** 23 July, 2022, QC No. Q-80950; **Revised:** 28 July, 2022, Manuscript No. R-80950; **Published:** 02 August, 2022, DOI:10.37421/2470-6965.2022.11.188

How to cite this article: Dortet, Laurent. "Identification of Lymphocyte in Lymphatic Vessels and Spleen Sections in Fatal Neonatal Malaria." Malar Contr Elimination 11 (2022):188.