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Commentary

Cytokine Imbalance Associated with Co-Infection of Malaria

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DESCRIPTION

The outcome of infections with intracellular pathogens, such as viruses, bacteria, and protozoa, depends on the balance between inflammatory and regulatory cytokine responses. One such coinfection is malaria, which can be contracted together with an infection from a bacterial, viral, parasitic, or other malarial species. Products from the malaria parasite, such as DNA coupled to hemozoin and glycosylphosphatidylinositol anchors, activate toll-like receptors to cause the release of pro-inflammatory cytokines. Due to the co-circulation of several disease agents and the sensitivity of the host, these coinfections may develop. In contrast, malaria may raise the chance of contracting another infection. These coinfection's clinical characteristics could make diagnosis difficult. Treatment for concurrent disorders must be more involved. Any one of the four plasmodial species that are known to cause malaria is an infection. Other human malaria parasite species, the most significant of which is *Plasmodium vivax* species that causes malaria, always coexist with *Plasmodium falciparum*. *Plasmodium vivax* and *Plasmodium falciparum* co-infections are known to increase the risk of morbidity, intra-host competition between the two species makes sense. Mixed-species infections also lower parasite recurrence after antimalarial treatment, and a decreased risk of anaemia. In addition, compared to single-species infections with *Plasmodium vivax* or *Plasmodium falciparum*, mixed-species infections may be linked to milder symptoms. By escalating the inflammatory reactions that are characteristic of acute-phase malaria, bacterial co-infections can aggravate the consequences of malaria. By causing neutrophil dysfunction, human malaria can enhance the vulnerability to concurrent bacteremia. Among the pro-inflammatory cytokines, which are associated with severe malaria and death, are TNF-α, IFN-γ, IL-6, IL-8, IL-18 and MCP-1. However, anti-inflammatory cytokines like IL-10 and TGF-β are the regulatory cytokines. They dampen the effect of pro-inflammatory response.

Pathophysiology

Virus and bacterial contamination: The biology of the severe illness brought on by *Plasmodium vivax*, a human malaria parasite that is largely benign. The remarkable inflammatory imbalance seen in severe *vivax* malaria may be caused by unidentified bacterial or viral co-infections. Patients with *Plasmodium vivax* malaria create incredibly high quantities of IL-1beta in their mononuclear cells. Potentially inflammatory effects of these infections may be exacerbated by infectious comorbidities, such as endothelial dysfunction that results in respiratory discomfort and circulatory problems. Coinfection might also be caused due to the mycobacterial species like *Mycobacterium tuberculi* or *Mycobacterium leprae*, resulting to cause coinfection of either tuberculosis or leprosy along with malaria. Co-infections with *Plasmodium falciparum* which causes malaria is a detrimental effect of viral infection on malaria-related outcomes.

CONCLUSION

Constant vigilance makes it possible to quickly identify the cause of comorbidities like malaria, leading to early diagnosis. Concurrent infections can also make inflammatory stimuli of bacterial origin more sensitive, aggravating the overall severity of the illness. Variable levels of parasite virulence and co-infections with unrelated pathogens are key factors in this process. The potential impact of co-infections on the clinical outcome of infections is very common. Thus, to inhibit malaria parasite that arises along with mycobacterial species, medications with regular dose of malaria along with drugs that relief Tuberculosis (TB) or leprosy should also be strictly followed.

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