Background: The role of the circulating platelet has until in relatively recent times, been mainly considered in terms of cellular mediators of thrombo-haemorrhagic activities. It has most recently also been shown to play important role in modulating host immune response to infections such as malaria infection, both in the early and later phases of the infection. Data on the role that platelets play in early malaria infection is relatively scanty. This review highlights changes in platelet characteristics and function that have been reported in acute malaria infection.

Methods: Literature from Pubmed (MEDLINE), Google Scholar, Google search, textbooks and Cochrane Library were reviewed covering the period

Results: It is observed that Thrombocytopenia which had hitherto been considered as the hallmark of the complication of acute malaria infection, occurs in 40-80% of human acute malaria infection and in 100% of murine models. It results from platelet activation mechanism. The evidence in support of this view includes associated findings of elevated plasma concentrations of Beta-thromboglobulin (BTG) and Platelet Factor 4 (PF4) as well as enhanced production of Thromboxane A, (TXA,) and6-keto prostaglandin F l a (6-KPFla). There is also loss of total platelet sialic acid associated with reduction of platelet life span. A more recent finding of platelet killing of the parasite inside the infected red cell has revealed a hitherto little known potential which shows that early interaction between circulating platelets and the malaria parasite in the course of infection may result in reduction of parasitaemia thus mediating host survival to malaria infection. The mechanism(s) of platelet protective activity in early acute malaria is/are yet to be fully clarified in order to provide better understanding of the phenomenon. Clinically, it has also been reported that in acute malaria infection, the severity of clinical manifestations correlates closely with the parasite load.