



## MALARIA AND SALMONELLA INFECTION: RELATIONSHIP OR CASUALITY?

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### INTRODUCTION

In Sub-Saharan Africa, non-Typhoid Salmonella (NTS) bacteriemia is a common and often fatal complication of Plasmodium falciparum malaria. Thus WHO protocols recommend to treat children with severe malaria also with broad-spectrum antibiotics.

### CASE REPORT

We describe the case of B.M. a four year old boy, born in Italy, admitted to our Department for severe Plasmodium falciparum malaria after visiting relatives in Burkina Faso for the first time.

The child spent one month in Burkina Faso and travelled back to Italy two weeks before the admission to our Department. Parents told that the antimalaric prophylaxis with mefloquine was not properly performed; and that some doses were skipped.

One week after the return in Italy, the child's older brother was admitted to Infectious Diseases Department of our Hospital for P. falciparum malaria and treated with Artemether/lumefantrine.

B.M. was brought to the Emergency Department for a 24 hour history of high fever (BT >40°C), diarrhea and vomiting. The child was admitted with the suspect of Malaria infection. He presented in good general conditions, with no neuropathological signs, nor circulation impairment, with body temperature 40.3°C, pulse 139/min, ABP 102/56 mmHg, 100% SpO<sub>2</sub>. Abdominal examination revealed a palpable tip of the spleen, no hepatomegaly. Investigation revealed a haemoglobin of 10.9 g/L, total peripheral white blood cell count of 11,330/mm<sup>3</sup>, and platelet count of 207,000/mm<sup>3</sup>.

As blood smear examination revealed intermediate parasite density for P. falciparum, and malaria RDT was positive, intravenous quinine therapy (10 mg/Kg three times a day) was promptly administered with improvement of the conditions and decrease of fever after 24 hours.

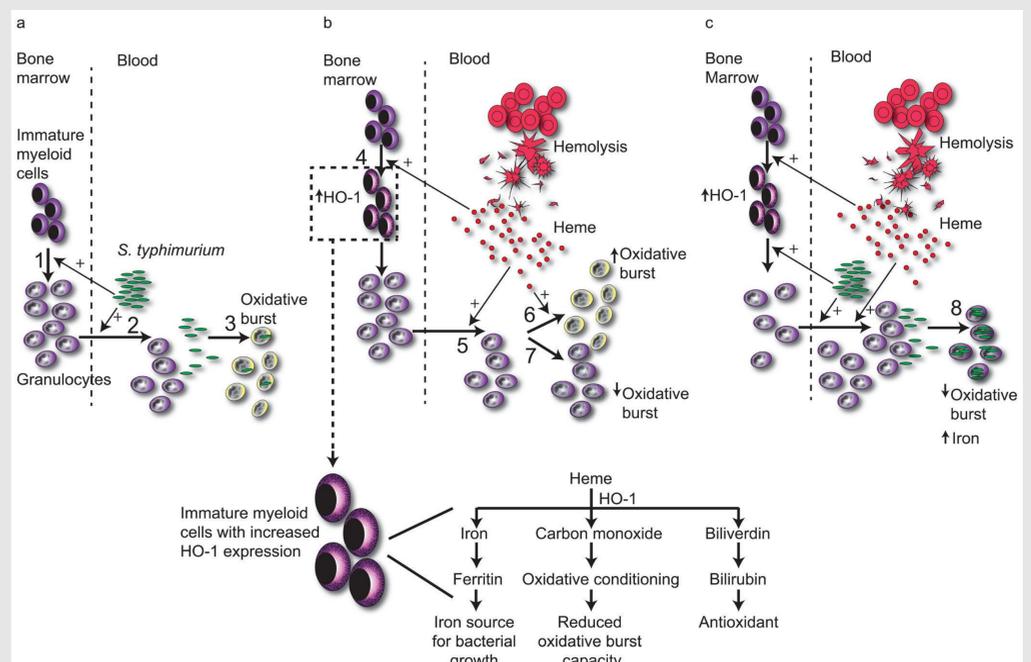
During the third day of hospitalization the temperature abruptly raised, and B.M. presented vomiting and worsening conditions. Laboratory testing showed increased CRP level (281 mg/L) and decreased total White Blood Cells count (6,200/mm<sup>3</sup>). Blood smear examination was negative for Plasmodia while blood culture resulted positive for Salmonella enteritidis; rectal swab grew no organism. The patient was treated with parenteral ceftriaxone (50 mg/kg twice daily) for 10 days and oral quinine for one week with a complete recovery.

### DISCUSSION

While co-infections of typhoid fever and malaria have quite commonly been reported in endemic areas (Oundo 2002), the underlying pathophysiological mechanism is still to be ascertained.

Malaria have been thought to predispose to Salmonella infections. (Keong et al., 2006).

Recent studies in mice (Cunnington et al., 2012) showed that the increased risk for developing NTS bacteriemia during malaria is caused by the hemolysis of red cells infected by Plasmodium. Intravascular hemolysis releases heme which triggers mobilization of functionally immature granulocytes from bone marrow, and induces heme oxygenase 1 (HO-1) in immature myeloid cells, reducing their oxidative burst capacity. This leads to the accumulation in peripheral blood of immature and functionally-impaired granulocytes, with reduced antimicrobial activity and consequent reduced resistance to NTS replication and dissemination. Moreover heme is degraded to biliverdin, carbon monoxide and iron, which accumulates supporting bacterial growth.



Cunnington et al., Malaria impairs resistance to Salmonella through heme- and heme oxygenase-dependent dysfunctional granulocyte mobilization, Nat Med 2012

### CONCLUSIONS

Concurrent malaria and salmonella infections are frequently described in endemic areas. This vulnerability to NTS in malaria infection is being assessed in literature. In Medicine coexisting pathologies are rarely explained by casuality.