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LETTERS TO THE EDITOR

Dear Sir:

We read with great interest the prospective study of 288 pediatric cases of *Plasmodium falciparum* malaria in a hypoendemic region in Senegal reported by Gerardin and others.[1] This study reported that children with severe malaria have a lower median platelet count, and that a platelet count less than 100,000/mm^3 was independently associated with a 13.3-fold increase in mortality. However, in a larger study, we were unable to find an association between thrombocytopenia and death.[2]

Our group recently analyzed the clinical and hematologic parameters of 1,369 children aged 3 months to 12 years admitted between January and December 2000 to the Kilifi District Hospital in Kilifi, Kenya, which is located on the Kenyan coast in a mainly rural malaria-endemic region with two seasonal peaks of transmission per year.[3] This hospital has a 36-bed general pediatric ward and a 5-bed high-dependency unit for approximately 5,000 children a year.

Using platelet counts of 111 asymptomatic, aparasitemic children in the community as a control group, we were able to define the normal platelet count reference range (mean ± 2 standard deviations) for our pediatric population as 150–700,000/mm^3. Overall, 56.7% of the children with malaria had thrombocytopenia (platelet count <150,000/mm^3) compared with children admitted with other diseases (16.4%) (χ^2 = 498, $P << 0.0001$, odds ratio = 6.66, 95% confidence interval = 5.57–7.96).

The median platelet count was inversely associated with age (χ^2 for trend = 28.2, $P << 0.0001$) (Figure 1) and parasite count (χ^2 for trend = 113.3, $P << 0.0001$) (Figure 2). However, it was not associated with abnormal bleeding as in the study of Gérardin and others,[1] and 1,016 (74.2%) of 1,369 children fulfilled the broad World Health Organization (2000) definition for severe malaria, which is comparable to the value of 74.7% in the study of Gérardin and others. In our study, the median platelet count was higher (139,000/mm^3 versus 115,000/mm^3; $P = 0.0006$) and the risk for thrombocytopenia was lower (539 of 981, 54.3% versus 242 of 388, 62.4%; $P = 0.008$) in children with severe malaria compared to those with mild, uncomplicated malaria. Indeed, of all the features of severe malaria, thrombocytopenia increased only with prostration (137 of 213 versus 639 of 1,156; $\chi^2 = 6.0$, $P = 0.014$). This association persisted when thrombocytopenia was defined as a platelet count < 100,000/mm^3. There was no

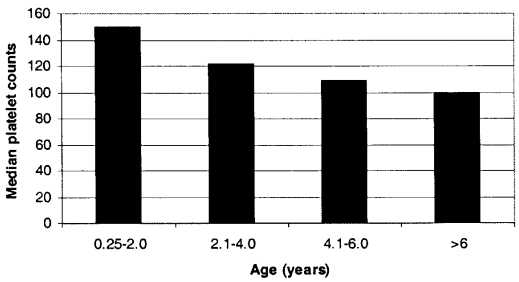

FIGURE 1. Median platelet counts versus age (χ^2 for trend = 113.3, $P << 0.0001$).

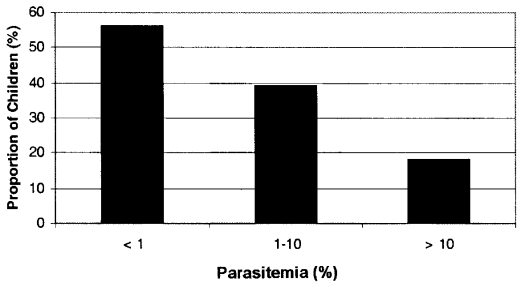

FIGURE 2. Proportion of children with a normal platelet counts (>150,000 × 10^9/L) versus parasitemia (χ^2 for trend = 113.3, $P << 0.0001$).

significant difference in severe thrombocytopenia (<50,000/mm^3) among children with severe and mild malaria (105 of 1,016 versus 40 of 353; $\chi^2 = 0.27$, $P = 0.60$). As in the study of Gérardin and others, none of the patients in either of these groups had any bleeding diathesis despite very low platelet counts.

The median platelet count for the 27 children who died (overall mortality = 2.0%) was not different from that of those who recovered (130,000/mm^3, interquartile range = 63–221,000/mm^3 versus 131,000/mm^3, interquartile range = 78–227,000/mm^3; $P = 0.62$), and thrombocytopenia was not associated with death (16 of 27 versus 760 of 1,342; $\chi^2 = 0.07$, $P = 0.79$).

Our study, albeit retrospective in design, included almost five times the number of patients as did Gérardin and others. It also had several comparable features, such as the proportion of children with thrombocytopenia (56.7% versus 56.2%) and severe malaria (74.2% versus 74.7%) and the inverse association with age (Figure 1) and parasite count (Figure 2). However, we were unable to find any significant association between thrombocytopenia and severity or mortality. This may be due to differences in the patient population or the geographic location. For example, the median age of the children at Kilifi was 2.1 years compared with 7.5 years in the study of Gérardin and others, but both studies have shown that the risk of thrombocytopenia in children with *P. falciparum* malaria increases with age. This is supported by the observation that the median platelet count for all children was 109,000/mm^3 in their study compared with 131,000/mm^3 in our study. Similarly, the overall mortality was 9% in their study compared to 2.0% in our study, and it is possible that the number of children who died in our group was too low for us to detect any significant difference in platelet counts. Other differences included the number of children admitted with *P. falciparum* malaria (319 children over an 18-month period compared with 1,369 over a 12-month period), management (Kilifi has a five-bed high dependency unit for severe malaria cases), and treatment (e.g., we administer a quinine loading dose) of children with malaria. Thus, further studies are clearly needed to determine the significance of platelet counts in African children with *P. falciparum* malaria.

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