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Treatment of Anaemia Associated with Acute Falciparum Malaria Infection

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SUMMARY

A prospective clincal study evaluating various forms of treatment for the anaemia associated with acute falciparum malaria is reported. It is shown that chloroquin treatment alone, with no supplementary therapy, still results in a return to near-normal hemoglobin levels.

INTRODUCTION

The anaemia often associated with acute infection by Plasmodium falciparum is usually attributed to hemolysis and increased activity of the reticuloendothelial system. (Areekul et al, 1972). This anaemia is often severe, with as many as 10% of children with malaria having hemoglobins below 5 gm%. (Schmitz and Gelfand, 1976). In clinical practice, the question often arises as to whether supplemental treatment with iron or folic acid or both is necessary in addition to the chloroquin given for the underlying malaria.

This study was pursued in an effort to see if supplementary treatment for the anaemia was desirable or necessary. It appears commonplace to treat any patient seen with anaemia in a developing country with iron, with the assumption that dietary lack of iron is widespread. Also, since folic acid is required for erythrogenesis, it has been recommended by Morley (1973), that children with anaemia and malaria in developing countries be treated with folic acid. The study was set up to evaluate these treatment modalities.

PATIENTS AND METHODS

The study was carried out at Macha Mission Hospital, a 208 bed rural hospital in the Southern Province of Zambia. The protocol was designed to include all pediatric patients with clinical malaria, positive blood smears for Plasmodium falciparum, and hemoglobins less than 9 mg%. It was conducted from February to July, 1977. Each child was treated with a standard chloroquin course and also assigned in sequence to one of four groups.

The groups were as follows:

Group A: received parenteral iron dextran (Imferon) daily for five days, dosage depending on weight.

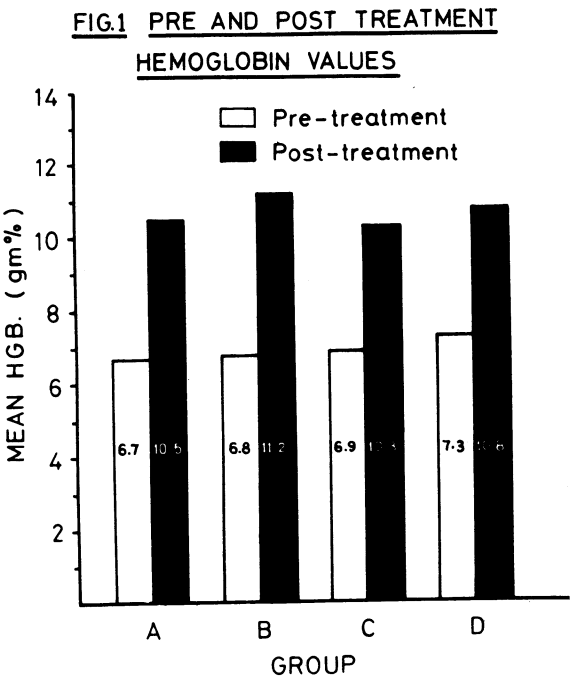
Group B: received folic acid 5 mg. orally, daily for five days.

Group C: received parenteral iron and also folic acid, dosages as above, daily for five days.

Group D: received no supplemental therapy (control group).

Stool specimens were also obtained on as many children as possible, and all cases of intestinal parasites were treated appropriately. This was done so that intestinal parasites, e.g. hookworm, would not contribute to on-going anaemia. After the full chloroquin course, and the supplemental treatment for anaemia, the child was discharged with a supply of

FIG. I



pyrimethamine (Daroprim), to use for weekly anti-malarial prophylaxis. Instructions were given to return in four weeks for a post-treatment hemoglobin check.

All patients with such severe anaemia that they were in heart failure, were given blood transfusions, and omitted from the study. Of the 128 children admitted to the study, only 45 returned for hemoglobin re-checks, and so the results and following discussions are based on these 45 cases.

RESULTS

The ages of the children studied ranged from six months to thirteen years, with the mean age being three years and five months old. The breakdown of the four groups is as follows:

Group A: (iron only)	– 9 cases
Group B: (folic acid only)	– 14 cases
Group C: (iron plus folic acid)	– 12 cases
Group D: (no therapy)	– 10 cases

The results are shown in Figure 1, giving the mean pre-treatment and mean post-treatment hemoglobin values for each group.

The pre-treatment hemoglobin values for all groups ranged from 2.6gm% to 8.8gm%, with the mean hemoglobin being 6.9gm%. The post-treatment hemoglobin values for all groups were all above 9gm%, except for five cases, with the mean hemoglobin being 10.7gm%. There was no relationship between the degree of anaemia and the parasite count on the blood smear, which agrees with the study by Nkrumah (1973).

DISCUSSION

In studying the results of the four groups of children, it can be seen that regardless of what group a patient was in, the hemoglobins all rose considerably. Although some of the groups showed a higher rise in post-treatment hemoglobin values than others, due to the small number of cases studied, it cannot be said if this was a significant difference. It should be noted that the hemoglobin rose 3.5gm% in those that received no supplemental therapy at all, while the mean rise for all groups was 3.8gm%.

Unfortunately, serum iron studies are unavailable to us, as it would have been interesting to study the ferrokinetics involved in all these patients. However in spite of this, on the basis of our clinical studies, it would seem that iron therapy is not necessary in the treatment of anaemia associated with malaria. This conclusion, of course, can only be applied to the population of children that we studied. The slightly higher mean rise of haemoglobin in those cases receiving folic acid alone was not statistically significant. In summary, treatment with chloroquin alone will result in a significant rise in haemoglobin after four weeks. Since 'normal' haemoglobin values in the population studied are unknown, it is not clear whether this rise in haemoglobin can be considered adequate.

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