Unusual presentation of malaria as a leukaemoid reaction

A case report

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Summary

In a 4-year-old black patient with Plasmodium falciparum malaria the diagnosis was established by observing plentiful gametocytes in the peripheral blood, although ring forms were very scanty. The blood picture was that of a leukaemoid reaction with severe anaemia, high total leucocyte count and thrombocytosis. Treatment with chloroquine and primaquine, together with packed red cell transfusions, was successful in eliminating both the malaria parasites and the leukaemoid blood picture. The rarity of malaria presenting as a leukaemoid blood picture is discussed.

Leukaemoid reactions are extremely unusual in malaria, although the appearance of frankly immature myeloid cells has been recorded. Such cases notwithstanding, there are to our knowledge no reported cases of malaria with a markedly elevated white cell count (> 50 x 10⁹/l) in which the evidence suggests leucocytosis reactive to the presence of parasites. Although mild leucocytosis (up to 15 x 10⁹/l) is not uncommon during febrile periods (due probably to shifts in the distribution between the circulating and marginating compartments), leucopenia is the usual picture in malaria.

Case report

A black boy aged 4 years was admitted to Natalspruit Hospital with pyrexia (38°C). There was striking pallor but no obvious jaundice, and the pulse rate was 110/min. Examination of the ears, nose, throat and lungs revealed no abnormalities. There were no symptoms or signs referable to urinary tract infection and the urine was macroscopically clear. The spleen was not palpable but the liver was enlarged — 5 cm below the costal margin. A blood count and thick and thin blood films yielded the following results:

- Haemoglobin concentration 2.4 g/dl, reticulocyte count 15% (suggesting haemolysis as the cause of the anaemia), total leucocyte count 56.0 x 10⁹/l, neutrophils 42%, monocytes 7%, lymphocytes 35%, eosinophils 3%, basophils 1%, metamyelocytes 6%, myelocytes 4% and promyelocytes 2%. Scanty normoblasts were also observed and platelets were markedly increased. In both thick and thin blood films numerous gametocytes and very scanty ring forms of Plasmodium falciparum were found (Fig. 1). A routine chest radiograph was normal and urinalysis revealed small quantities of protein and haemoglobin, possibly reflecting intravascular haemolysis.

Course of illness and treatment

The child was immediately transfused with packed red cells and treatment with chloroquine was instituted. The patient's condition remained unsatisfactory, however, with fever, a tachycardia of 100/min and gallop rhythm. More packed cells were transfused and by the third day after admission the haemoglobin concentration had risen to 9.8 g/dl and the liver had decreased in size (palpable 2 cm below the costal margin). There were still large numbers of gametocytes in the peripheral blood but no ring forms could be seen. The total leucocyte count had fallen to 15.2 x 10⁹/l.

Apart from chloroquine, supportive therapy of furosemide 40 mg orally and digoxin 0.2 mg every 8 hours was given. As gametocytes still persisted in the blood, albeit in smaller numbers, primaquine 1 tablet daily for 3 days was administered 18 days after admission. This proved successful and the child was dis-
charged from hospital in a satisfactory condition 7 days later. The total leucocyte count was $10.4 \times 10^9/\text{l}$ a few days before discharge.

**Discussion**

This 4-year-old boy presented with severe anaemia, a high total leucocyte count, fever and hepatomegaly. It was known that he resided in a malarial area. The very high white cell count suggested the possibility of a chronic myeloid leukaemia with coincidental infection by *P. falciparum*. However, the reduction of the white cell count to normal after antimalarial therapy alone indicates a leukaemoid reaction. To the best of our knowledge this is the first reported case of a frankly leukaemoid reaction, with markedly elevated white cell count, in malaria. Conceivably the unusual prominence of gametocytes in this case was associated with the atypical haematological manifestations.

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**REFERENCES**


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**Hard-metal lung disease**

**A report of 4 cases**

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**Summary**

The properties, manufacture and uses of hard metal are briefly described, and its harmful effects on the respiratory tract are enumerated, discussed and illustrated in 4 subjects who had been drill sharpeners on goldmines. It cannot be unequivocally claimed that these are cases of interstitial fibrosis due to the inhalation of hard-metal dust, as both radiography and histology in this disease are nonspecific. There appears however to be good reason to encourage further research on drill sharpeners on goldmines and tool-room workers in general.

Hard metal is useful because it is only slightly less durable than a diamond. It has the unique quality of becoming harder with increases in temperature and is therefore, among many other applications, used extensively in cutting-tool tips. Hard metal is manufactured by powder metallurgy. The chief components are tungsten and titanium carbides with smaller amounts of other metal carbides. Cobalt is included as a binder and may constitute up to 25% of the alloy. 1-3 The powders are mixed, pressed and formed into shapes and then sintered at high temperature. When cooled they are ground to produce the required article. 1 All these processes generate dust but the population at risk is rather small — not more than a few hundred in the RSA. The cutting edges on tools have to be reshaped at intervals by grinding and workers can then be exposed to dust. Possibilities for exposure occur in any large tool-room and affected workers may give as their occupations such descriptions as tool-maker, tool-grinder or tool-setter. 4-6 Among other uses, hard metal is now extensively used in the goldmining industry in the RSA for the tips of drills. The number of people at risk in this sector is unknown, but is certainly much larger than in the manufacturing process. Wet grinding cannot be assumed to reduce exposure. 3-7

Since World War II it has been realised that hard-metal dust exposure may result in serious health hazards. 1-3 Among these are chronic bronchitis, asthma and parenchymal lung disease, from desquamative alveolitis to chronic interstitial fibrosis, which was first described by Jobs and Ballhausen 8 in Germany in 1940. According to one authority 9 bronchitis and asthma are very common, occurring in up to 40% of employees exposed to dust during the manufacturing process, but are reversible on cessation of exposure. Parenchymal lung disease may stabilise 10 or even regress if detected early. 6, 10-12 But many exposed people die, often as a result of cor pulmonale. It is therefore important to be aware of and recognise the condition early.

The prevalence of parenchymal disease in exposed populations is not known. At least 100 cases and 30 deaths have been reported in the literature, and in many countries the condition...