AN UNUSUAL CASE OF CEREBRAL MALARIA

BY

CHARLES S. D. DON, M.D., M.R.C.P.

Temp. Major, R.A.M.C.; Medical Specialist

AND

P. F. MEYER, M.D.

Captain, R.A.M.C.; Graded Physician

This case is reported because of the large number of parasites in the blood film and the severe clinical signs, in spite of which recovery followed.

Clinical History

The patient, a soldier aged 22, was admitted to a C.C.S. in North Africa on July 10, 1943. We subsequently learnt that while on local leave in North Africa he became ill, but attributed the illness to the heat and did not consult a medical officer. He had no clear memory of how he reached this C.C.S.; he last remembers travelling in a train with other soldiers, who finally took him to the central M.I. room of this area.

He was admitted late at night, about five or six days after the onset of the illness. On examination he complained of headache and vomiting. He was drowsy but not unconscious, and could answer questions reasonably well. The temperature was 102.4° F. The spleen was easily palpable. The urine was of normal colour. Blood slides showed a very heavy malarial infection. The thick film was crowded with parasites. Examination of the central parts and the edges of the thin film revealed that, on an average, 35% of the red cells contained parasites. Of the infected cells 82% had one ring, 17% had two rings, and 1% had three or four rings per cell. Several crescents of *Plasmodium falciparum* were also seen.

He was given 10 gr. of quinine dihydrochloride in 20 c.cm. of water intravenously and 12 gr. by mouth at 11.30 p.m., the latter dose being repeated in six hours. Next day his condition was only fairly satisfactory; he was still drowsy, but he took 10 gr. of quinine six-hourly by mouth without vomiting. The temperature remained normal all day. A blood film showed large numbers of parasites to be still present. He took fluids fairly well, but passed only very small amounts of urine.

Early on the morning of July 12 the patient was found to be quite unconscious, cold, and collapsed. The tongue was hard and dry. The temperature was 97° F., the blood pressure 80/60, and the pulse 90 per minute. Hiccup was present, and breathing was shallow. As the patient appeared to be dying he was given an immediate injection of nikethamide (coramine). No urine had been passed since the previous night and the bladder appeared to be empty, but when a catheter was passed an ounce of urine was obtained which contained albumin but no blood. Microscopically, granular and hyaline casts were seen. A glucose-saline drip was put up, and by this route he was given in the course of the day 30 gr. of quinine dihydrochloride and two blood transfusions, each of 500 c.cm., without a reaction of any kind. The patient was Group B, and each donor was of this group. The serum and red cells of the patient and of each donor were cross-matched and the results read microscopically at the end of one hour. A blood film of the patient showed large numbers of parasites to be still present. The haemoglobin before the transfusion was 70% (Sahli). By evening his condition had improved; the B.P. had risen to 105/80, and the temperature was 101° F. No urine was passed that day.

The following morning—July 13—the patient was semiconscious, and was able to take fluids by the mouth. The temperature was normal, the conjunctivae showed slight jaundice, and a specimen of urine voluntarily passed was typical of that seen in blackwater fever. Spectroscopic examination revealed the presence of oxyhaemoglobin. No red cells were seen in the centrifugized deposit, but granular and hyaline casts were present. While the compatibility of the transfused blood appears to have been established by indirect grouping and careful direct cross-matching, the possibility remains that the haemolysis observed was not due to quinine but to antibodies in the recipient's blood which could not be detected by the methods at our disposal. There was no history of any previous blood transfusion. The intravenous drip was continued; no further quinine was prescribed, and the patient was given by mouth one drachm of sodium bicarbonate hourly to alkalize the urine rapidly. In the course of the day he was given two intramuscular injections of mepacrine methanesulphonate (atebrin musonate), each of 0.1 g., and the urine gradually returned to a normal colour. At 6 p.m. the temperature was 103.6° F.; he was given mepacrine hydrochloride (atebrin) 0.1 g. by mouth, and this was repeated in 6 hours. At 9 p.m. the temperature, which had been taken hourly, had risen to 106° F., and the patient, after having remained semiconscious all day, became delirious and restless. Cold sponges were given half-hourly until 11 p.m., when the temperature had fallen to 102° F.

The intravenous drip was stopped, as oedema of the sacrum was found to be present; he was passing urine freely.

On July 14 the patient's general condition was much improved; for the first time he was quite conscious and rational. The morning temperature was 98° F. A blood film showed no parasites; the red cells exhibited a marked degree of macrocytosis, and megaloblasts were present. The urine still contained albumin and casts. Mepacrine hydrochloride was continued in a dosage of 0.1 g. t.d.s. That evening the temperature rose to 104.8° F.

On July 15 his general condition was better, but he was found to have thrombophlebitis of the left axillary vein (this arm was used for the drip) with oedema of the forearm and hand. The morning temperature, which so far had always been normal, was 100.4° F.

Temperature chart of the case.

There were rales over the lower lobes of both lungs but no dullness. It was decided that he had a hypostatic pneumonia, and was put on sulphadiazine 1 g. four-hourly. The temperature reached 102° and 101° F. on the next two nights, and thereafter settled, remaining normal all day on July 21. By this time the lung signs had disappeared.

Summary

A case of cerebral malaria is reported in which 35% of the red cells were infected by one or more parasites of *Plasmodium falciparum*.

The most-pronounced cerebral symptoms came on after the patient had been on large doses of quinine for 36 hours.

Transient haemoglobinuria followed 30 gr. of intravenous quinine and two blood transfusions.

Hyperpyrexia was found to be developing after three days of intensive quinine treatment.

Hypostatic pneumonia complicated recovery.

We wish to thank Major-Gen. E. M. Cowell, the D.M.S., North African Force, for permission to submit this report for publication.

EFFECTS ON RATS OF PROLONGED FEEDING
WITH THE STAPLE AFRICAN DIET

BY

JOSEPH GILLMAN, D.Sc., M.B.

(From the Department of Anatomy, University of the
Witwatersrand, Johannesburg)

It is known from numerous surveys and observations that pulmonary tuberculosis and other diseases of the respiratory tract are especially prevalent among the Africans (South African negroes). It is also common knowledge that cirrhosis of the liver and especially primary cancer of the liver are very often encountered in this people. In an extensive investigation into the incidence of cancer in South Africa, Berman (1940, 1941) has shown that in a series of 253 cases of cancer of all organs in adult male Africans working in the Witwatersrand gold mines no fewer than 222, or 90%, were primary in the liver. This remarkable state of affairs is even more strongly emphasized when it is remembered that liver cancer is the rarest neoplasm in Europeans.

Several factors are regarded as contributory to this high incidence of cancer, one of which is cirrhosis, which is almost invariably associated with liver cancer. There is a considerable body of opinion which regards this susceptibility to liver cancer