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Cerebral malaria in Tanzania. Its epidemiology, clinical symptoms and neurological long term sequelae in the light of 66 cases

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Summary

A neurological study of 66 patients with the cerebral form of *Plasmodium falciparum* malaria is presented. The patients were diagnosed, treated and re-examined at the Mnero Hospital, south-east Tanzania. Epidemiological aspects, neurological symptoms and the results of re-examination within six months of discharge from hospital are described. Although the mortality rate was low, some degree of neurological disability could be detected in a number of children at the follow-up examination.

Introduction

In European countries indigeneous malaria is almost extinct (BÖRSCH *et al.*, 1982) but foreign travellers frequently import malaria (BRUCE-CHWATT, 1982). In West Germany there has been a steady rise in the number of imported cases from 30 per year in 1963 to 539 in 1978 (WEISE, 1979). 43·3% of these cases were *Plasmodium falciparum* infections and, according to WEISE (1979), almost 5% of these imported *P. falciparum* infections were fatal.

Cerebral malaria is one of the most threatening plasmodial infections. It was first described as "febris perniciosa comatosa" by Kraepelin (1881). Many authors reported on malaria complicated by various symptoms of the central nervous system (BOSCHES, 1947; DAROFF *et al.*, 1967). BERNER (1975) reported 39 cases of cerebral malaria in children and WARRELL *et al.* (1982) reported a series of 100 patients with cerebral malaria in Thailand. Some authors studied EEG alterations in cerebral malaria (COLLOM, 1977), but few neuroradiological findings have been described (VIETZE, 1978).

Materials and Methods

At Mnero Hospital, Tanzania, 8456 cases of malaria were diagnosed by thick film examination during the three-year period 1979-1981 but in many cases the thin films were used for further differentiation. Of these 8456 cases, 541 (6·4%) were *P. vivax* and 7915 (93·6%) *P. falciparum*. 66 (0·83%) of the 7915 *P. falciparum* patients developed cerebral malaria.

Diagnosis of cerebral malaria was based on clinical features (impairment of consciousness, acute convulsions and focal cerebral signs), positive blood slide and exclusion of septic or aseptic meningoencephalitis by examination of the cerebrospinal fluid. Monthly rainfall was measured at the Catholic Mission Station Mnero, District Nachingwea, Tanzania.

Epidemiology

In non-immune individuals cerebral malaria occurs at all ages, but in holoendemic areas it mainly strikes children less than five years old (MANSON-BAHR, 1982). This age distribution can also be seen in the 66 patients treated at Mnero Hospital.

Table I—Age distribution in 66 patients with cerebral malaria

Age (in years)	No. of patients
0-0·5	0
0·5-1	9
1-2	22
2-3	20
3-4	8
4-5	5
5-15	2
15+	0
	66

The peak frequency is between the second and third year of age; no cases were observed in newborn babies and infants aged less than six months.

Fig. 1 shows the monthly distribution of all malaria infections at Mnero Hospital from 1979-81 with a clear maximum in February to June; this is related to the climate in southern Tanzania. The wet season starts early in December with the first big rains early in January and second peak at the end of March/April. About mid-May the rain usually stops completely. The incidence of cerebral malaria is high during the rainy season and continues so for a further two months (Fig. 2). The average fatality rate of all cerebral malaria infections was 18·2%. The staple food is maize and millet which are harvested in May to June. The months before harvest are often characterized by lack of sufficient food and we found that the incidence of diarrhoeal diseases and anaemia in children reached its peak during the rainy season. In addition to the intensity of *P. falciparum* infection, malnutrition, diarrhoea and anaemia seem to play a decisive role in the outcome of cerebral malaria.

Observations

Clinical symptoms

The first symptoms of cerebral malaria in the patients treated at Mnero Hospital were hyperpyrexia, headache, vomiting and restlessness. Their onset

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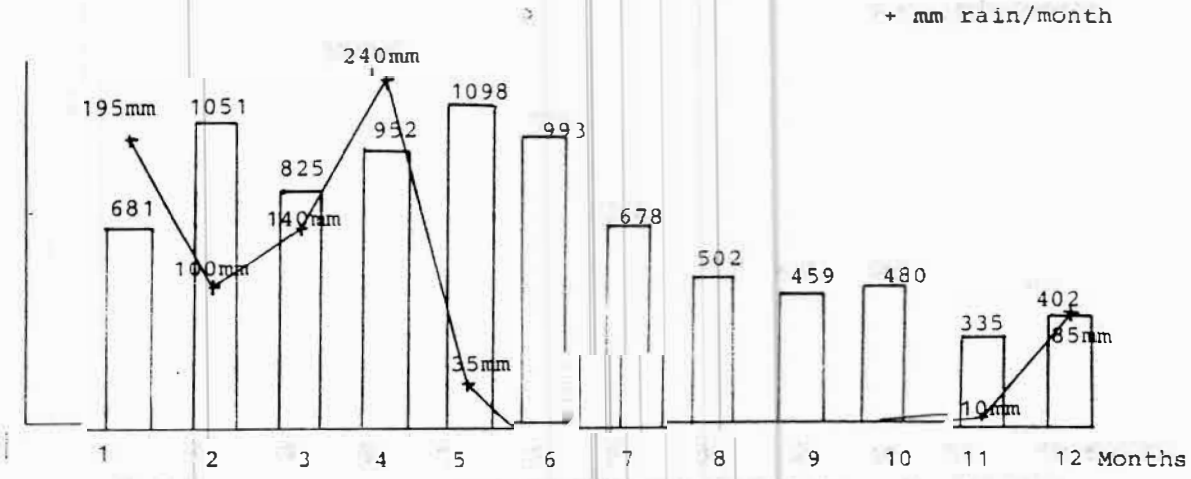

Fig. 1. Monthly distribution of 8456 cases of malaria 1979–81 compared with the mean monthly rainfall 1979–81

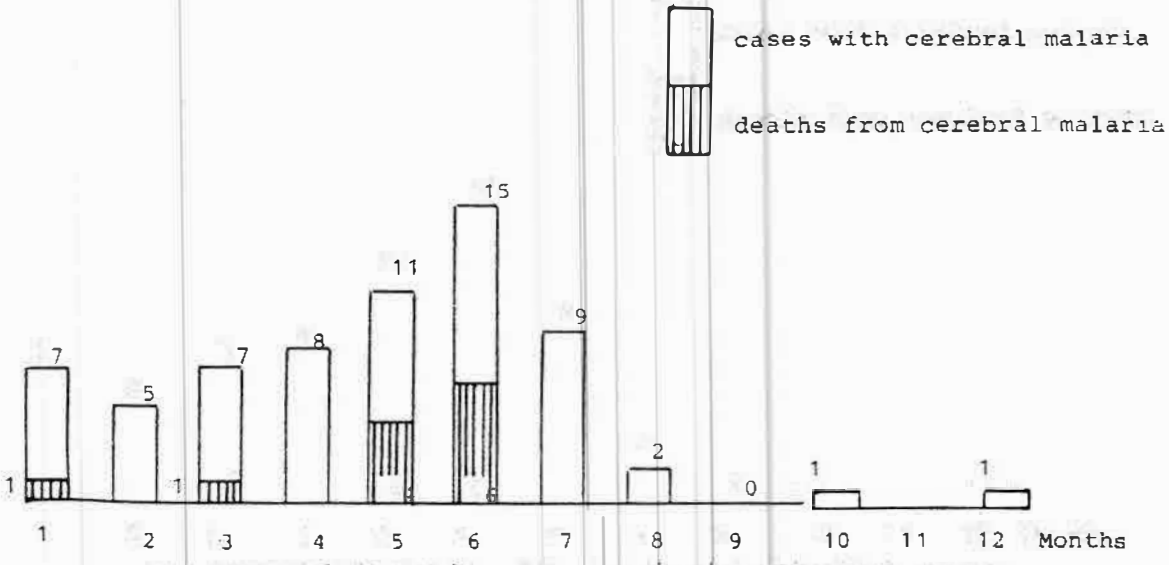

Fig. 2. Monthly distribution of admissions of 66 patients with cerebral malaria and deaths per month

was usually abrupt and dramatic. Many children developed convulsions, followed by a coma with all the signs of midbrain syndrome, but some cases showed lateralization. In all 66 patients blood slides were positive for *P. falciparum*.

Lumbar puncture often revealed a raised CSF pressure a slight to moderate positive Pandy reaction and, sporadically, lymphocytes. The frequency of cerebral symptoms in all the patients with cerebral malaria who were admitted at Mnero Hospital during the three years period 1979-81 was as follows:

1. Hyperpyrexia	62	94%
2. Headache	31	47%
3. Vomiting	40	60·6%
4. Neck stiffness	10	15·2%
5. Organic psychosyndrome	10	15·2%
6. Epileptic fits— focal or generalized	42	63·3%
7. Comatose state with signs of midbrain syndrome	48	72·7%
8. Focal signs	16	24·2%
9. Abrupt onset of symptoms	53	80·3%

Table II—Results of the re-examination of 66 patients with cerebral malaria within six months of discharge from hospital 1979-81

Finding	No.	Percentage
No residual symptoms	39	59%
Organic psychosyndrome	5	7·5%
Hemiparesis/hemihypaesthesia	3	4·5%
Residual epileptic fits	2*	3%
with residual hemiparesis	1	1·5%
Death in bulbar brain syndrome	12	18%
Patients not seen	5	7·5%

*includes one who also had hemiparesis.

Table II analyses the results of re-examination of the 66 children who had suffered from cerebral malaria. 12 children died with signs of bulbar brain syndrome. Re-examination within six months of discharge from hospital showed 39 children to be completely free from symptoms; five children had signs of slight to moderate organic psychosyndrome, three had spastic hemiparesis, two suffered from a residual epilepsy and one of these also showed slight paresis of the left arm. Five children could not be seen at the follow-up examination.

Conclusions

Cerebral malaria is a life-threatening infection with *P. falciparum*. In holoendemic areas the peak age distribution is between two and three years. There is a direct correlation between morbidity/mortality of cerebral malaria and climate, supply of staple foods in rural areas where agriculture is run on a subsistence level, diarrhoeal diseases, decompensated anaemias and malnutrition. The onset of cerebral malaria in children is usually very abrupt and dramatic.

In various series reported the mortality rate ranges from 4 to 50% (BERNER, 1975; MANSON-BAHR, 1982; WARRELL *et al.*, 1982). In our series of 66 children it was 18%. The percentage of cerebral malaria in all parasitologically proved *P. falciparum* infections was 0·83% which, compared with 1·5% in similar reports (DAROFF, 1967; MADECKI *et al.*, 1966), appears to be low. The main reason seems to be the simple but well distributed medical infrastructure of Tanzania with first aid posts and dispensaries.

Although in this series of children suffering from cerebral malaria and treated in a rural East African hospital, the mortality rate was rather low (18%), the percentage of children in whom some degree of disability could be detected, in a neurological control examination within six months of discharge from hospital, is fairly high. This seems to confirm that the true rate of disability resulting from cerebral malaria is higher than usually assumed.

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