# Recent Experiences with Severe and Cerebral Malaria

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## SUMMARY

**Experience in cerebral and other severe forms of falciparum malaria in a Transvaal lowveld mission hospital in the years 1971 and 1972 is discussed.**

**Treatment of these patients with chloroquine, heparin, low molecular weight dextran and steroids in 1972 was found to be more successful than other conventional regimens used in 1971.**


*S. Afr. Med. J.*, **48**, 1353 (1974).

We present some of our experiences of malaria, especially severe and cerebral forms, during the years 1971 and 1972 in the Shongwe Mission Hospital, a 253-bed general hospital with an average of 389 inpatients per day. There are 2 resident doctors at this hospital which lies in the Swazi homeland at an altitude of approximately 360 metres. The area we serve varies in altitude from about 150 to 400 metres. Our borders are Swaziland, Mozambique, the lower part of the Kruger National Park and westwards as far as Kaapmuiden.

In 1971 we admitted 116 malaria cases out of a total of 4 474 admissions and in 1972, 283 malaria cases out of 4 367 admissions. Our criteria for admission were: diagnosis in doubt; severe forms; complications.

Those patients whose clinical diagnosis we were reasonably sure of were treated as outpatients, that is, if they could walk unaided. Of these there were more than 200. In 1971 most of our malaria cases were inpatients, while in 1972 a much greater proportion were treated as outpatients. This will explain the discrepancy of our comparative figures for these 2 years (Table I).

Before entering into an analysis of our cerebral malaria patients we will put them into their rightful place in the over-all malaria picture as we see it at Shongwe. For this analysis we have only considered the patients admitted.

## CEREBRAL MALARIA

Cerebral malaria is defined as those cases where the patient is comatose or semicomatose and only responds to painful or irritating stimuli. If a patient is able to respond to the spoken word but has evidence of central nervous involvement in the form of fits, neck stiffness or local neurological signs, we call them cerebral signs—we tried not to include patients with the confusional state of a rigor, which is usually of short duration, in this group. In our experience those with cerebral signs are only a very short step away from true cerebral malaria and thus these patients also demand prompt and adequate treatment.

In 1971 we treated cerebral malaria and severe malaria patients with intramuscular chloroquine (Resochin), routine intravenous and other supportive therapy, and with Daraclor if they were able to take and retain oral medication. Some were given sodium pentosan polysulphate (Tavan-SP-54) but we were not convinced of its value.

There were 6 cerebral malaria patients, of whom 4 died, 1 on the 1st day; 1 on the 2nd day; 1 on the 18th day and 1 on the 28th day. One discharged 20 days afterwards was mentally subnormal; and 1 was discharged well after

TABLE I. SUMMARY OF MALARIAL PRESENTATION

| | 1971 | Deaths | 1972 | Deaths |
|---|---|---|---|---|
| Total hospital admissions (excluding maternity) | 4 474 | | 4 367 | |
| Malaria admissions | 116 | | 283 | |
| Cerebral malaria | 6 | 4 | 41 | 9 |
| Cerebral signs | 11 | | 53 | 1 |
| Haemoglobin below 10 g/100 ml (not all tested) | 30 | | 87 | |
| Enlarged tender liver or jaundice, or both | 43 | | 69 | |
| Gastro-enteritis | 39 | 1 | 68 | 2 |
| Enlarged spleen | 32 | | 43 | |
| Respiratory involvement | 22 | | 42 | 2 |
| Blackwater fever | 0 | | 1 | 1 |
| Deaths — total | | 5 | | 15 |

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Paper presented at the Malaria Symposium held at Nelspruit, Eastern Transvaal, on 27 January 1973 under the auspices of the Lowveld Division of the Northern Transvaal Branch of the Medical Association of South Africa.

8 days. Of those with cerebral signs 5 responded on the 1st or 2nd day of treatment and 6 took 3 days or longer to respond. In view of this gloomy picture we were understandably depressed.

Towards the end of April 1971 we read the excellent article by H. Smitskamp and F. H. Wolthuis entitled 'New concepts in treatment of malaria with malignant tertian cerebral involvement' which appeared in the *British Medical Journal* of 27 March 1971. Our management of cerebral malaria and some of our severe malaria patients during 1972 is based on their regimen which includes:

(a) chloroquine phosphate (Resochin) intramuscularly—dosage according to weight;

(b) heparin 6-hourly intravenously for 4 to 6 doses; (adults 12 000 units, then 6 000 units every 6 hours, children and infants—smaller doses);

(c) low molecular weight dextran (Macrodex or Rheomacrodex 500 ml/24 hours — usually only one dose);

(d) steroids; Solu-Cortef (100 mg) or Venocortin (112 mg) 6-hourly intravenously for 4 to 6 doses;

(e) blood and other intravenous and supportive therapy as dictated by the needs of the individual patient;

(f) Daraclor or chloroquine taken orally when the patient is able to.

We have had no experience with quinine due to difficulty in obtaining it.

Of the 41 cases of cerebral malaria treated in 1972, 9 died, one of the deaths being due to incorrect diagnosis in a patient treated for meningitis. Our treatment of these cases depended on our experience, at the time, of this regimen and on the availability of the respective drugs.

**TABLE II. DRUGS USED AND THE RESULTS**

| Drugs | Cases | Deaths |
|---|---|---|
| Heparin<br>Low molecular weight dextran<br>Steroids | 30 | 5 |
| Steroids<br>Heparin | 6 | 1 |
| Heparin<br>Low molecular weight dextran | 2 | — |
| Steroids | 1 | 1 |
| Only chloroquine | 1 | 1 |

All patients received chloroquine intramuscularly and intravenous supportive therapy, but the drugs varied, as is briefly summarised in Table II.

The assessment of deaths of those patients who received the full treatment is summarised as follows:

A young man of 18 years died 15 hours after admission. He was not seen again by a doctor after admission. It is therefore not possible to assess the reason for the failure of the treatment.

A young woman was admitted in a shocked and comatose state after an incomplete abortion. Her haemoglobin level was 3,6 g/100 ml. Heparin was withheld for about 2 hours and only one unit of blood was available. She died 7 hours after admission.

A young woman was admitted in a shocked and comatose state. Her haemoglobin level was 3,5 g/100 ml. She delivered a stillborn infant with postpartum haemorrhage. Only one unit of blood was available and she died after 16 hours.

An 11-year-old child died 4 hours after admission; a 2-year-old child died half an hour after admission and start of treatment, and a 7-month-old infant died 7 hours after admission and only received heparin and steroids.

One of the most impressive aspects of this regimen was the rapid response. Twenty of those who received the full treatment woke up on the 1st or 2nd day and only 5 took 4 days or longer to awake.

No patient who survived more than 16 hours after the start of this regimen died, and only 2 were discharged with obvious residual cerebral signs. One was an infant who arrived gasping and cyanosed, and the other, an infant in whom treatment was delayed for 2 days after admission.

Our biggest problems in the 1972 epidemic were: lack of blood, which was virtually unavailable; overtaxed and inadequate laboratory services; an overfull hospital with a shortage of nursing staff; and the fact that the other work of the hospital had to continue.

Bearing in mind these problems and the fact that we could not give these patients our undivided attention, we are more than satisfied with our results and feel that we can recommend this regimen unreservedly.