Periodic paralysis complicating malaria

NIMAL SENANAYAKE*
M.D., M.R.C.P.

SUNIL J. WIMALAWANSA†
M.B. B.S.

Departments of *Medicine and †Biochemistry, Faculty of Medicine, University of Peradeniya,
Peradeniya, Sri Lanka

Summary

Episodic muscular weakness, commonly associated with alterations of serum potassium, is the cardinal feature of periodic paralysis. The combination of transient hyperkalaemia and rigors occurring during febrile episodes of malaria is suggested as the underlying cause which precipitated the muscular paralysis. Three patients with malaria who developed a similar paralysis during the paroxysms of fever are described to illustrate this.

Introduction

Neurological complications of malaria may result in a spectrum of clinical manifestations ranging from peripheral neuritis to coma (Russell *et al.*, 1963). Three patients are described who, during febrile episodes of malaria, developed transient generalized muscular paralysis very similar to that seen in periodic paralysis.

Case reports

Patients 1 and 2

Two males aged 39 and 50 years were admitted to hospital with a history of febrile illness of 2 days' duration. The description of the fever which was accompanied by chills and rigors, was very suggestive of malaria. The unusual feature of the history was that both patients complained of muscular weakness progressing to complete paralysis of limbs following rigors. On admission to hospital 4-6 hr later, the weakness had decreased and the patients were able to sit up unaided. None of the patients had experienced similar muscular paralysis in the past and there was no similar history among the family members.

Physical examination on admission revealed weakness of all 4 limbs, and diminished tendon reflexes. The rest of the nervous system was normal. The spleen was palpable 3 cm below the left costal margin in both cases. The cardiovascular and respiratory systems were clinically normal.

The patients developed a further attack of rigors on the second day in hospital. This was followed by

generalized muscular paralysis similar to that described on the previous occasion. The lower limbs were affected first. Within one hr all limbs were totally paralysed. Physical examination at this stage showed flaccid paralysis of the limbs, depressed tendon reflexes and flexor plantar responses. There was no sensory impairment, the respiratory muscles and sphincters were unaffected, and the patients were conscious and fully orientated. Signs of improvement became apparent 4-6 hr later with the muscles affected last being the first to recover.

Complete recovery occurred in 8 hr in case 1 and in 10 hr in case 2 after the first onset of weakness. The clinical diagnosis of malaria was confirmed in both cases by examination of a peripheral blood film which showed heavy mixed infections with *Plasmodium vivax* and *P. falciparum*. The patients were treated with oral chloroquine. They remained afebrile and asymptomatic thereafter, and were discharged after 2 days.

Patient 3

A 30-year-old male presented with a 3-day history of intermittent fever with chills and rigors. On the day of admission, at about 2 p.m. he had developed an attack of chills and rigors lasting 4-5 min. Soon after, he had noticed weakness of the lower limbs. The weakness soon spread to the upper limbs and in half to one hr, paralysis of all 4 limbs was complete. He was admitted to hospital at this stage.

There was no past history or family history of similar illness.

On examination, the signs were very similar to those in patients 1 and 2, with flaccid paralysis of all 4 limbs and depressed tendon reflexes. The spleen was palpable 2 cm below the costal margin.

The patient showed signs of improvement after 10 hr. By midnight, he was able to move his arms, and by next morning the weakness had completely disappeared. On the fifth day of his illness he developed another bout of fever, but on this occasion it was not accompanied by rigors or

muscular weakness. Examination of a peripheral blood film showed erythrocytic stages of *P. vivax* confirming the diagnosis of malaria. The patient was treated with chloroquine and primaquine and was asymptomatic thereafter.

Discussion

The muscular paralysis which affected these patients closely resembled the episodic weakness seen in familial periodic paralysis. The pathophysiological mechanisms underlying this condition are not well understood, but the attacks seem to be closely related to changes in plasma potassium concentration. Three types of periodic paralysis have been identified depending on whether the plasma potassium falls, remains normal or rises during the attacks (McArdle, 1969). Paralysis which occurs with hypokalaemia may be provoked by i.v. administration of glucose and insulin which lowers plasma potassium. Conversely, paralysis which occurs with hyperkalaemia may be provoked by administration of potassium chloride orally or i.v. (McArdle, 1969). Severe alterations in plasma potassium associated with potassium depletion or retention may produce sporadic cases of periodic paralysis (McArdle, 1969).

Transient rise of plasma potassium during febrile episodes of malaria is well known (Russel *et al.*,

1963; Manson-Bahr, 1966; Adams and Macgrath, 1966) and this has been ascribed to lysis of red cells and intense muscular contraction during rigors. It is also of relevance to note that one precipitating factor of periodic paralysis is vigorous muscular exercise (McArdle, 1969). In the present patients, the plasma potassium was not estimated either during or after the attacks of paralysis. However, it seems reasonable to postulate that the 2 factors mentioned above, namely, the transient hyperkalaemia and the rigors causing intense muscular contraction occurring during febrile episodes precipitated the muscular paralysis in these patients. The rarity of this occurrence may suggest the possibility that the patients described had some genetic predisposition.

References

- ADAMS, A.R.D. & MACGRAITH, B.G. (1966) *Clinical Tropical Diseases*, 4th Edn, p. 217. Blackwell Scientific Publications, Oxford.
- MANSON-BAHR, SIR P.H. (1966) *Manson's Tropical Diseases*, 16th Edn, p. 45. Baillière, Tindall & Cassell, London.
- MCARDLE, B. (1969) Metabolic and endocrine myopathies. In: *Disorders of Voluntary Muscles* (Ed by Walton, J.N.), 2nd Edn, pp. 607–614. Churchill, London.
- RUSSELL, P.F., WEST, L.S., MANWELL, R.D. & MACDONALD, G. (1963) *Practical Malariology*, 2nd Edn, pp. 369, 387–389. Oxford University Press, London.