Malaria myositis

Muscular pain and weakness are typical presenting features of polymyositis. Our patient's myositis syndrome proved to be the principal feature of falciparum malaria. A 25-year-old professional football player, born in Ghana but residing in Britain for the age of 11 years, visited his family in Kamshe, Ghana in May 1991. Six weeks after arriving in Ghana he developed fever and diarrhoea. These symptoms continued for two weeks. At the local hospital he was treated with two daily injections of chloroquine. He returned to England two days later but still felt unwell and was unable to resume training because of muscular pain and fatigue. He was required to run five miles as part of this training, but found that he could not keep up with his team-mates. He was referred to a physician, who noted tenderness in his thighs, but found no other abnormalities, but no weakness. The haemoglobin was 11.4 g/l, white blood cell count 6600/mm³, erythrocyte sedimentation rate 42 mm in the first hour, and, bilirubin 16 mmol/l. Falciparum and malariae infections and alkaline phosphatase were normal. The creatine kinase (CK) level at this time was 11,000 IU/l (normal < 195 IU/l). Several days later the CK level was normal and resting muscle pain ceased. However, because of muscle and muscle pain on exercise, he was still unable to resume training. Markers of autoimmune disease, an antibody screen for viral and other infections, and a sickle-cell test, were all negative. Electromyography showed myopathic features. A diagnosis of idiopathic inflammatory myopathy was considered likely. Muscle biopsy was refused.

Five weeks after the onset of the illness he was referred for advice on management. Review of the history revealed that fever, fatigue, and muscle pain had become periodic, occurring every third day. He admitted that he had not taken antimalarial prophylaxis while in Ghana, believing himself immune since he had been born in that country. Physical examination was normal, but a blood film revealed parasites of Plasmodium falciparum in red blood cells. He was treated with a course of quinine sulphate 600 mg three times a day for a week with Fansidar (pyrimethamine 25 mg with sulfadoxine 500 mg) three tablets on the eighth day. After completing this treatment he resumed training but, after a three-month run, his muscles ached and the blood CK rose to 1650 IU/l, falling to 572 IU/l three days later. He returned to play as centre-forward in the first team two months later; CK levels at this time were normal.

Inflammatory myopathy is the commonest cause of muscle disease in adult life. In developing countries, most cases are idiopathic, but a number of infections form part of the differential diagnosis. In our patient the diagnosis of falciparum malaria was suggested by the history.

Circulatory changes occur in the microvasculature in malaria, consisting of increased viscosity, obstruction of capillaries with agglutinated red blood cells and, sometimes, microvascular coagulation, 2 changes that can cause skeletal muscle necrosis with myoglobinuria. 3 These concepts resemble those suggested in idiopathic inflammatory myopathies. 4 Exercise releases CK from damaged muscle fibres. The delayed recovery observed in our patient, who had been partially treated earlier in the course of the disease, is consistent with these mechanisms.

There were 2300 imported cases of malaria in the UK in 1991, with 12 deaths. 5 The risk of infection during unprotected travel in West Africa is 2% per month. 6 Falciparum malaria often presents with non-specific symptoms, of which muscle pain is a component. 7 A quarter of children admitted to hospital in coma with P falciparum malaria in The Gambia had a raised CK level, whereas those with mild symptoms had normal CK levels. 8 Our patient had unusual in that involvement of muscle, causing muscle pain, loss of exercise tolerance, and exercise-related increase in CK level was the principal manifestation of malaria.

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Subtle cerebral lesions in "chronic whiplash syndrome"?

In an interesting study of whiplash injury, Ettlin et al 4 claim "possible damage to basal frontal and upper brainstem structures" evident in largely reversible "defects of attention and concentration", and by abnormal oto-neurological tests (in nine of 17 patients) within two weeks of injury. This implies subtle structural damage which increases the behavioural response to pain. To explain the claimed continuation of neck pain and stiffness, other authors have incriminated, but not consistently demonstrated, subtle lesions in the cervical cord, roots, or facet joints.

The issue may, however, be clouded, unless patients with acute nerve root or cord symptoms or signs are excluded from the study. Recovery from acute whiplash injury is complete within three months in about 75% patients in most large published series. Chronic whiplash, however, is contentious. 3 The outstanding features are: the unexplained high incidence in women; the prolonged nature of symptoms and apparent disability without attendant objective signs; the common association with anxiety, fatigue, irritability 1,2 and, sometimes, simulated physical disability. 3,4 The commonest sequelae of trauma, immobilisation and analgesics are often ineffective. In published series no good control group exists; most sufferers are involved in litigation.

MRI provides a superior and sensitive method of showing such putative structural lesions, 4 and brainstem auditory evoked potentials (BAEP) are commonly abnormal in brainstem lesions, including vestibular pathologies. I have therefore reviewed the major published findings (table), including results of Ettlin et al. 1 Despite reports 1 of non-specific defects of attention, concentration, and vestibular function, the absence of demonstrated structural lesions in the brain and cervical canal and joints is the most telling, and, in my view, conclusive evidence against any theory that invokes an anatomical disorder as the explanation for the continuation of symptoms. It corresponds to the absence of conventional physical and radiological signs and to the normal results of BAEP studies. 4, 5, 6 I have almost all of 14 patients, nine with acceleration hyperventilation "whiplash" injuries and five injured by direct, frontal head trauma who underwent MRI within four months of injury. Five of these patients with anterior spinal column injuries showed characteristic separation of the disk from the vertebral end plate, lesions still evident as late as nine months after injury. 4 This small mixed series, however, includes all lesions which should be excluded by definition. Yarnell and Rossie 7 in patients with severe debility 12 months after injury, conclude that "neurological examination, imaging and clinical electrophysiological studies were unable to localize structurally or functionally, the source of the (neuropsychological) dysfunctions".

Objective evidence shows that victims of acute whiplash injury have sustained no more than a muscular-ligamentous sprain; therefore, unusually protracted complaints ("chronic whiplash") may demand explanations that lie outside the fields of organic and psychiatric illness,—a view possibly supported by a current publication 8 claiming striking alleviation due to soft-tissue injections with water.

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| MRI of brain and cervical canal, and brainstem auditory evoked potential (BAEP) studies in whiplash injuries | Table 3 |
|---|---|---|---|---|
| | Maimani et al. | Van Meyden et al. | Yarnall and Rossie 8 | Ettlin et al. 1 | Total |
| No. patients | 4 | 15 | 27 | 15 | 61 |
| Abnormal MRI/brain | 0/4 | 4/15 | 0/27 | 0/15 | 4/61 |
| Abnormal MRI/neck | 0/4 | 0/15 | 0/27 | 0/15 | 0/61 |
| BAEP abnormal/total | 0/17 | 0/14 | 0/23 | 0/31 |

* Only mild arthropathy, 1 small non-specific frontal white matter signal. ** ND = no data.
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