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 since the age of 11 years, visited his family in Kamase, 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 muscle 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 did not take antimalarial margins, 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, haemoglobin A1c 7.3%. Blood and urine for microscopy 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 had disappeared. However, because of the ague 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 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. He was drawn 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 the treatment he resumed training but, after a three-mile 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 different cases 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, haemorrhagic coagulation, changes that can cause skeletal muscle necrosis with myoglobinuria. These concepts resemble those suggested in idiopathic inflammatory myopathies. Exercise releases CK from damaged skeletal 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. The risk of infection during unprotected travel in West Africa is 2% per month. Falciparum malaria often presents with non-specific symptoms, of which muscle pain is a component. 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. Our patient was 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.

M SWASH
Department of Neurology,
Section of Neurological Science,
Royal London Hospital,
London E1 1BB, UK
M S SCHWARTZ
Department of Neurology,
Addenbrooke’s Hospital,
Copp's Hill, Whaddon,
London SW20 1NE, UK

Correspondence to: Dr M Swash


Subtle cerebral lesions in “chronic whiplash syndrome”?

In an interesting study of whiplash injury, Ettlin et al 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 months 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 incubated, 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 have signified onto pre-accident spondylosis are excluded from whiplash syndromes. Recovery from acute whiplash injury is complete within three months in about 75% patients in most large published series. Chronic whiplash, however, is contentious. 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, irritability1-3 and, sometimes, simulated psychological phenomena. The sequence 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 pathways. I have therefore reviewed the major published findings (table), including results of Ettlin et al.5 Despite reports of non-specific defects of attention, concentration, and exercise-related increase in CK level, the absence of demonstrated structural lesions in the brain and cervical canal is still the most telling, and, in my view, conclusive evidence against any theory that involves anatomical disruption, 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.6-7 Davis and colleagues8 almost all 14 patients, nine with acceleration hyperextension “whiplash” injuries and five injured by direct, frontal head trauma who underwent MRI within four months of injury. Five of seven patients with anterior spinal column injuries showed characteristic separation of the disc from the vertebral end plate, lesions still evident as late as nine months after injury.8 This small mixed series, however, is conclusive. The absence of lesions which should be excluded by definition. Yarnell and Rossie9 in patients with severe debility 12 months after injury, concludes that “neurological examination, imaging and clinical electrophysiological studies were unable to localize structurally or functionally, the source of the (neuropsychological) dysfunctions”.

Objectie 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 publication10 claiming striking alleviation due to soft-tissue injections with water.

J M S PEARCE


Table

<table>
<thead>
<tr>
<th>MRI of brain and cervical canal, and brainstem auditory evoked potential (BAEP) studies in whiplash injuries</th>
</tr>
</thead>
<tbody>
<tr>
<td>Maimaris' Van Meydam et al</td>
</tr>
<tr>
<td>No. patients</td>
</tr>
<tr>
<td>Abnormal MRI/brain</td>
</tr>
<tr>
<td>Abnormal MRI/neck</td>
</tr>
<tr>
<td>BAEP abnormal/total</td>
</tr>
<tr>
<td>ND</td>
</tr>
<tr>
<td>0/14</td>
</tr>
<tr>
<td>0/31</td>
</tr>
</tbody>
</table>

1 Only mild myopathy, 1 small non-specific frontal white matter signal.

ND = no data.
Malaria myositis.

M Swash and M S Schwartz

_J Neurol Neurosurg Psychiatry_ 1993 56: 1328
doi: 10.1136/jnnp.56.12.1328

Updated information and services can be found at:
http://jnnp.bmj.com/content/56/12/1328.1.citation

These include:

Email alerting service
Receive free email alerts when new articles cite this article. Sign up in the box at the top right corner of the online article.

Notes

To request permissions go to:
http://group.bmj.com/group/rights-licensing/permissions

To order reprints go to:
http://journals.bmj.com/cgi/reprintform

To subscribe to BMJ go to:
http://group.bmj.com/subscribe/