Comment

Although other drugs could not always be excluded, in most of the cases reported here chloroquine appeared to be responsible for the involuntary movements. In a previous case of involuntary movements attributed to amodiaquine[1] the patient had also taken chloroquine, so either drug may have produced the side effects. Benztropine, which is well recognised in the treatment of Parkinsonian syndromes, was successfully used in this case,[1] although it was not available to us. We would recommend the same treatment for chloroquine-induced involuntary movements. Although chlorpromazine may produce extrapyramidal effects it was successful in controlling involuntary movements in one of our patients, who responded rapidly when the drug was given parenterally.

Normal therapeutic doses of chloroquine may induce involuntary movements whether it is given by mouth or by injection. Furthermore, some of the patients had taken chloroquine before without adverse reactions. In the past five years we have used chloroquine to treat over 25 000 patients with malaria, and these five patients are the only ones to develop this side effect. The incidence is less than 1/5000. It is also notable that all our patients were under the age of 30, which appeared to be the case in those reported previously,[1] though the ages were not always stated.

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Intramuscular iron and local oncogenesis

Seven cases of sarcomas arising in the area of a previous iron injection site have been reported in man.[1-3] We report here what seems to be another such case.

Case report

A 35-year-old White Caucasian woman (para 2+0) was referred to our outpatient clinic with a four-month history of pain and swelling in the left hip. Examination showed a large craggy mass in the left gluteal region. Biopsy of this mass showed a poorly differentiated spindle cell fibrosarcoma. There was no stainable iron present. The patient received radiotherapy to the tumour site.

Further questioning showed that 14 years earlier this patient had received a short course of intramuscular iron dextran (Imferon) after delivery of one of her children. Her haemoglobin concentration had been 9·4 g/dl at the time. She received one injection into the right gluteal muscles, after which she developed a mass in that area. The lesion resolved slowly over three weeks. The other four injections were therefore given into the left gluteal muscles. A few days after the end of this short course of injections the patient developed an itchy purpuric rash on her legs. More detailed information on the dose and frequency of the injections was not available as the case notes had been destroyed. The proprietary name was mentioned only in a discharge letter lodged in her general practitioner's records.

There was no history of other allergies or of any other intramuscular injections having been given to either buttock. The only other injections the patient could remember having received were childhood vaccinations into the deltoid muscle. The patient did not take any regular medication.

Comment

The carcinogenic risks of iron dextran were discussed in an editorial in the *British Medical Journal* in 1960.[4] Extensive animal studies had shown that sarcomas readily arose at the sites of large intramuscular iron injections, and this raised the possibility that sarcomas might arise at the sites of such injections in man. It was thought, however, that oncogenesis was local and dose-dependent,[5 6] and the product was not removed from the market.

The average interval between injection and the appearance of the neoplasm in the other seven cases reported in man was five years (range a few months to 13 years). In our case it was 14 years. Other unusual features, although they have been reported before,[7] were the development of swelling at the injection site and the occurrence of a rash after the iron dextran injections.

Sarcomas arising at the site of intramuscular iron injections are much rarer in man than in animals, possibly because of the different sizes of the muscles. The much larger human gluteal muscle may allow the iron to be dispersed to such an extent that the concentration necessary to trigger the induction mechanism is rarely achieved in humans. There may also be a long induction period in man. For this reason we think that it is important that this and all similar cases should be recorded.

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Guanidine treatment and impaired renal function in the Eaton-Lambert syndrome

The myasthenic-myopathic or Eaton-Lambert syndrome and its electrophysiological characteristics were described in 1956[1] and 1957.[2] The case reported here is unusual in that guanidine, which was essential for maintaining power,[3] seemed to produce renal impairment.

Case report

A 61-year-old White post office engineer weighing 72·6 kg suddenly developed diplopia and severe muscular weakness in March 1969. Examination by one of us (JM) six weeks later showed bilateral ptosis, diplopia, and variable muscle weakness. At worst he could not raise his arms to the horizontal nor rise from the squatting position; at best he could do both, albeit with effort. There was no wasting or fasciculation. Tendon reflexes were sluggish but became brisker after strong contraction of the muscles. Plantar responses were flexor, and sensation and co-ordination were normal. The blood pressure was 150/85 mm Hg with no abnormality in the cardiovascular or other systems. Edrophonium (Tensilon) 10 mg intravenously produced a very slight increase in power, much less than that expected in myasthenia gravis.

Investigation for carcinoma was negative; in particular a chest radiograph was normal and tomography showed no evidence of a mediastinal mass. Plasma urea was 4·1 mmol/l (25 mg/100 ml), and alkaline phosphatase was slightly raised at 121 IU/l (normal 12-65 IU/l). The muscle action potential in abductor digiti minimi in response to stimulation of the ulnar nerve was of low amplitude with a post-tetanic contraction potentiation of two and a half times the baseline value.