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Steroid psychosis after an intra-articular injection

Intra-articular steroid injections are a well recognised treatment for rheumatoid arthritis and osteoarthritis with an inflammatory component. It is also clear that the effects of intra-articular steroid treatment are not confined to the joint injected. Steroid is absorbed, inducing improvement in the indices of general inflammation and a clinical[1] and thermographic improvement in other joints.[2 3] Peak serum steroid levels occur from two to 12 hours after injection, and the drug is completely cleared within three to five days. However, cortisol levels are suppressed by 64–81% at 24 hours after injection, with most patients' values returning to normal by one week; depo-medrone (methylprednisolone acetate) 40 mg is sufficient to induce maximum suppression.[4] Many complications are known to arise after systemic steroid administration, but we are not aware of any reports of an acute psychosis after a single intra-articular steroid injection in a previously normal person with no past psychiatric history.

A 75 year old woman presented with osteoarthritis of her left hip. Past medical history showed mitral stenosis, atrial fibrillation, and ischaemic heart disease. She had no history of psychiatric illness or dementia. Drug treatment included furosemide (frusemide), digoxin, nifedipine, and warfarin, which she omitted for four days before the injection. The hip was injected with 80 mg depomedrone and 10 ml 0.5% marcain under *x* ray control with local anaesthesia. Urograffin contrast was used to ensure correct needle placement. No sedatives were given. As the patient required further treatment with warfarin she was kept in hospital. Thirty six hours after the injection she developed paranoid delusions, visual and auditory hallucinations. There was no evidence of infection, with a normal chest *x* ray and clear midstream urine.

Urea and electrolytes, glucose, a full blood count, and calcium were normal. The patient required sedating with stelazine owing to severe agitation, and the psychosis persisted for three days, then resolved, and the patient was discharged. At a six week follow up a mini-mental state examination was performed, which showed no underlying abnormality.

A previous case has been reported[5] of a 41 year old patient with rheumatoid arthritis who became elated, disorientated, and emotionally labile after intra-articular injections of 40 mg methylprednisolone into both shoulders, but this patient had already developed an acute organic confusional state after being treated with prednisolone 2.5 mg three times a day for 12 days only two weeks previously.

In a multicentre prospective study, psychiatric symptoms have been recorded in 1.3% of subjects receiving less than 40 mg/d prednisolone, in 4.6% of those receiving 41–80 mg/d, and in 18.4% of those receiving more than 80 mg/d.[6] However, a lack of a past psychiatric history does not prevent psychiatric complications as symptoms only occur in 11% with a known psychiatric history.[7] Methylprednisolone (80 mg) injected into an osteoarthritic knee joint has been shown to lead to a mean peak plasma concentration of 169 ng/ml at eight hours after injection.[8] After a 20 mg oral dose of prednisolone a peak plasma concentration at two hours of 220 ng/ml has been shown.[9] As the equivalent dose of methylprednisolone to prednisolone (and prednisone) is 4 to 5 respectively these represent comparable levels.

It may be expected that a more rapid absorption of methylprednisolone would occur in patients with rheumatoid arthritis rather than osteoarthritis owing to the hypertrophied and inflamed synovium. However, the rate or extent of absorption is not significantly different,[8] and therefore patients with rheumatoid arthritis or osteoarthritis are equally likely to have systemic effects. If two joints are injected with 80 mg depo-medrone then the mean maximum serum concentration is almost six times greater than if only one joint is injected.[4]

Intra-articular injections are commonly given to outpatients and inpatients by all grades of medical staff. Many potential problems may arise and it should be recognised that these may be induced by a single intra-articular dose.

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Familial macrophagic myofasciitis

Macrophagic myofasciitis is an emerging entity that was first reported in the *Lancet* in August 1998.[1] Between May 1993 and 1999 more than 50 cases have been described in France.[2] We report the first familial case of macrophagic myofasciitis.

A 45 year old woman was first admitted to hospital in July 1997 for pain and swelling of the right foot lasting for few months. Muscle biopsy of the right foot showed interstitial fibrosis with marked perivascular lymphocytes infiltrate. Only a few macrophages were seen but not in the subcutaneous tissue, epimysium, perimysium, and perifascicular endomysium. Periodic acid-Schiff (PAS) staining was negative. A diagnosis of focal myositis of the right foot was made[3] and she received colchicine 1 mg daily. In November 1997 she was referred to our institution because her condition had not improved. At that time erythrocyte sedimentation rate (ESR), creatine kinase (CK), aldolase, and autoantibodies were normal or negative. Because focal myositis can evolve to polymyositis,[4] an electromyography was performed showing a diffuse myopathic process. A muscle biopsy was performed in the deltoid but was normal. Myopathic electromyography was imputed to colchicine and this treatment was discontinued. A diagnosis of reflex sympathetic dystrophy of the right foot was proposed and calcitonin injections were started. In April 1998 the foot symptoms completely disappeared. In February 1999, diffuse myalgia appeared. Clinical examination was normal as were the laboratory and the second electromyography findings. However, a third muscle biopsy of the deltoid was performed and showed infiltration of the subcutaneous tissue, epimysium, perimysium, and perifascicular endomysium by sheets of non-epithelioid PAS-positive cells of macrophage lineage typical of macrophagic myofasciitis (fig 1A).

In February 1999 her 11 year old son was referred to our institution for mild chronic myalgias and asthenia of two years' duration. Clinical examination was normal as were laboratory findings (including CK, aldolase, ESR) and electromyography. Nevertheless, in view of the mother's disease and despite the absence of objective signs, a left deltoid muscle biopsy was carried out. The findings were characteristic of macrophagic myofasciitis (fig 1B).

Our two cases of macrophagic myofasciitis underline the pitfalls of this diagnosis, especially the influence of the biopsy site for the demonstration of its typical pathological features. Owing to the rarity of macrophagic myofasciitis, the occurrence of the disease in the mother and her son is unlikely to be coincidental, and therefore probably reflects either a common genetic predisposition or the presence of the causative agent in the environment, or both. Of note is the fact that onset of clinical

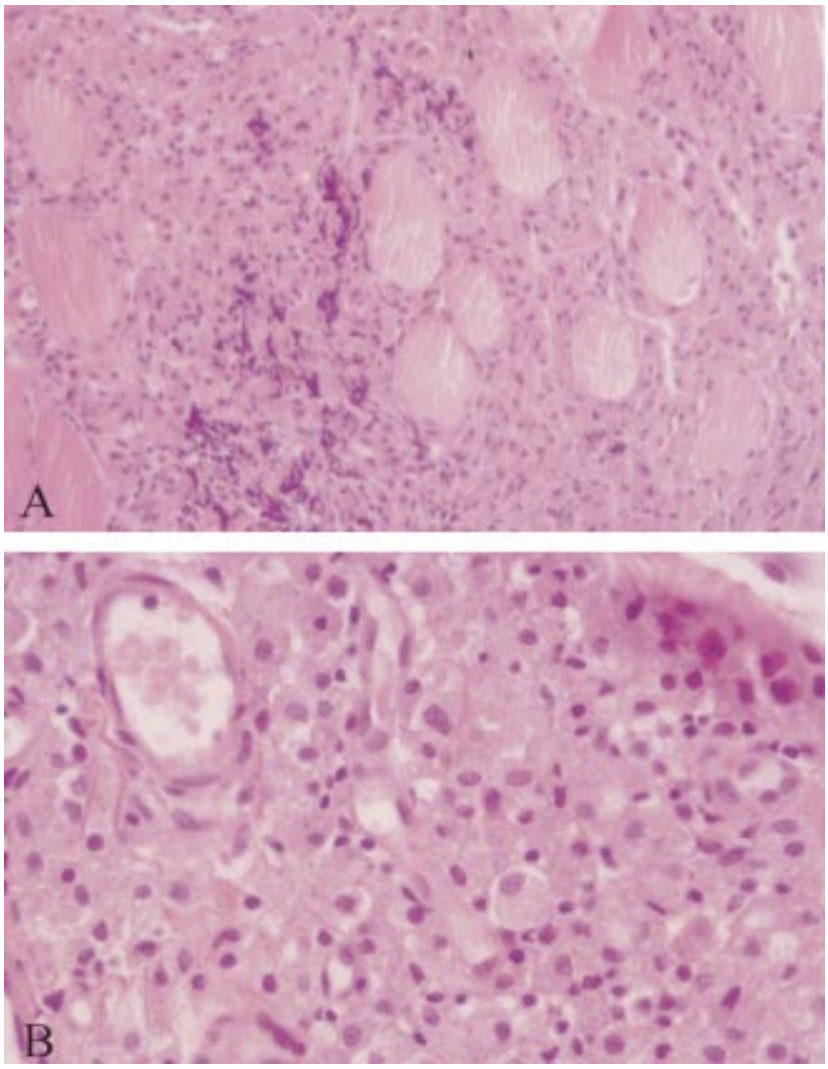

Figure 1 Muscle biopsy on light microscope. (A) Sheet of macrophages with some lymphocytic cells infiltrated from epimysium to perimysium surrounding muscle fibres. (Mother's case, paraffin embedded section, haematoxylin and eosin stain, ×270.) (B) Infiltration of the subcutaneous tissue by densely packed large and grossly rounded cells with clear, slightly basophilic cytoplasm corresponding to macrophagic cells. (Children's case, paraffin embedded section, haematoxylin and eosin stain, ×540.)

manifestations leading to the diagnosis of macrophagic myofasciitis occurred 24 and 18 months after immunisation against hepatitis B virus (HBV) (Genevac B, Pasteurs vaccins, Lyon, France and Engerix B, Smithkline Beecham, Nanterre, France) in mother and son, respectively. Additionally, both patients were vaccinated in the same side as the positive muscle biopsy (that is, the left deltoid) and a prior biopsy of the right deltoid of the mother was negative.

The putative role of the aluminic component of several vaccines—namely, those against HBV, has been recently suggested.[5] If confirmed, this hypothesis should also consider the potential influence of the genetic background, as millions of French people have been recently immunised against HBV, whereas only a few dozen cases of macrophagic myofasciitis have been diagnosed. However, the possible role of currently unidentified environmental factors cannot be ruled out, given that macrophagic myofasciitis occurred concomitantly in our familial case report.

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