unspecific symptoms such as weight loss have been described. Histological changes can comprise muscle fibre loss or atrophy and interstitial immune cell infiltration, thus corroborating our biopsy findings.

Taken together, this case illustrates that a detailed family history is essential in patients having myopathic symptoms and can help to avoid expensive diagnostic tests.

S G Meuth, C Kleinschnitz, M Frank, C Wessig
Klinik für Neurologie, Julius-Maximilians-Universität
Würzburg, Würzburg, Germany

M Bendszus
Abteilung für Neuroradiologie, Julius-Maximilians-Universität Würzburg, Würzburg, Germany

W Kress
Institut für Humangenetik, Julius-Maximilians-Universität Würzburg, Biozentrum am Hubland,
Würzburg, Germany

H Wiendl
Klinik für Neurologie, Julius-Maximilians-Universität
Würzburg, Würzburg, Germany

Correspondence to: H Wiendl, Neurologische Klinik
Universität Würzburg, Josef-Schneider-Straße 11,
D-97080 Würzburg, Germany;
heinz.wiendl@klinik.uni-wuerzburg.de

Myotonic dystrophy type 1 (DM1) is a progressive muscle disease with characteristic neurological symptoms such as distally accentuated paresis and myotonia. Diagnostic hallmarks include a positive family history (autosomal-dominant inheritance), myotonic discharges in the electromyogram and attendant metabolic disturbances.

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Panniculitis and lipoatrophy after subcutaneous injection of interferon β -1b in a patient with multiple sclerosis

Recombinant interferon (IFN) β is currently the most commonly prescribed treatment for long-term immunomodulation in multiple sclerosis, and is generally a well-tolerated treatment. Reactions to IFN β at the injection site are a common side effect, ranging from the frequently observed transient erythema to a more severe cutaneous necrosis in 5% of patients.¹ We describe a patient presenting with panniculitis and subsequent lipoatrophy, secondary to IFN β -1b treatment for multiple sclerosis. To our knowledge, this

case is the first to show this syndrome in relation to IFN β -1b injections.

A 37-year-old man with relapsing–remitting multiple sclerosis presented with a 5-day history of pain in the left thigh, diaphoresis and rigors. This had started 6 days after subcutaneously injecting IFN β -1b (Betaferon, Schering, AG, Berlin, Germany), which he had been using for 27 months, at that site. He did not have arthralgia or myalgia. He had been diagnosed with multiple sclerosis 3 years earlier after an episode of transverse myelitis, and had had four relapses in the interim. Flu-like symptoms and mild reactions at the injection site had developed after initiation of IFN β treatment, but had resolved spontaneously. He also had a diagnosis of migraine, for which he took sumatriptan and diclofenac. There was no history of drug allergies. He did not smoke. He denied previous tuberculosis infection, or a history of connective tissue disorders. On examination, the patient was febrile, and the anteromedial left thigh was swollen, warm, erythematous and tender, with inguinal lymphadenopathy on the same side. Fluctuance was not elicited. Full blood count showed a white cell count of $13.8 \times 10^9/l$, with neutrophilia.

Cellulitis secondary to subcutaneous injection was diagnosed, and he was treated with intravenous flucloxacillin and benzylpenicillin. IFN β was stopped temporarily. An attempt at aspirating the area did not produce any pus, and blood cultures were negative. Ultrasound of the thigh showed an abnormal hypo-echoic area 2.5 cm long and up to 1 cm deep, with some oedema of the surrounding soft tissues. The lesion gradually improved and he was discharged. On review 1 month later, swelling was still evident, although it continued to improve. Interferon was restarted 1 month later.

Four months after the initial episode, the patient had similar symptoms and physical findings, which did not improve with oral antibiotics and a cyclo-oxygenase-2 inhibitor. Again, cellulitis was diagnosed, and IFN β was stopped. A venogram was carried out to rule out an underlying venous thrombosis. The lesion slowly improved after treatment with antibiotics and the patient was discharged. One month later, he started treatment with glatiramer acetate, which he did not inject into the left thigh.

However, the patient presented with pain and swelling of the left thigh on two further occasions, 14 and 19 months after the initial episode. On both occasions, he was treated with intravenous antibiotics. A dermatology opinion was requested, and a skin biopsy was carried out during the second of these episodes. This showed a septal panniculitis, with septal fibrosis and paraseptal lymphohistiocytic inflammation, consistent with erythema nodosum (fig 1). No evidence of vasculitis, neutrophilic infiltrate or histological evidence of deep vein thrombosis was seen on biopsy. The serum angiotensin-converting enzyme (ACE) concentration was moderately raised at 92 U/l (normal range 0–45 U/l). Mantoux test was negative and chest x ray was normal. Serum calcium, urinary calcium excretion, α -1-antitrypsin levels, autoimmune serology, pulmonary function tests and bronchoscopy were also normal. Endobronchial biopsy was normal and did not show any granulomas. We diagnosed panniculitis secondary to IFN β injection. One year after diagnosis, the patient has not had a recurrence; however, the anterior left thigh

Figure 1 Punch biopsy of skin and subcutaneous fat, showing septal fibrosis and paraseptal lymphohistiocytic inflammation.

Figure 2 Lipoatrophy postpanniculitis.

shows marked lipoatrophy (fig 2), which does not affect the other injection sites where he continues to administer glatiramer.

The panniculitides are a group of heterogeneous inflammatory diseases involving the subcutaneous fat, of which erythema nodosum is the most common type. Numerous drugs including sulphonamides, oral contraceptives, phenytoin and salicylates have been associated with panniculitis. Connective tissue diseases such as systemic lupus erythematosus, pancreatic diseases and α -1-antitrypsin deficiency are also associated with panniculitis. Panniculitis may be seen in infections or trauma involving subcutaneous fat. Lipoatrophy is the loss of subcutaneous fat due to a previous inflammatory process involving the subcutis, and it may be the late-stage lesion of several types of panniculitis.²

On literature review, we identified another case of panniculitis after IFN β injections in a patient with multiple sclerosis.³ This patient was taking IFN β -1b for 4 years before the development of panniculitis, with a history of erythematous patches developing at injection sites shortly after the initiation of IFN β treatment. Vasculitis was not seen on dermis and fatty tissue biopsy, and subsequent lipoatrophy was not documented. Although glatiramer acetate has been associated with lipoatrophy, only one recent report describes non-reversible lipoatrophy occurring in a patient with multiple sclerosis during IFN β -1a treatment.⁴

Buttmann *et al*⁵ discuss theories of how IFN β -1a may induce cellular skin infiltration by at least three different pathways: (1) IFN β itself may exert a chemotactic effect on immune cells; (2) IFN β may directly stimulate local adhesion molecule and chemokine expression; and (3) IFN β may indirectly stimulate local adhesion molecule and

chemokine expression by induction of proinflammatory cytokines. This study showed a strong chemokine IP-10/CXCL10 expression associated with extensive perivascular T cell and macrophage infiltrates at the injection sites of IFN β -1a. We conclude that IFN β -1a may therefore trigger inflammatory skin reactions through local chemokine induction followed by rapid immune cell extravasation.

We report the case of a recurrent panniculitis with subsequent lipoatrophy occurring at the injection site of a patient using subcutaneous IFN β -1b for multiple sclerosis. We acknowledge that the panniculitis occurring during IFN β -1b treatment may not solely be due to specific local chemokine induction by IFN β -1b as new episodes were observed months after IFN β -1b treatment had been discontinued. To our knowledge, this case is the first to show this syndrome in relation to IFN β -1b injections. Further studies are required to determine what role, if any, inflammatory chemokines play in the development of this rare side effect of IFN β treatment.

S S O'Sullivan, E M Cronin, B J Sweeney

Department of Neurology, Cork University Hospital, Wilton, Cork, Ireland

J F Bourke

Department of Dermatology, South Infirmary/Victoria Hospital, Old Blackrock Road, Cork, Ireland

J Fitzgibbon

Department of Histopathology, Mercy University Hospital, Grenville Place, Cork, Ireland

Correspondence to: Dr Sean S O'Sullivan, Reta Lila Weston Institute, Institute of Neurology, UCL, 1 Wakefield Street, London WC1N 1PJ, UK; sosulliv@ion.ucl.ac.uk

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Selective brain activity in response to one's own name in the persistent vegetative state

The persistent vegetative state (PVS) is a condition in which awareness of the self and the environment is absent. However, neuroimaging studies suggest that normal brain activity in response to speech and faces can remain in the PVS, although it is not clear if this activity reflects higher levels of perception and cognitive processes, as only meaningless stimuli were used as control conditions.¹ One way to investigate such higher cognitive processes is to examine whether differential activity can be evoked by different contents of language.

We examined brain activity in a patient in the PVS during hearing his own first name compared with another name. Previous studies found activation of the medial prefrontal cortex (MPFC) in healthy subjects during hearing their own name and self referential processing.²

We investigated the case of a 50-year-old man, who had a myocardial infarction with cardiac arrest in March 2003. The diagnosis of PVS was confirmed by two neurologists. The patient showed normal hypothalamic

Figure 1 Brain regions showing higher activity during hearing one's own name than another name in the medial prefrontal cortex (MPFC) ($p < 0.05$, small volume correction (svc)) for the patient with persistent vegetative state (A) and for the three controls (B). Other brain regions showing the same effect ($p < 0.005$, uncorrected) for the patient with persistent vegetative state (C) and for the three controls (D). Activations for the controls are shown on the structural scan from one participant.