that intravenous fluorescein angiography (IVFA) may show more vascular involvement than is clinically obvious [5]. Despite this suggestion we were not able to find a patient who had evidence of involvement on IVFA without fundoscopically evident retinopathy.

The precise incidence of visual loss in dermatomyositis is not known, and in prior reports decreased vision has been attributed to retinopathy and infarction within the retinal nerve fibre layer [6]. Intraretinal haemorrhages and macular exudates have also been described as causing decreased vision in patients with dermatomyositis [6]. The retinopathy and visual loss appear to be reversible in most patients but persistent visual loss has also been noted despite treatment with corticosteroids and other immunosuppressive agents [6].

Optic neuropathy has been described in patients with dermatomyositis, but only in association with retinopathy [2]. We were unable to find a prior report of a patient with dermatomyositis who had optic nerve involvement without retinopathy using the following search terms: dermatomyositis, optic disc, vision, blindness, retinopathy (Pub Med, www.ncbi.nlm.nih.gov June 2003).

The timing of this patient’s visual loss, 1 day after discontinuing prednisone and in association with increasing myalgias and weakness, suggests that reactivation of the dermatomyositis was the cause of her visual loss. The relative afferent pupillary defect, dyschromatopsia, visual field defects and pallid optic disc oedema in the absence of retinopathy are evidence that the visual loss was from an optic neuropathy and not a retinopathy. Fortunately, reinstitution of corticosteroid therapy led to stabilization of her optic neuropathy with resolution of optic disc oedema and stabilization of visual function. She had no history of vascular disease, simultaneously swollen optic nerves and a relatively large cup-to-disc ratio in each eye, all of which would be extremely unusual for another cause of optic neuropathy such as non-arteritic ischemic optic neuropathy [7]. We cannot totally exclude the possibility of subclinical retinopathy and we did not perform IVFA, however there were no cotton wool spots or intraretinal haemorrhages characteristic of ocular involvement in patients with dermatomyositis.

It is not clear why retinopathy did not develop in this patient. Perhaps prior treatment with corticosteroids somehow protected the retinal vascular circulation. Spontaneous regression of retinal neovascularization has been noted in juvenile dermatomyositis, months after the initial fundus findings were documented [8]. The retinopathy in our patient may have been present prior to initial ophthalmic evaluation and disappeared by the time fundoscopy was performed, however her visual loss occurred just 2 weeks prior to neuro-ophthalmic assessment, during which time visual-imparing retinopathy would be expected to persist.

Optic neuropathy may occur in the absence of retinopathy in a patient with dermatomyositis. The absence of retinopathy does not preclude visual loss and should not delay the institution of corticosteroid therapy.

The author has declared no conflicts of interest.

### Key points

**Rheumatology**

Optic neuropathy may occur without retinopathy in dermatomyositis.

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**Letters to the Editor**

Rheumatology 2004;43:393–394
doi:10.1093/rheumatology/keh050

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**Vitamin D deficiency in a patient with systemic lupus erythematosus**

Sir, You would not imagine that the patient illustrated opposite was vitamin D-deficient. However, she is a 21-yr-old Caucasian with systemic lupus erythematosus (SLE). Because of the nature of her condition she has to wear sunblock factor 30 all year round. Her tanned appearance is not derived from UVB exposure but from self-tanning agents.

She was diagnosed with SLE in 1997. Initially she presented with a widespread photosensitive rash. She was found to be positive for antinuclear antibodies and double-stranded DNA antibodies. A skin biopsy was consistent with cutaneous lupus. Photosensitivity tests demonstrated sensitivity to UVB light.

Soon after the diagnosis was established she was admitted to hospital with features of alveolitis, cerebral vasculitis and lupus nephritis. She was treated initially with steroids and intravenous cyclophosphamide. She is now maintained on prednisolone (11 mg once per day) and azathioprine (75 mg once per day).

The patient approached us concerned that she might be vitamin D-deficient because she was avoiding all sunlight exposure. Measurements revealed that she was vitamin D-deficient (25-OH vitamin D3 10.0 ng/ml, normal range 15–60 ng/ml). Other parameters were normal, including parathyroid hormone, alkaline phosphatase, calcium corrected for albumin and 25-OH vitamin D2 (which reflects preceding dietary intake). Her food diary confirmed a normal dietary intake of vitamin D. She was initiated on Calcichew D3 Forte (Shire, Basingstoke, UK) which contains 400 IU of cholecalciferol (vitamin D3). However, 25-OH vitamin D3 levels remained below the normal range despite normal hepatic function. The patient was felt to be compliant. Previous reports have suggested that high-dose steroids can lead to low serum concentrations of vitamin D3 [1]. This might explain the resistance in our patient. However, there is contradictory evidence, especially in patients taking low or moderate doses of prednisolone, such as our patient [2, 3]. It was felt more likely that the use of sunblock would necessitate doses greater than 400 IU, as reported in Moslem women [4]. She was started on alfacalcidol 1 mg in order to increase the dose of vitamin D without increasing the calcium intake. Her renal function tests were normal at this time, though she did have persistent, stable proteinuria (0.25 g/l). Renal dysfunction would not explain her inability to convert vitamin D3 to 25-OH-vitamin

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Rheumatology Vol. 43 No. 3 © British Society for Rheumatology 2004; all rights reserved
Dihydroxy-vitamin D3 has been demonstrated to be reduced in corticosteroid-treated patients with systemic lupus erythematosus. Clin Rheumatol 1995;14:397–400.


Rheumatology 2004;43:394–396
doi:10.1093/rheumatology/keh056

Failure of infliximab treatment and occurrence of erythema nodosum during therapy in two patients with Behçet’s disease

Sir. Here we describe two Behçet’s disease (BD) cases in which treatment with infliximab failed and erythema nodosum (EN) developed during therapy.

References


