erythematous, occurs most commonly in diseases of altered immunity, such as rheumatoid arthritis, and may be due to an effect on regulatory T lymphocyte function.

Evidence in support of T cell mediation as a secondary possible mechanism for the induction of morphoea by penicillamine is illustrated by the cases of two patients who developed morphoea following bone marrow transplantation after initial development of acute and chronic skin changes of graft versus host disease. The latter represents immune attack on host antigens (the epithelial cells in the skin) by donor T lymphocytes. The morphoea appeared to represent the final healing stage of longstanding inflammation.

Acute sensitivity reactions to penicillamine present as local or generalised erythematous, macular, and papular urticarial eruptions. This may account for this patient's initial skin rash, which subsequently progressed to morphoea.

I would like to thank Dr A W McKenzie, consultant dermatologist, Norfolk and Norwich Hospital, for permission to report his patient.

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References

Inflammation of the uveal tract as a presenting feature of temporal arteritis

Sir, Visual symptoms occur in 25 to 50% of patients with temporal arteritis. Of these, loss of vision due to ischaemia of the optic nerve is most well known. We present a case where the presence of marked ocular inflammation caused difficulty in the diagnosis of temporal arteritis.
A 64 year old woman presented with redness in the left eye and pain in the temples of one week's duration. Ophthalmological examination showed injected conjunctiva: a hypopyon on the left side with cells in the anterior chamber. Fundal examination showed peripheral increased white fluffy lesions suggestive of inflammatory choroidal effusions. With a diagnosis of anterior and posterior uveitis treatment was started with topical steroids and mydriatic drugs, to which she had a good ocular response. She continued to have headaches, however. Investigations showed a normal full blood count, erythrocyte sedimentation rate (ESR) 50 mm/h, normal liver function tests, urea, and electrolytes, chest radiograph, and urine analysis. Autoantibodies screen was negative.

Two months later she was referred to the rheumatology clinic with persisting temporal headaches. There were no ocular, polyalgal, or any other systemic symptoms. Her ESR had risen to 77 mm/h and she was exquisitely tender over the left temporal artery. After a temporal artery biopsy treatment was started with steroids and within 12 hours her two month long headaches completely cleared. The ESR dropped to 25 mm/h after four days and was 15 mm/h after four weeks. Her temporal arterial biopsy specimen, however, did not show any signs of arterial inflammation. Her peripheral blood T cell subsets were studied by standard immunofluorescence techniques. The absolute numbers of CD8+ cells in the peripheral blood (normal range 0.5-0.8×10⁹/l) were 0.058×10⁹/l at presentation and 0.07×10⁹/l after four weeks' treatment. The absolute numbers of CD4+ cells were normal on both occasions.

This patient had arterial tenderness, raised ESR, headaches, visual symptoms, and a prompt resolution of clinical features with steroids, thus fulfilling all the diagnostic criteria for giant cell arteritis. Uveitis is much less common in giant cell arteritis, they usually result from arteritis of the posterior ciliary or the ophthalmic artery. Uveitis is much less common and to our knowledge this is the first report of acute anterior and posterior uveitis occurring as a presenting feature of temporal arteritis. The only other case reported with a similar association is that of subacute uveitic glaucoma, in which giant cell arteritis was shown by temporal artery biopsy, though the patient did not have any symptoms of temporal arteritis. As uveitis itself does not cause any changes in peripheral T cell subsets the profound CD8+ lymphopenia must reflect the disease activity of giant cell arteritis. We have previously shown that both giant cell arteritis and polymyalgia rheumatica are characterised by a selective depletion and activation of CD8+ cells in the peripheral blood. This case report further substantiates our observations.

In conclusion we have described a case in which acute uveitis occurred as a presenting feature and delayed the diagnosis of giant cell arteritis. The mechanism of this association is unknown at present.

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Effects of evening primrose oil in rheumatoid arthritis

Sir, The trial of Naudicelle evening primrose oil (EPO) in rheumatoid arthritis (RA) reported by Jäntti et al showed no benefit for Naudicelle EPO in RA compared with olive oil used as a placebo. Unfortunately, the design of the trial rules it out as a valid test of the hypothesis that γ-linolenic acid as EPO is a useful new approach to treatment of RA.

The effects of EPO in RA are slow in onset. In a double blind, placebo controlled trial of Efamol EPO (also known as Epogam), published earlier in this journal, the beneficial effects were shown to take place over a period of 12 months. The trial was conducted in this unusually long period of time because preliminary open investigations had shown that the beneficial effects of EPO are not usually detectable in less than three to four months. The trial of Naudicelle was therefore stopped just before the time when beneficial effects might have been expected. Moreover, non-steroidal anti-inflammatory drugs (NSAIDs) were stopped just before starting the Naudicelle trial. As the effects of these drugs wear off quickly deterioration in clinical condition might have been expected. A valid conclusion might have been that over 12 weeks olive oil and EPO are as good as non-steroidal anti-inflammatory drugs at controlling symptoms of RA. As with disease modifying agents, long studies are required before the place of EPO or other oils in RA can be determined. Short term studies can offer no evidence one way or the other.

In their discussion Jäntti et al imply that the effects of EPO might not be favourable with regard to cardiovascular disease, based on the small fall in eicosapentaenoic acid and the small rise in arachidonic acid in the patients given EPO. It has been shown clearly, however, that low concentrations of linoleic acid, dihomogammalinolenic acid, arachidonic acid, and eicosapentaenoic acid are all markers of an increased risk of cardiovascular disease. A rise in the concentrations of these fatty acids in the blood should therefore be an indication of protection against cardiovascular risk. In the group receiving EPO the initial concentration for the sum of these four essential fatty acids was 338 mg/l and the final concentration 422 mg/l. In the group receiving olive oil, possibly because they were on