

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, polymyalgic, 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 $0.5-0.8 \times 10^9/l$) were $0.058 \times 10^9/l$ at presentation and $0.07 \times 10^9/l$ 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.² Although visual symptoms are fairly common in giant cell arteritis, they usually result from arteritis of the posterior ciliary or the ophthalmic artery.¹ 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.³ As uveitis itself does not cause any changes in peripheral T cell subsets⁴ 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.⁵ 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.¹ 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.² The trial was conducted for 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, dihomo-gammalinolenic acid, arachidonic acid, and eicosapentaenoic acid are all markers of an increased risk of cardiovascular disease.^{3,4} 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

average 12 years younger than the EPO group, the initial value was more favourable at 371 mg/l. But the rise was much smaller, to only 383 mg/l. On this basis EPO might be considered to have a more favourable protective action than olive oil. Moreover, EPO also significantly lowered the concentration of apolipoprotein B, a risk factor for coronary disease.

The strongest prediction of cardiovascular risk may be provided by the formula: (total cholesterol \times 0.42)—(HDL-cholesterol), where HDL=high density lipoprotein.⁵ If this formula is used EPO and olive oil produced almost the same reductions in risk factors, 23% in the case of EPO and 25% in the case of olive oil.

At the end of the discussion the authors say that polyunsaturated fatty acids decrease HDL-cholesterol. They imply that because EPO is rich in polyunsaturated fatty acids it also will lower the desirable HDL-cholesterol. The paper they quote, however, says nothing whatsoever about EPO. The authors' own findings show a slight rise in HDL-cholesterol in response to EPO. Other studies have shown that EPO either has no effect on HDL-cholesterol^{6,7} or that it significantly increases it.^{8,9} There is therefore no evidence at all that EPO reduces HDL-cholesterol and some evidence that it raises it.

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SIR, We are grateful for having an opportunity to reply to Dr Horrobin's letter about our paper.¹

Dr Horrobin stresses interesting and possibly important aspects. The effects of evening primrose oil (EPO) in rheumatoid arthritis may be slow in onset and first appear later than the observation period (12 weeks) in our study. From an ethical and practical point of view, however, at least in the Finnish rheumatic population, it was not possible to continue the study longer with ineffective treatment. On the other hand, the trial was long enough to cause changes in serum fatty acid composition.

The conclusion presented by Dr Horrobin that EPO and olive oil may be as good as non-steroidal anti-inflammatory drugs at controlling symptoms of rheumatoid arthritis is interesting, but is premature and too speculative.

We agree that the changes caused by EPO in serum fatty acid composition are small, maximally about 23% increase in arachidonic acid and about 42% decrease in eicosapentaenoic acid percentage share. Low concentrations of polyunsaturated fatty acids have been suggested to indicate increased risk for cardiovascular disease in the two studies referred to by Dr Horrobin. Our point of view was, however, to stress the precursor fatty acids balance for thromboxane production.

Dr Horrobin refers at the end of his letter to our discussion on published reports of the total cholesterol and high density lipoprotein (HDL)-cholesterol lowering effect of polyunsaturated fatty acids. Contrary to his interpretation we did not discuss EPO because it had no effect either on total or on HDL-cholesterol.

In summary, our findings showed no marked effects on serum lipid or fatty acid pattern of rheumatic patients treated with either EPO or olive oil in a 12 week trial. The effects of olive oil, if any, should be regarded as theoretically more favourable than those of EPO. On the other hand, it is possible that in other types of trial—for example, with longer treatment, with higher doses, with other variables measured—different results might be obtained, as previous publications show. Still we do not believe that evening primrose oil can be propagated as an effective medicine in rheumatoid arthritis, though some patients may receive subjective benefit from it.

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Reference

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Disease remission in rheumatoid arthritis

SIR, I read with great interest the fine and thought-provoking viewpoint of Drs Scott, Spector, Pullar, and McConkey.¹ We have also been concerned about the efficacy of the indices used by rheumatologists to assess the outcome of patients with rheumatoid arthritis (RA) when