α-interferon for intractable pruritus in polycythaemia vera

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IgG subclass deficiencies (Ambrosino DM, personal communication, Dec 19, 1990). It would be interesting to do such a study in these patients.

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α-interferon for intractable pruritus in polycythaemia vera

SIR,—Pruritus occurs in about 40% of patients with polycythaemia vera.[1] It is often unresponsive to conventional antipruritic treatment and phlebotomy.

Human α-interferon (IFNα) is a potent inhibitor of certain malignant haematopoietic progenitor cells and has been used successfully in a few patients with polycythaemia vera.[2] This disorder is characterised in vitro by two populations of erythroid progenitor cells (burst-forming-units erythrocyte, BFU-E). One population requires, as do normal erythroid progenitors, erythropoietin for its proliferation and differentiation. The second population can form BFU-Es in the absence of erythropoietin.[3] We noted in patients with polycythaemia vera a dose-dependent suppression of in vitro erythroid colony formation in the presence of various concentrations of IFNα. The reduction was especially noticeable in erythropoietin-independent erythroid colony formation. In view of these results we decided to treat such patients with IFNα.

All 5 patients (aged 54–76) investigated were dependent on phlebotomy to maintain their packed cell volume below 45%; 4 had severe itching and the 5th had severe fatigue due to iron deficiency. Antipruritic therapy consisted of cimetidine, ranitidine, acetylsalicylic acid, antihistamines, and cromoglycic acid, but none was effective in relieving symptoms. The patients were treated for 6 months with subcutaneous recombinant α-2b-interferon ('Intron A'; Essex, Amstelveen, Netherlands) thrice weekly (table). The weekly dosage varied between 4·5 and 24 × 10^6 U. Striking relief of pruritus occurred within 6 weeks: 3 patients became symptom-free, and in 1 pruritus almost disappeared. After a long period of abstinence all patients were once again able to bathe. In addition, the need for phlebotomy disappeared in 4 patients. The patient who had extreme fatigue caused by iron deficiency was given iron during IFNα treatment. The mean cell volume increased during treatment from 63 to 79 fl (normal 80–100), and haemoglobin remained stable. Although histamine might have a central role in the aetiology of pruritus in polycythaemia vera[4] we could not find a correlation between the excretion of N-methylhistamine (table) and the response during IFNα treatment, nor with the severity of pruritus.

3 patients had side-effects: 2 had an influenza-like syndrome during the first weeks of treatment; and 1 had persistent fever and fatigue. These data suggest that IFNα is a new option for the treatment of polycythaemia vera, preventing abnormal haemoglobin and resulting in disappearance of pruritus.

LABORATORY INDICES BEFORE AND AFTER TREATMENT

Patient	1	2	3	4	5
*Haemoglobin (mmol/l)**					
Before treatment	9·6	9·2	8·4	7·9	9·4
After treatment†	9·1	8·6	8·3	9·4	8·9
PCV (%)					
Before treatment	31	48	45	40	47
After treatment†	46	45	43	46	45
Platelets (× 10^9/l)					
Before treatment	270	230	215	559	678
After treatment†	197	140	230	188	242
Urinary N-methylhistamine‡ (μmol/mol creatinine)					
Before treatment	123	236	182	232	ND
After treatment†	129	291	115	130	ND

*mmol/l × 1 611 = g/dl †Treatment for 6 mo. ‡Normal = 0–150 ND = not done

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Late treatment of chronic Lyme arthritis

SIR,—Dr Gasser and Dr Dusleag's letter (Nov 10, p 1189) on the successful treatment of late neuroborreliosis with roxithromycin and co-trimoxazole, after penicillin and then ceftriaxone had failed, prompted us to try this drug combination in a similarly unresponsive patient with chronic Lyme arthritis.

A 51-year-old man who had been bitten by ticks several times in previous years, was bitten in May, 1989, after which a rash, which was described as erythema migrans, developed. Arthritis in the metatarsophalangeal joints and knees ensued, and in January, 1990, he was referred to a rheumatologist. He then had synovitis in four joints. His borrelia IgG antibody was positive, while IgM was negative. Oral doxycycline 100 mg twice daily for 21 days had no effect and 1 month later borrelia titres were still IgG positive and IgM negative. In early March the patient was referred to us with arthritis in most metatarsophalangeal joints, ankles, and knees and unchanged borrelia IgG and IgM titres. Borrelia titre in CSF was negative. He was treated with phenoxymethylpenicillin 1600 mg thrice daily plus probenecid 500 mg once daily for 21 days without effect. He complained of morning stiffness lasting 1½ hours. X-rays of the hands and feet showed juxta-articular halisteresis with a single erosion. X-ray of the knees showed erosions on both medial tibia condyls. The arthritis was thought to be either chronic Lyme arthritis or seronegative rheumatoid arthritis and sulfasalazine 1 g twice daily was started in April. When this proved ineffective hydroxychloroquine 250 mg daily was added 2 months later.

In November, 1990, the arthritis was still active and there was synovitis in most metatarsophalangeal joints, in both ankles, knees, and wrists, and in the right shoulder. Borrelia titres were still IgG positive, IgM negative. Treatment was then changed to roxithromycin 300 mg twice daily plus co-trimoxazole (trimethoprim/sulphamethoxazole) 320/1600 mg twice daily for 21 days and the arthritis promptly improved. After treatment the patient was in excellent health with no signs of synovitis. No adverse effects of the treatment were observed.

Combined therapy with roxithromycin and co-trimoxazole may prove effective in chronic Lyme arthritis where conventional antibiotics have failed.

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