first was a *staphylococcus albus* infection which arose in a primary ventriculo-atrial shunt inserted in a 60-year-old patient who had developed hydrocephalus in association with a cystic tumour of the posterior fossa. The second was an *Escherichia coli* infection which occurred after a revision of the upper end of a ventriculoperitoneal shunt in a 2-year-old child who had hydrocephalus caused by a Dandy-Walker cyst. The third was a *Staph albus* infection which occurred after revision of the lower end of a ventriculoperitoneal shunt in a 1-year-old child who had presented with non-communicating hydrocephalus at the age of 3 months. The age range of the patients and the number of shunts carried out were as follows: under 1 year, 34; 1 to 10 years, 64; 10 to 20 years, 43; and over 20 years, 59.

Comment

Recent reports suggest that prophylactic antibiotics may prevent shunt infection.[1 3] Malis[4] reported a series of 1732 consecutive major neurosurgical operations in which intraoperative prophylactic tobramycin, vancomycin, and streptomycin were given and in which no postoperative infections occurred.[4] Prophylactic antibiotics are not used in our unit, since they are thought to be of minor prophylactic value and give rise to resistant bacteria and consequently infections which may prove difficult to treat. We consider that careful surgical technique is the single most important factor in the prevention of shunt infection.[5]

Scrupulous surgical technique includes careful planning of incisions to ensure maximum blood supply to the healing tissues, gentle handling of tissue to avoid tissue necrosis, avoiding the overuse of unabsorbable sutures in the wound, and careful prevention of wound haematomas. No shunt infection occurred after operations performed by the experienced members of the neurosurgical team. Furthermore, two of the infections arose after operations performed by one registrar during the first 10 weeks of training. This registrar has since carried out 57 shunt procedures over 12 months with no postoperative shunt infections. We therefore suggest that scrupulous surgical technique alone should be enough to prevent infection after cerebrospinal fluid shunt operations.

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Remission from polymyositis after total body irradiation

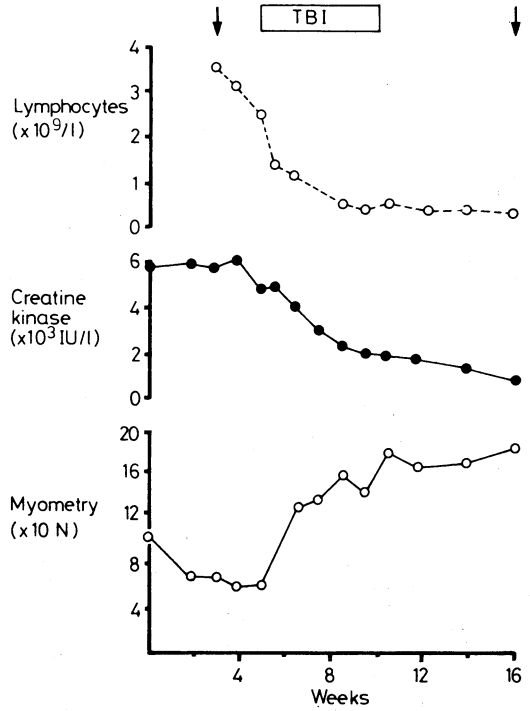
Cellular immune mechanisms have been implicated in the pathogenesis of polymyositis. The modulation of these mechanisms by ionising radiation is clinically effective in the treatment of animal models of autoimmune disease and in some patients with rheumatoid arthritis.[1]

Case report

A 44-year-old man presented in November 1980 with fever, malaise, and proximal muscle weakness. Polymyositis was diagnosed. Activities of all muscle enzymes were greatly increased and creatinine kinase activity was 2175 IU/l. Muscle biopsy and electromyography confirmed the diagnosis. Respiratory function values were normal. Prednisolone, 60 mg/day, and azathioprine 125 mg/day were instituted.

The patient responded well; within three months he had no weakness and muscle enzyme activities were normal. Steroids were progressively reduced while maintaining azathioprine. He relapsed for the first time in May 1981 with severe proximal weakness and a creatine kinase activity of 2350 IU/l. Prednisolone was increased to 60 mg/day but on this occasion the response was poor. After three months he was still symptomatic, though muscle enzyme values had fallen. In September he relapsed for the second time, again with severe proximal weakness and creatine kinase activities at their highest value (>5000 IU/l). A repeat muscle biopsy showed active inflammation. High-dose oral steroids and azathioprine failed to bring about any improvement. Pulse methylprednisolone (1 g intravenously daily for three days) was tried unsuccessfully, with no improvement in muscle enzyme activities after three weeks. His weakness continued to increase and he became unable to lift his head from the pillow.

In an attempt to control his condition, total body irradiation was administered (150 cGy (rads) over five weeks, fractionated as two 15 cGy treatments each week; an 8 MeV linear accelerator was used, delivering a midplane dose rate of 12 to 14 cGy/min). The effect of this treatment was monitored with serial muscle enzyme estimations, lymphocyte counts, and strain-gauge myometry of major muscle groups. Within 10 days of starting treatment his muscle strength had improved and muscle enzyme values began falling. After four weeks he was ambulant, and three months after the course of treatment returned to work. Absolute lymphocyte counts fell rapidly and remained low. A muscle biopsy 10 weeks after total body irradiation showed resolution of the inflammatory process. Six months after treatment the patient showed signs of relapse with increasing weakness and muscle enzyme activities.



Reduction in blood lymphocyte count (normal 1·5-3·5 × 10^9/l) and serum creatine kinase activity (normal < 200 IU/l) after total body irradiation (TBI), with strain-gauge myometric recordings of left quadriceps. Arrows denote muscle biopsies.

Comment

Irradiation depresses peripheral lymphocyte counts and has diverse effects on lymphocyte function. These effects may persist for many years.[2] There is one reported case of a patient with polymyositis treated successfully with total body irradiation after failure of conventional treatment.[3] In our patient the dose of 150 cGy given as total body irradiation produced mild dyspepsia and transient granulocytopenia as the only side effects. In assessing the effect of treatment we employed serial strain-gauge myometric estimations of major muscle groups, a method which in our experience produced reliable and reproducible measurements of muscle strength.[4] These data supported the clinical, biochemical, and histological evidence of improvement in muscle inflammation after total body irradiation. Throughout this period there was no change in the doses of oral immunosuppressive drugs.

Radiation-induced immunosuppression, given as total lymphoid irradiation, has recently been used in the treatment of intractable rheumatoid arthritis. Total body irradiation offers advantages over this method in the ease of administration, the low incidence of side

effects, and the substantially lower dose. The main long-term hazard—carcinogenesis or leukaemogenesis—should be lower, possibly no more than a lifetime fatal cancer risk of 1% after 25 years.[5]

This case provides further evidence that total body irradiation may provide a useful additional form of management in cases of polymyositis resistant to conventional drug treatment, although a lasting effect has not been demonstrated.

We thank Mrs J Coppen, senior physiotherapist, and members of the Jerry Lewis Muscle Research Laboratories, Royal Postgraduate Medical School, London, for their help with this case.

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Effect of nifedipine on histamine reactivity in asthma

The release of mast-cell mediators and the contraction of smooth muscle are associated with influx of calcium ions. Williams *et al*[1] reported that the calcium antagonist nifedipine given sublingually modified histamine-induced bronchoconstriction in asthma. Our observations, however, show that the calcium antagonist verapamil given by inhalation does not modify histamine or methacholine bronchoconstriction in asthma.[2] We report the effect of 20 mg nifedipine and a matched placebo on histamine reactivity in eight patients with allergic asthma.

Patients, methods, and results

We studied eight patients aged between 17 and 33 years with allergic asthma and reversible airflow obstruction. Sodium cromoglycate and bronchodilators were discontinued for at least 24 hours before each test was carried out. Forced expiratory volume in one second was measured with a water-sealed spirometer (Godart Pulmotest) and values corrected for body temperature, pressure, and saturation. Each subject was then given 20 mg nifedipine or a matched placebo sublingually on a double-blind basis. Pulse rate and blood pressure were recorded at five-minute intervals for 30 minutes. Measurement of forced expiratory volume in one second was repeated at 30 minutes and followed by two-minute inhalations of histamine dihydrochloride through a Wright nebuliser in doubling concentrations from 0·025 to 8 g/l at tidal breathing.

After each inhalation forced expiratory volume in one second was recorded at 0·5, one, three, and five minutes and subsequent intervals of two minutes to obtain the lowest value after inhalation. Inhalation of histamine was continued until the forced expiratory volume in one second had fallen by 20% or more. The responses were expressed in terms of the provocative concentration of histamine producing a 20% fall in forced expiratory volume in one second (PC_{20H}) and calculated as described previously.[2] Results of the study are shown in the table. There was no significant difference in the mean baseline forced expiratory volume in one second before and after nifedipine or a matched placebo. Furthermore, the mean percentage changes in forced expiratory volume in one second and the PC_{20H} for histamine were not significantly altered by nifedipine. Pulse rate and blood pressure changes were also not significant.

Comment

In contrast to the results reported by Williams *et al*,[1] nifedipine did not modify histamine reactivity in the patients we studied. We did not observe the significant changes in blood pressure or pulse rate reported by Millar and Struthers[3] in normal subjects with a similar dose of nifedipine. Our observations with verapamil and nifedipine suggest that the effect of calcium antagonists on the bronchial smooth muscle in patients with asthma is minimal. The beneficial effect of verapamil and nifedipine in exercise-induced asthma[4 5] is probably on mast-cell degranulation, which is calcium dependent.

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Effect of nifedipine on histamine-induced fall in forced expiratory volume in one second and PC_{20H} in eight asthmatic patients

		Placebo				Nifedipine			
		Baseline				Baseline			
Case No	Sex/Age	Before treatment	After treatment	% Fall	PC_{20H} (g/l)	Before treatment	After treatment	% Fall	PC_{20H} (g/l)
1	M 18	5·22	5·29	34·2	0·12	5·04	4·97	27·2	0·07
2	F 33	1·87	1·91	34·6	0·03	1·67	1·66	33·7	0·06
3	M 21	4·45	4·55	33·0	0·12	4·59	4·79	36·3	0·88
4	F 22	1·87	1·77	29·9	0·03	1·67	1·74	36·8	0·05
5	M 18	4·55	4·54	32·8	3·05	4·57	4·54	20·7	2·90
6	M 21	2·74	2·78	20·1	0·10	2·38	2·51	21·5	0·05
7	M 32	4·70	4·69	23·7	0·68	4·62	4·82	20·7	1·54
8	M 19	3·02	2·98	32·2	0·06	2·99	3·25	28·0	0·29
Mean ± SEM	23	3·55 ± 0·47	3·56 ± 0·48	30·1 ± 1·88	0·52 ± 0·37	3·54 ± 0·50	3·54 ± 0·50	28·1 ± 2·42	0·73 ± 0·36
p			NS				NS		NS