up. Three other subjects suffered nausea for about one hour after taking cyclosporin A.

After 12 weeks' treatment cyclosporin A was withdrawn, and in all 10 patients the psoriasis relapsed, the time taken for relapse ranging from three days to two months. All side effects disappeared on withdrawal of the drug.

Comment

This study has shown low dose cyclosporin A to be effective in the treatment of severe psoriasis. Conventional treatment had failed to control the disease in the 10 patients studied, and this suggests that cyclosporin is more effective than other drugs that are currently available. Indeed, nine of the 10 patients said that cyclosporin was the most efficacious and tolerable treatment that they had received. Blood cyclosporin concentrations did not correlate with therapeutic response; psoriatic activity at the time of treatment is more likely to dictate individual response.

The effectiveness of cyclosporin in psoriasis supports the hypothesis that psoriasis is a T cell mediated disorder[3] and advances our understanding of this common and often intractable disorder.

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Haemorrhagic cystitis due to gentian violet

Gentian violet has been used extensively in children and adults to treat oral and vaginal candidiasis. Although many regard gentian violet as innocuous, there have been reports of mucosal ulceration after its use.[1] There is, however, no reported instance of damage to the bladder.

We describe a case of severe haemorrhagic cystitis due to accidental injection of gentian violet through the urethra.

Case report

A 32 year old woman presented to her local hospital with a two day history of gross haematuria, preceded by three days of severe pain in the lower abdomen, terminal dysuria, and half hourly urinary frequency both day and night.

An intravenous pyelogram suggested a mass lesion in the left side of the bladder. Cystoscopy showed gross inflammation and oedema of the left side of the bladder with acute ulceration of the overlying mucosa, and a large mass on the left side of the bladder was noted when the patient was examined under anaesthesia.

She was transferred to this department for further study with a provisional diagnosis of tumour of the bladder. On admission she told us that two weeks previously her general practitioner had advised her to inject gentian violet into her vagina (using a plastic syringe) to treat a severe pruritus. She admitted that by mistake she had injected some of the solution through the urethra and within a few seconds had developed burning pain in the lower abdomen, followed by urinary frequency and urgency and dysuria. Two days later she noticed haematuria, which frightened her and led to her admission to the local hospital.

Her condition gradually improved with a high intake of fluids, and a further cystogram showed a normal outline of the bladder with no evidence of the mass lesion seen in the original intravenous pyelogram. Cystoscopy and examination under anaesthesia at this stage showed only slight thickening of the left side of the bladder with fairly intense oedema and inflammation of the left base. Tissue was taken from this area for biopsy. Histological examination showed extensive ulceration and non-specific inflammatory changes with large numbers of eosinophils but no evidence of neoplasia; the urine was sterile.

Comment

The patient produced the gentian violet solution, which was analysed and found to be 1% gentian violet in 2% alcohol.

It is interesting that such violent symptoms developed within less than a minute of injecting the gentian violet into the bladder.

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Seasonal mortality among elderly people with unrestricted home heating

Mortality statistics for England and Wales show hypothermia as causing only about 300 out of the 40 000 excess deaths that occur every winter, with coronary and cerebral thrombosis and respiratory disease causing most of the rest.[1,2] The increase in thrombosis can be explained by haemoconcentration and hypertension after mild exposures to cold.[3] The role of cold houses and other factors in causing this is unknown; Alderson stated that mortality increases in winter among people in institutions in Great Britain, although indoor temperatures and the people's outdoor activities were not specified.[2] This paper describes seasonal mortality among people in well heated houses.

Methods and results

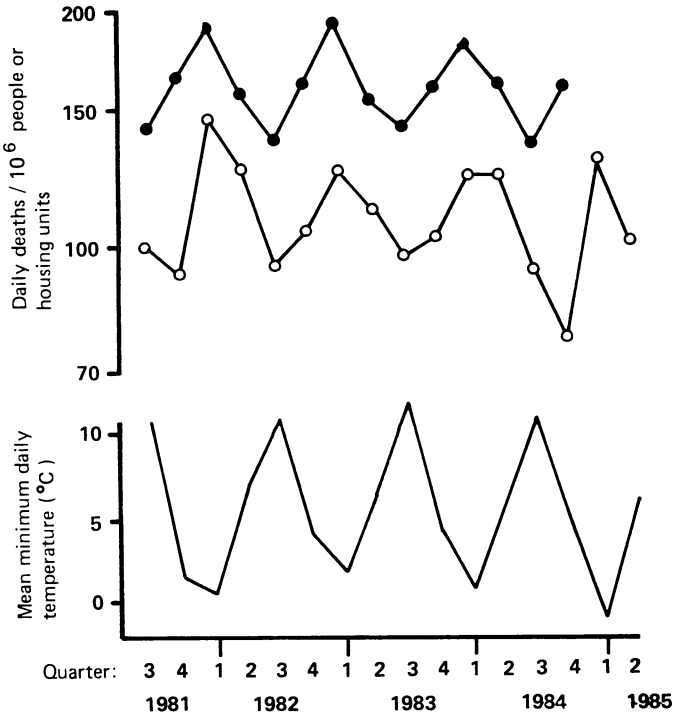
Anchor Housing Association provides centrally heated accommodation, with wardens, throughout England for generally able bodied elderly people, mainly widows. The charge does not vary according to how much heating a resident uses. Termination of a lease owing to death postdated death by up to 14 days; the number of such terminations was available for each three month period from July 1981 to June 1985. The number of housing units increased from 13 624 to 17 765 in this time, with a mean of 1·2 residents per unit. Mortality among the general population up to 1985 was provided by the Office of Population Censuses and Surveys, and minimum daily temperatures by the Meteorological Office. Thermistor probes (Digitron) were used to measure temperatures in 14 residents who volunteered to cooperate at two Anchor homes built in 1971 and 1981. Sublingual readings were taken, with the mouth closed for two minutes.

The figure shows that deaths among residents were 35·3% higher ($p<0·001$, group t test) during the four winter quarters (January-March) than the four summer quarters (July-September), when minimum daily air temperatures were 10·7°C higher. Among the general population aged over 65 mortality rose by 35·8% in the winter quarters before 1985, compared with 36·0% among residents. Mortality lagged behind air temperature[2]; a tendency to larger lags among residents than the general population was attributed to the delays of a few days in recording their deaths. Overall mortality was lower among residents, who were a selected population.

Between 0900 and 1600 on 17 January 1986, after overnight frost, the air temperature outside two homes was 2·3-4·4°C; the sublingual temperature of 14 residents, seven in each home, was (mean (SE)) 36·5 (0·1)°C, the skin temperature on the back of their hands 30·7 (0·4)°C, and the temperature indoors where these 14 readings were made 22·1 (0·3)°C. Each of the residents had a window open and had set all radiators at below maximum. All but one said that they switched off all heating, with windows open, at night. All of the seven who were fit enough made daily excursions outside, walking up to four miles and waiting for buses for up to 40 minutes. All of nine who had moved from accommodation without central heating said that they preferred the warmer accommodation, though some had had initial discomfort from "stuffy nose."

Comment

The continuous high daytime temperatures maintained in these homes did not prevent mortality among the residents from rising in winter by a percentage similar to that among the general population. Extensive outdoor excursions by able bodied residents, and perhaps the residents' preference for open windows and no heating at night, provided their only substantial



Seasonal mortality among people in warm housing (deaths/10^6 housing units containing 1·2 people/unit) and among general population of England and Wales aged 65 and over (deaths/10^6 population) related to mean minimum temperature at London (Kew), Birmingham, and Bradford. ○——○=People in warm housing. ●——●=General population.

exposure to cold. The simplest explanation is that though the quality of life was higher with heated housing, the beneficial effects on mortality of the high indoor temperatures were balanced by the adverse effects of increased exposure to cold outdoors. The results therefore suggest that the traditional tendency of the British to expose themselves to fresh air may be as important as poor heating in causing excess mortality during the winter. They also support previous evidence that cold weather causes death mainly by means other than hypothermia.[4]

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Response of secondary amyloidosis in psoriasis to treatment with etretinate and ultraviolet light

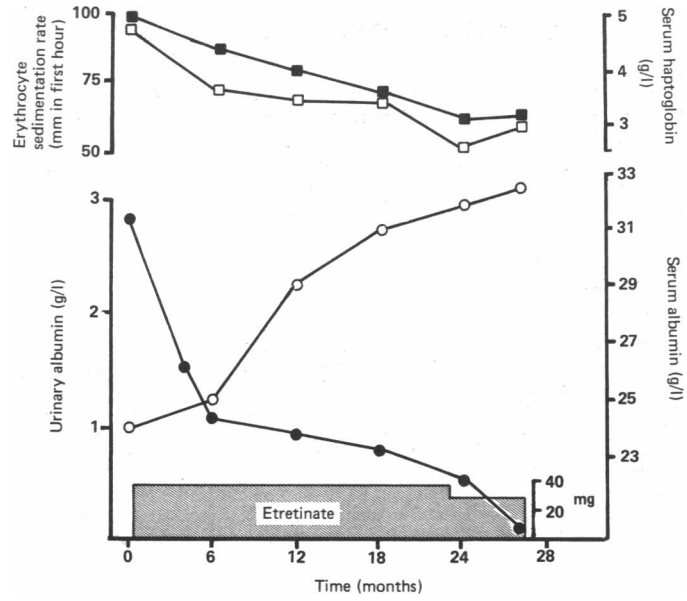
Secondary amyloidosis is a rare but severe complication in psoriasis. Attempts at treatment, with, for example, immunosuppressive agents, have not prevented its progression, and amyloidosis has often been reported as being fatal. We report on a patient with severe psoriasis and destructive arthropathy who developed secondary amyloidosis with the nephrotic syndrome and malabsorption.

Case report

A 23 year old woman developed psoriasis of the plaque type in 1970 and psoriatic arthropathy three years later. She was treated with penicillamine and gold salts without effect, and various non-steroidal anti-inflammatory drugs induced widespread papular dermatitis. Topical steroids and tripsoralens with ultraviolet light A (PUVA baths) were tried, but her psoriasis did not improve and even worsened; for long periods she had erythrodermia. Charnley arthroplasty of the hips took place from 1980 to 1983.

In 1981 proteinuria was detected and secondary amyloidosis diagnosed from biopsy specimens of the rectal submucosa and subcutaneous fat tissue. She had begun to lose weight and her xylose absorption was abnormal. She had a consistently high inflammatory activity; erythrocyte sedimentation rate 70-100 mm in the first hour (normal <15 mm), serum haptoglobin concentration about 5 g/l (normal 0·2-2·0). After secondary amyloidosis had been diagnosed she was treated firstly with methotrexate and then bromocriptine; neither drug cured her psoriasis or proteinuria. Her glomerular filtration rate remained normal.

In June 1983 she still had erythrodermia and also severe synovitis of the large joints, and treatment with etretinate 40 mg daily was started. During the first three months her skin and joint symptoms improved only marginally, so PUVA baths were added. During this regimen her arthritis became inactive, and after nine months her skin was nearly healed. The acute phase reaction also decreased (figure). Her proteinuria (about 3 g albumin day) steadily decreased and was less than 100 mg/day after more than two years of treatment with etretinate. Serum albumin concentrations increased simultaneously from 23 to 33 (normal range 42-55) g/l (figure). After nine months the PUVA treatment was replaced by treatment with ultraviolet light B. Her skin and joint disease remained under good control, her weight returned to normal, and her xylose absorption in December 1985 was almost normal. Amyloid deposits were not detectable. Side effects were sparse, she suffered mild xerostomia and irritation of the conjunctiva.



Serial measurements of erythrocyte sedimentation rate (■), serum haptoglobin (□), urinary albumin (●), and serum albumin (○) in woman with secondary amyloidosis treated with etretinate.

Comment

Treatment with etretinate resulted in apparent total regression of secondary amyloidosis and its renal and intestinal manifestations in this patient, and we believe that the outcome was not simply spontaneous. Her clinical condition due to amyloidosis had been steadily deteriorating with time, and previous treatments with other drugs had not had any effect. Although the excellent outcome was probably mostly due to etretinate, the combined treatment with PUVA baths and ultraviolet light B possibly contributed to the psoriasis and amyloidosis being brought under control.

The beneficial effect of retinoids in skin and joint manifestations of psoriasis is well documented.[1] The possible mechanisms of their action include their antikeratinising effect, anti-inflammatory activities, and effects on cell differentiation and proliferation.[2-4] Our observation that retinoids also may mobilise amyloid deposits in psoriasis has not to our knowledge been previously reported.

The mechanism behind secondary amyloidosis in psoriasis and other chronic inflammatory conditions is not fully understood, but a longstanding active inflammatory process is always present. For many years this patient suffered from inflammation with a prominent acute phase reaction. During treatment with retinoids her clinical and laboratory inflammatory activity