day, which did not increase his insulin requirements. He was fully orientated and vision had returned to normal. The results of investigations on discharge were as follows: plasma sodium 139 mmol/l; plasma potassium 3·6 mmol/l; blood urea 3·8 mmol/l (23 mg/100 ml); plasma bicarbonate 35 mmol/l (35 mEq/l); haemoglobin 11·4 g/dl; packed cell volume 35·7%; and white blood cell count 9·8 x 10^9/l (9800/mm^3).

Discussion

The clinical picture in this case has the typical features of non-ketotic diabetic precocious. The hyperosmolar syndrome has been associated with chronic cardiovascular and renal disease. Although our patient had severe chronic respiratory disease there is no evidence that this alone was the cause of the glucose intolerance. His glucose tolerance was normal three years before he presented in non-ketotic precocious. He had normal renal function and had not been taking any drugs known to be associated with non-ketotic hyperosmolar coma. Therefore frusemide, which is now a recognised factor in this biochemical abnormality, was probably the responsible agent. It should be noted that the patient's illness was precipitated by a vast increase in dosage of frusemide.

This case emphasizes that the high doses of frusemide now used in cases of "resistant" oedema associated with chronic cardiac, respiratory, and renal disease may precipitate glucose intolerance and non-ketotic diabetic precocious.

Requests for reprints to Dr P R W Tasker.


Long-term treatment with 0·01 % flucloxacillin acetone in children

The use of long-term, full-strength fluorinated steroids in children is deprecated because of well-documented side effects of collagen atrophy and interference with adrenal function.1-4 I thought it worth while, therefore, to study the effect of low concentration flucloxacillin acetone (0·01%) used regularly for 12 months in children suffering from unremittent chronic atopic dermatitis.

Patients, methods, and results

Ten children with mild or moderate atopic dermatitis aged from 6 to 12 years were treated with 0·01% flucloxacillin acetone in fatty acid and propylene glycol (FAPG) for one year. The patients were assessed clinically at one to two and two to three monthly intervals, and urine was collected for estimation of 17-hydroxycorticosteroids (17-OHCS) and 17-ketogenic steroids (17-KGS) as a check on adrenal function. Levels of plasma cortisol and stress tests of the hypothalamic-pituitary axis would have given useful information but multiple venepunctures would have been rather traumatic for the children. Throughout the study flucloxacillin acetone was applied continuously; the minimum quantity used was 15 g/week and the maximum 22·5 g/week.

The skin disease was kept under adequate control in all patients though never completely cleared. There was no evidence of skin atrophy in any case. The table shows that there was very little decrease in the 17-OHCS and 17-KGS values with this treatment. Only the 17-OHCS value at 12 months was significantly different from the baseline value (P < 0·01), and this difference was an increase rather than a decrease.

Variations in 17-OHCS and 17-KGS values. Values are means ± SD

<table>
<thead>
<tr>
<th>Baseline value</th>
<th>Value at four months</th>
<th>Value at four months</th>
<th>Value at four months</th>
<th>Value at four months</th>
</tr>
</thead>
<tbody>
<tr>
<td>17-OHCS (µmol/24 h)</td>
<td>9·0 ± 1·8</td>
<td>8·8 ± 1·4</td>
<td>9·7 ± 0·42</td>
<td>9·4 ± 1·1</td>
</tr>
<tr>
<td>17-KGS (µmol/24 h)</td>
<td>137 ± 21</td>
<td>141 ± 1·5</td>
<td>135 ± 1·0</td>
<td>134 ± 2·7</td>
</tr>
</tbody>
</table>

Conversion: 1 µmol to traditional units—17-OHCS and 17-KGS: 1 µmol/24 h = 0·28 mg/24 h.

Discussion

I do not suggest that topical steroids should exclusively be the treatment of atopic dermatitis for which tar preparations and emollients have an important place. Nevertheless, topical steroids are widely used for treating these patients, and atrophy of the skin collagen and suppression of hypothalamic-pituitary-adrenal axis can be a problem.3 This report suggests that, if it was necessary, maintenance treatment with low concentration flucloxacillin acetone (0·01%, in FAPG base) is suitable for children with mild to moderate atopic dermatitis.

5 Munro, D D, and Wilson, L, British Medical Journal, 1975, 3, 626.

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Improvement in paraplegia in vertebral Paget's disease treated with calcitonin

Calcitonin is now accepted as an effective treatment for the bone pain of Paget's disease. We report a case of vertebral Paget's disease with spinal cord compression in which the neurological condition improved dramatically during treatment with porcine calcitonin.

Case report

The patient, a 54-year-old man, was first seen in 1972. He had been treated for hypertension for 10 years and was a gouty man, was first seen in 1972. He had been treated for hypertension for 10 years and for 9. Paget's disease had been diagnosed three years previously. For two years he had had pain in the lower lumbar region extending to both buttocks and thighs, which was worse on walking. His legs were also weak and for one year he had felt cold and numb. He had retired from work as a clerk. On examination he was greatly disabled by pain and weakness, could not walk more than 50 yards (46 m), and used two sticks. Frontal bossing was present, gouty tophi were seen on the hands and elbows, the blood pressure was raised, and a right carotid bruit was heard. Neurological examination of the cranial nerves and arms was normal but he had weakness of the legs with increased tone, brisk tendon reflexes, and extensor plantar reflexes. Vibration sense and proprioception were impaired, Romberg's test was positive, but pain and touch were felt normally. X-ray examination showed Paget's disease of the skull, thoracic vertebrae 7, 8, 10, 11, and 12, lumbar vertebrae 3 and 4, the pelvis, and right femur. Myelography showed partial blocks at T7, T12, and L3 at the anterior aspect of the spinal canal opposite the vertebral bodies. Because of the multiple blocks and the patient's opposition to surgery decompression was not performed. He was referred back one year later with a history of six months' severe lumbar pain, unrelied by analgesics, and spasms in the thighs for some months. He was confined to a wheelchair. At this time he had a sensory level at T6, with spastic legs in which all sensation was reduced. Urinary hydroxyproline was 4·7 mmol/24 h (620 mg/24 h) and serum alkaline phosphatase was 3310 IU/L. Biopsy of bone from the iliac crest showed active Paget's disease. Treatment with porcine calcitonin 80 U intramuscularly daily was begun in October 1973. After four weeks pain and the spasms were less and he was walking with two sticks again, but the neurological signs had not changed. After three months' treatment he had little pain but the neurological signs were still present.