thyroid with increased irregular uptake of 4.7% (normal 0.2-2%). Radiographs of the thoracic inlet showed considerable tracheal compression.

We decided to perform a subtotal thyroidectomy after rendering the patient euthyroid with carbimazole 20 mg three times daily. After 18 days T3 and T4 concentrations had fallen slightly (5.8 nmol/l (3.8 ng/ml) and 224 nmol/l (174 ng/ml), respectively); and at her insistence she went home on continued treatment.

One week later she was found unconscious at home and readmitted in grade 1 coma. She had discarded all tablets on leaving hospital. She was jaundiced, apyrexial, with a pulse rate of 160 beats/min and had signs of bronchopneumonia.

Investigations showed: T3 7.3 nmol/l (4.8 ng/ml), T4 >290 nmol/l (225 ng/ml), and a polymorphonuclear leucocytosis of $20.7 \times 10^9/l$. Blood gas tensions and serum and cerebrospinal fluid biochemical values were normal, except for raised alanine aminotransferase (183 U/l (normal <43 U/l)), bilirubin (73 μ mol/l (4.3 mg/100 ml) (normal <23 μ mol/l (1.3 mg/100 ml)), and γ -glutamyltransferase (54 U/l (normal <3.5 U/l)) concentrations and prothrombin time (15.12 s).

Progressive tracheal compression and displacement 10 days before thyroidectomy.

She was treated with practolol, potassium iodide 500 mg three times a day, carbimazole 30 mg three times a day, hydrocortisone 100 mg four times a day, antibiotics, and physiotherapy. While her conscious level improved her respiratory state deteriorated. She became increasingly dyspnoeic with stridor and could not expectorate or swallow her secretions due to a bulbar palsy. Emergency surgery was therefore undertaken. The goitre with its bilateral retrosternal extensions was mobilised with difficulty and a subtotal thyroidectomy performed. The thyroid was oedematous, engorged in a tight retrosternal compartment, and weighed 108 g. Histological examination showed the goitre to be multinodular with areas of hyperplasia and lymphocytic infiltration.

Her postoperative course was stormy, being complicated on the first day by a severe metabolic alkalosis: pH 7.65, PCO_2 5.2 kPa (39 mm Hg), PO_2 6.9 kPa (52 mm Hg), standardised bicarbonate 39 mmol(mEq)/l, base excess +18, plasma sodium 142 mmol(mEq)/l, plasma potassium 3.5 mmol(mEq)/l, urinary sodium 6 mmol/l, urinary potassium 8 mmol/l. She was treated with a glucose, insulin, and potassium infusion. On the second day she suffered a full-thickness, extensive anterior myocardial infarct, which was uncomplicated. Symptomless hypocalcaemia (calcium 1.76 mmol/l (7.0 mg/100 ml), albumin 29.5 g/l) developed on the fifth day and was treated with oral calcium supplements. She was discharged on the 20th day, and when reviewed as an outpatient at nine months had gained 12.7 kg, looked and felt well, and was taking no drugs.

Comment

We considered that this patient suffered from the "apathetic" thyroid crisis described by Montgomery and Welbourn³ and characterised by "profound prostration, weakness, clouding of consciousness, and eventually coma." As the condition often lacks the hyperreflexia and hyperactivity thought to be typical of thyroid crisis, it is often overlooked and carries a high mortality.

The hypocalcaemia in our patient was attributed to temporary

parathyroid ischaemia, as serum calcium concentrations returned to normal after two months without calcium supplement.

The association between jaundice and severe hyperthyroidism was noted by Newark *et al.*¹

The severe postoperative metabolic alkalosis was thought to be due to total body potassium depletion, which was partly explained by diuretic treatment, longstanding self purgation, and corticosteroid treatment.

Reeve⁵ emphasised the dangers of a longstanding retrosternal goitre accompanied by gradual reduction in the tracheal lumen presenting as an insidious respiratory insufficiency or as acute respiratory distress. To our knowledge there have been no recorded cases of tracheal compression complicating thyrotoxic crisis.

¹ Newark, S R, Himathongkam, T, and Shane, J M, *Journal of the American Medical Association*, 1974, **230**, 592.

² Ehrenhaft, J L, and Buchwalter, J A, *Archives of Surgery*, 1955, **71**, 347.

³ Montgomery, D A D, and Welbourn, R B, *Clinical Endocrinology for Surgeons*. London, Edward Arnold, 1963.

⁴ Eriksson, M, *et al*, *New England Journal of Medicine*, 1977, **296**, 263.

⁵ Reeve, T S, *et al*, *Surgery, Gynaecology and Obstetrics*, 1962, **115**, 223.

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Outpatient suite: a new design

A doctor's time at a clinic is often wasted because many procedures connected with consultations, including the patients' undressing and dressing, take place in one room. In 1970 a new articulation of rooms for dermatological use, designed to improve efficiency, was built at the Wrekin Hospital, Telford.

Design and use

The space (55 m²) is divided into four rooms and four changing cubicles (figure). The consulting room (14 m²) has a desk, chairs, washbasin, and cupboards with a worktop. It is smaller than the standard A-size room (16 m²) recommended by the DHSS in pamphlet HN(76)193 for dermatological clinics. On either side is an examination room (5.5 m²), each with two changing cubicles (2.5 m²). Each examination room has a couch, angle-poise lamp, chair, and trolley. Each cubicle contains a seat, mirror, hooks for clothes, and a dressing gown. The outer door leading into the waiting area can be locked from the inside. One examination room can be blacked-out for examinations under ultraviolet light. Next to the suite is the treatment room (20 m²).

One button in the consulting room operates a buzzer and light over the door into the waiting area to summon a patient. A clerk tells patients their place in the waiting order, and presents their notes in that order to the doctor. After telling his story the patient is taken to a changing cubicle. He locks the outer door, undresses to the required extent, and, when ready, presses a button which lights a panel. While the first patient is undressing the second is called into the consulting room for interview. He then goes to another of

Plan of dermatology outpatient consultation suite, Wrekin Hospital.

the cubicles. A third and a fourth patient can then be interviewed. As the panel lights go on the doctor can see who is ready. If interviews are long or patients undress quickly the first two may be ready before the third can be summoned. If interviews are short or patients undress slowly four patients may be interviewed before any are ready. After examination the first patient returns to his changing cubicle. When dressed he lights his panel. Meanwhile the second patient is examined, and so on until all have been examined. While they are dressing, notes and forms may be completed. Those requiring further interview can be called back into the consulting room: others can leave through the outer door of their cubicle. Patients not needing to undress can be seen entirely in the consulting room. Those requiring a biopsy, patch-test, or some other procedure go to the treatment room via the waiting area. They are given a note for the nurse, who prepares the necessary equipment. Soundproofing of rooms and cubicles is adequate, so that privacy of consultation is achieved. Belongings are safe in the locked cubicles during examination, and the indignity of walking about a clinic in communal ill-fitting gowns is avoided.

Improvements in the suite were made when designing the new Shrewsbury district general hospital. The doctor can operate a light in the changing cubicle from his desk to call the patient into the examination room and, later, into the consulting room. There is a door from one examination room to the treatment room so that the doctor and patient need not go via the waiting area. Ceiling fans prevent cubicle stuffiness in one suite and a more open style of cubicle in another, and in each suite the doors on one changing cubicle are wide enough to take a wheelchair.

Comment

Only one clerk is needed to bring patients and their notes to the clinic and a qualified nurse to organise special tests and treatments. At both hospitals the suites have been duplicated, and the same staff serves two doctors. The design of the rooms is best suited for clinics with a high turnover of patients who need to undress. The doctor has to overcome the difficulty of remembering the problems of up to five patients at a time, but with practice the facility is readily acquired and may even be stimulating.

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Steroid-responsive leukaemia

Here we describe a patient with acute myelomonocytic leukaemia in whom steroids given alone induced a remission lasting 15 months.

Case report

A 74-year-old woman presented with weakness, dyspnoea on exertion, and tender lumps on the limbs. Clinical anaemia was present, and there were skin nodules on both thighs and the right arm. She had buccal ulcers, and a Stevens-Johnson reaction developed during treatment with ampicillin. Results of blood and bone-marrow analysis were consistent with acute myelomonoblastic leukaemia (table).

Progressive improvement succeeded treatment with prednisone 45 mg daily, the nodules taking five months to resolve. She led an active life, enjoyed two holidays, and resumed the care of her disabled husband. Blood and bone-marrow profiles were normal after eight months' treatment. The patient died in blastic relapse after a short terminal illness 15 months later, the blood containing 80% monoblasts and the marrow showing 90% blast cells.

Comment

This response of acute myelomonocytic leukaemia to steroids is most unusual and contrasts with the fairly common response in acute lymphatic leukaemia.^{1,2} In acute myelocytic leukaemia combination treatment with cytotoxic agents and a steroid produces a 30% remission rate in adults.¹ It is doubtful whether steroids alone, even in massive dosage, ever benefit adults with acute myelocytic leukaemia.¹ Spontaneous remission is rarely described³ in association with infection and carcinoma, which were not present here.

Results of analyses of blood and bone marrow before and after treatment

	Before treatment	After treatment
Blood		
Haemoglobin (g/dl)	8.8	14.4
Red cell count ($\times 10^9/l$)	2700	4780
White cell count ($\times 10^9/l$)	2	8
Neutrophils ($\times 10^9/l$)	0.86	6.5
Lymphocytes ($\times 10^9/l$)	1	0.72
Monocytes ($\times 10^9/l$)	0.14	0.56
Platelets ($\times 10^9/l$)	130	217
Erythrocyte sedimentation rate (mm in first hour) (Westergren)	113	2
Bone marrow		
Blast cells (%):		
Undifferentiated	10.5	—
Monoblasts	8.0	—
Myeloblasts	11.5	1.5
Neutrophils (%)	9.5	43.0
Monocytes (%)	16.5	1.5

This case shows that steroids given alone are still worth while in cases of myeloid series leukaemia in which, especially if the patient is elderly, intensive and potentially toxic regimens are hazardous. They also have a useful euphoriant effect and help in cases of cytopenia and autoimmune haemolytic anaemia.

¹ Kline, M J, *Medical Clinics of North America*, 1973, **57**, 1203.

² Ezdinli, E Z, *et al*, *Cancer*, 1969, **23**, 900.

³ Wiernick, P H, *National Cancer Institute Monographs*, 1976, **44**, 35.

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Relapsing polychondritis: an autoimmune disease?

The diagnosis of relapsing polychondritis may be suggested by characteristic clinical features and confirmed by the histological appearance of a biopsy specimen from an affected area of cartilage.¹ The cause of the disease is unknown, though the appearance of circulating anticardilage immunoglobulins² and the ability of cartilage antigens to transform lymphocytes from these patients^{3,4} suggest an organ-specific autoimmune aetiology.

We report a case of relapsing polychondritis with antecedent thyroid disease, diabetes mellitus, and vitiligo in which antibodies to human cartilage, thyroglobulin, intrinsic factor, and gastric parietal cells were present.

Case report

A 46-year-old woman developed hypothyroidism in 1971, 21 years after undergoing thyroidectomy for thyrotoxicosis. At the age of 31 she had developed insulin-dependent diabetes mellitus. Seven years later nasal swelling and tenderness occurred. After another seven years a second episode of nasal swelling resulted in destruction of the nasal septum. Both episodes remitted spontaneously. After 15 years of progressive, right-sided conductive deafness stapedectomy was performed at the age of 41. Intubation was difficult on two occasions. Aged 46 years she developed severe pain and inflammation in the third, terminal interphalangeal joint of the left hand, with similar changes in the right first metatarsophalangeal joint. Soon afterwards both pinnae became swollen, red, and extremely tender. Extensive vitiligo was present together with lipoatrophy at sites of insulin injections. Bilateral exophthalmos with slight ophthalmoplegia was present.

Erythrocyte sedimentation rate was 90 mm in the first hour, and there was normochromic anaemia (Hb 10.9 g/dl) and polymorphonuclear leucocytosis (5.8×10^9 - $10.65 \times 10^9/l$ (5800-10 650/mm³)). Serum vitamin B₁₂ and urate concentrations were normal. Tests for antinuclear factor and lupus erythematosus cells and the RA latex test were negative and serum immunoglobulin concentrations normal. IgG antibodies reacting with human fetal cartilage matrix but not with rat cartilage were detected by immunofluorescence (titre 1/4). Reactions with antisera to IgM, IgA, and complement (β1C) were negative. Thyroglobulin haemagglutination titre was 1/320;