100 ml to 18.8 µg/100 ml) at 90 minutes in response to an insulin-induced hypoglycaemia of 2.2 mmol/l (40 mg/100 ml) at 30 minutes, while the growth hormone concentration fell paradoxically from a resting value of 14 mU/l to 0.8 mU/l at 60 minutes. There was loss of diurnal variation in the concentrations of growth hormone and prolactin. Hypothalamus-pituitary-thyroid function and gonadotrophin values were normal. A sleep electroencephalogram (EEG) showed no appreciable decrease in slow-wave (stages 3 and 4) sleep.

He was treated with cyproheptadine 8 mg/day, increasing to 24 mg/day over two months. He immediately became less irritable and emotionally labile, and his sleep pattern improved so that he no longer woke at the slightest sound. There was no increase in height. His adrenal function was unaltered (table), and after six months bilateral adrenalectomy was performed (Professor L P LeQuesne). Two rounded, fleshy adrenal glands were removed, histological examination of which confirmed diffuse hyperplasia of the cortex of both glands.

Results of pituitary and adrenal function tests

Date	Treatment status	Plasma cortisol (nmol/l)		Urinary free cortisol (nmol/24 h)	ACTH (ng/l)	
		9 am	Midnight		9 am	Midnight
12 Jan '76	Baseline	557	380	330	56	50
13 Jan '76	Baseline	460	467	365	27	50
14 Jan '76	Baseline	555	350	386	27	30
27 Feb '76	Cyproheptadine 8 mg/day	365	340	605		
30 April '76	Cyproheptadine 16 mg/day	440	410	405		
11 June '76	Cyproheptadine 24 mg/day	440	410	570		

Conversion: SI to traditional units—Plasma cortisol: 1 nmol/l $\approx$ 0.04 µg/100 ml. Urinary free cortisol: 1 nmol/24 h $\approx$ 0.4 µg/24 h.

Comment

ACTH release is controlled by the hypothalamus, and serotonin may act as a transmitter. In Cushing's disease the underlying fault is in hypothalamic regulation, and other abnormalities of hypothalamic function, such as loss of diurnal variation in growth hormone and prolactin concentrations, abnormal growth hormone responsiveness to hypoglycaemia, and an abnormal sleep EEG, may be found.³

Cyproheptadine is a centrally acting serotonin antagonist that blocks hypoglycaemia-induced cortisol secretion.⁴ Its use in hypothalamo-pituitary-mediated Cushing's disease seems logical and theoretically provides a treatment of Cushing's disease that avoids bilateral adrenalectomy and reduces the risk of Nelson's syndrome. Its use was unsuccessful in our patient. Krieger recently reported⁵ that out of 40 patients with pituitary-dependent Cushing's disease, some 60% were successfully treated with cyproheptadine; she also reported successful treatment of three out of four patients with Nelson's syndrome. One of her patients, however, who had a normal proportion of slow-wave sleep failed to respond to cyproheptadine. This was also the case in our patient. Perhaps the sleep EEG will be useful in predicting which patients will respond to cyproheptadine.

We thank Dr Lesley Rees, of St Bartholomew's Hospital, for measurements of plasma ACTH, and Professor R W Gilliatt, of the National Hospital, Queen Square, for recording the sleep EEG.

Requests for reprints should be addressed to Dr C G D Brook.

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Pruritus as a presentation of myelomatosis

Cutaneous manifestations of plasma cell dyscrasia are uncommon but have been the subject of several reports.^{1,2} We report two patients who presented with generalised pruritus in whom diagnoses of myelomatosis were unequivocally established. To our knowledge, this association is unreported.

Case reports

Case 1—A 74-year-old retired factory inspector presented with a widespread itchy infected rash which was initially thought to be infected eczema with sensitisation. Treatment was started with topical steroids and antihistamines to relieve the itch. Investigation showed no evidence of any systemic disease. During the next eighteen months the rash improved and severe generalised pruritus became the dominant complaint. Physical examination now showed small eczematous patches on both ankles and slight axillary lymphadenopathy. Investigation showed a raised ESR (58 mm in 1 h, Westergren) but otherwise normal peripheral blood values. Total serum proteins were 92 g/l with a globulin concentration of 51 g/l. A paraprotein band in the beta-2 position was shown on electrophoresis and immunoglobulin measurement showed an IgG level of 49 g/l (IgA and IgM levels were both reduced). Bence Jones protein was absent. Bone marrow examination showed a plasmacytosis with pleomorphism and clumps of atypical primitive cells (see figure). Skeletal survey showed moderate osteoporosis, but no evidence of osteolytic lesions.

Bone marrow appearances in case 1 showing plasma cell infiltrate. (H and E $\times$ 560.)

The diagnostic criteria for myelomatosis were therefore fulfilled. Treatment was initiated with intermittent melphelan (0.25 mg/kg/day) and prednisolone (60 mg/day) for four consecutive days at six-week intervals. Pruritus cleared rapidly after chemotherapy but recurred some ten days before the second pulse became due. A similar sequence of events followed this pulse of chemotherapy but subsequently the patient remained free from pruritus. Biochemical evidence of response was also obtained, with the IgG concentration falling to 20 g/l and other immunoglobulins rising to normal. Eighteen months after starting treatment the patient remains in good health without further pruritus.

Case 2—An 87-year-old retired engineer presented with a one-year history of generalised pruritus and occasional night sweats. He had varicose eczema and mild oedema of both legs, areas of lichenification, and generalised excoriation. Investigation showed a raised ESR (110 mm in 1 h, Westergren), a mild normochromic normocytic anaemia (Hb 11 g/dl), and slight thrombocytopenia of $76.0 \times 10^9/l$. Serum globulins were increased at 41 g/l. Electrophoresis showed a paraprotein band and immunoglobulin quantitation showed a raised IgA level of 20.1 g/l. Bence Jones protein was absent and skeletal survey negative. Bone marrow examination showed a definite infiltrate of immature and atypical plasma cells, confirming the diagnosis. The results of all other investigations to establish the presence of an underlying systemic disease were negative. A similar chemotherapeutic regimen of melphelan and prednisolone was started and within four months pruritus had cleared completely. Insufficient time has elapsed to assess fully the response to treatment.

Discussion

Numerous cutaneous manifestations have been reported in association with myelomatosis and other immunoproliferative disorders. Pruritus, which is the outstanding feature of many skin diseases, is also well-recognised in systemic disorders³ and is commonly seen in association with lymphoid neoplasms, myeloproliferative disorders, and other haematological diseases. Pruritus often antedates the correct diagnosis by some time. The present cases illustrate that myelomatosis may present with generalised pruritus, an association that has apparently not been reported. Zelicovici *et al*⁴ reported three cases of generalised pruritus with a paraproteinaemia, but these patients were eventually classified as benign gammopathies. Rajka⁵ suspected myelomatosis in one patient with pruritus but did not substantiate the diagnosis. Pruritus has been reported in myelomatosis but only as a feature of renal disease and not underlying malignancy.¹ Renal hypofunction was not present in either of our patients. The pattern of response after treatment suggests a direct cause and effect relationship. Thus myelomatosis should be considered in the differential diagnosis of generalised pruritus due to internal disease.

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Acute splenic abscess due to *Salmonella chester*

Isolated splenic abscess presenting as an abdominal emergency is an uncommon occurrence. Moreover, in the case we report it was due to a salmonella of unusual serotype.

Case report

A 20-year-old man was admitted to hospital with a three-day history of increasing pain in the left hypochondrium and loin, worse on inspiration and associated with vomiting, constipation, and frequency of micturition. Two days earlier urinary infection had been diagnosed as his urine had been found to contain protein and blood; he had been treated with co-trimoxazole. He had no history of abdominal trauma or travel to the tropics.

He was ill and dehydrated, with a temperature of 37.7°C, pulse 112/min, and blood pressure 125/75 mm Hg. His abdomen was distended and a tender mass was palpable in the left upper quadrant; the bowel sounds were infrequent. A clinical diagnosis of intra-abdominal abscess was made. The results of investigations showed: haemoglobin 13.2 g/dl; leucocytes $12.5 \times 10^9/l$ ($12\,500/mm^3$; 90% neutrophils); ESR 105 mm in first hour; blood urea concentration 9.9 mmol/l (70 mg/100 ml); serum amylase concentration 50 Somogyi units; blood glucose concentration 6.8 mmol/l (120 mg/100 ml); blood culture sterile; x-ray films of the abdomen after intravenous urography confirmed the presence of a mass in the left hypochondrium displacing the left kidney inferiorly. Laparotomy disclosed a large splenic abscess, its wall being necrotic adjacent to the hilum, which contained over two litres of turbid fluid, which was aspirated to facilitate splenectomy. The splenic bed was drained. Postoperatively he was given ampicillin, 500 mg six-hourly for 10 days, and gentamicin, 80 mg eight-hourly for five days.

The spleen weighed 1430 g, and contained a large cyst, whose wall was composed of fibrous tissue with evidence of acute and chronic inflammation. The inflammation had destroyed the lining in many places, but the appear-

ances were those of an infected simple cyst. Culture of the fluid yielded *Salmonella chester*, sensitive to ampicillin, chloramphenicol, and gentamicin. The patient had been shown to have achlorhydria and to be passing *S chester* in his stools. A person involved in the preparation of the patient's food has been found to be a carrier of *S chester*.

Discussion

S chester is an unusual serotype which has been recognised as causing sporadic cases of salmonellosis. *S typhimurium*¹ and *S agona*² have been reported as causing splenic abscess, illustrating the need to consider *Salmonella* spp as a cause of splenic abscess.

Splenic abscess is commonly a metastatic infection, organisms multiplying in a post-traumatic subcapsular haematoma or simple cyst following bacteraemia, perhaps otherwise insignificant. In this case the achlorhydria was probably responsible for his developing bacteraemia. Less frequently, an abscess may arise as a direct extension of a contiguous infection, or as a primary feature in tuberculosis, typhoid, leprosy, melioidosis, and relapsing fever.³

The presentation with signs of infection and an intra-abdominal mass is typical,⁴ but it is difficult to differentiate splenic abscess from subdiaphragmatic, perinephric, and hepatic abscess, owing to the rarity, and the unfamiliar nature of splenic abscess. Careful plain and simple contrast abdominal radiography are the most useful investigations, but, where available, selective coeliac angiography may be more accurate.⁴

The treatment of choice is abdominal splenectomy and drainage, though in very sick patients, in the presence of dense adhesions and massive abscesses, splenotomy and drainage with interval splenectomy have been successful.^{1 4 5}

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Adrenergic crisis due to phaeochromocytoma

A sudden crisis is a recognised complication of poorly controlled hyperthyroidism in patients exposed to infection or surgery.¹ We report a similar situation arising from a phaeochromocytoma.

Case report

A man aged 38 presented with vomiting and pain in the left shoulder and a history of polydipsia and polyuria for one month with weight loss of 6.4 kg. He was very ill. Although not feverish, he was sweating excessively and had a peculiar blue, mottled skin. His pulse rate was 160/min, blood pressure was 190/130 mm Hg, and chest x-ray examination showed consolidation in the right lower lobe and a mass in the left hypochondrium thought to be the spleen. The ECG showed a supraventricular tachycardia. The urine contained glucose 2% and acetone. The plasma glucose was 31.5 mmol/l (568 mg/100 ml) and the actual bicarbonate 18.9 mmol (mEq)/l. He was rapidly rehydrated with physiological saline and given 20 units of insulin intramuscularly immediately, followed by 10 units intramuscularly hourly.

Four hours later his pulse rate had risen to 180/min, the blood pressure was 160/130 mm Hg, the plasma glucose was 33.9 mmol/l (611 mg/100 ml),