abscess fits into this group. Thirdly, true orbital cellulitis or inflammatory infiltration within the space enclosed by the orbital periosteum and orbital septum; case 3 fits into this group. In Scott's series⁴ two of the four patients in this group died and three of them developed cavernous sinus thrombosis.

In our series two diagnostic points emerged. Firstly, the results of nasal-swab culture may help. In all three patients *Staph aureus* was grown from the nose, but conjunctival swabs were initially sterile. Secondly, the x-ray view employed is important. Thus the occipitotemporal sinus x-ray film may not show the ethmoid opacity. In departments where an occipitotemporal view has become standard an occipitofrontal view should be requested specifically.

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Sodium nitroprusside and renovascular hypertension

Sodium nitroprusside is a peripheral vasodilator which has proved effective in hypertensive emergencies, acute myocardial infarction, resistant pulmonary oedema, and also during surgical procedures (including nephrectomy¹). We report its use in severe drug-resistant renovascular hypertension associated with left ventricular failure before and after corrective surgery. We suggest that the great advantage of sodium nitroprusside is the rapid control of blood pressure, which is easily maintained in the face of major changes in requirements for antihypertensive treatment.

Case report

A 21-year-old man was admitted in April 1977 after an incidental blood pressure (BP) recording of 260/160 mm Hg.

The results of routine investigations were normal including renal function tests. Intravenous pyelography and renal arteriography indicated left renal artery stenosis. Control of hypertension proved difficult and so a left endarterectomy with saphenous patch grafting was performed two months later. BP did not fall, however, and hypertension could not be controlled. The patient was transferred to the hypertension unit at Leicester. His BP was 260/156 mm Hg. He was treated with propranolol 480 mg, prazosin 20 mg, and bendrofluazide 5 mg daily. Urea was 6.9 mmol/l (41.6 mg/100 ml), creatinine 158 μ mol/l (1.79 mg/100 ml). Oral antihypertensive treatment was discontinued and BP control attempted with intravenous diazoxide until 48 hours before renal angiography and renal vein sampling for plasma renin activity (PRA). Large doses of diazoxide partly controlled the BP but the dosage could not be increased because of nausea and vomiting. Angiography showed a thrombosed left renal artery at the site of the previous operation. Some renal function remained on the left, maintained presumably by the well-developed collateral circulation. PRA results (ng angiotensin I/ml/h): peripheral vein 131, aortic 136, low inferior vena cava 141, high inferior vena cava 162, right renal vein 220, left renal vein 366 (upper limit of normal for peripheral vein PRA, 4.4 ng angiotensin I/ml/h). These results were consistent with an appreciable hypersecretion of renin by the left kidney though renin secretion on the right was not fully suppressed.² In view of the difficulty in controlling his BP, corrective surgery was performed.

Before operation he developed severe left ventricular failure resistant to frusemide. Sodium nitroprusside treatment was started according to the published protocol³ and BP was easily controlled at an acceptable diastolic level (see figure). Within hours left ventricular function improved, no further diuretic being needed. Operation confirmed thrombosis of the left renal

BP changes before and after left nephrectomy.

artery and because vascular surgery was not technically possible a left nephrectomy was performed. Control of BP during operation and for 48 hours afterwards was maintained with sodium nitroprusside treatment. At the end of this period it was discontinued. Four days after operation peripheral vein PRA was 1.9 ng angiotensin I/ml/h. Despite this, diastolic BP remained within the range 100-120 mm Hg and he required antihypertensive medication on discharge.

Comment

Sodium nitroprusside was of value in our patient in three ways. Firstly, there was rapid, precise, and sustained control of hypertension which had been resistant to other antihypertensive agents, including large doses of intravenous diazoxide. Secondly, severe left ventricular failure responded well, sodium nitroprusside being effective in reducing left ventricular work by decreasing peripheral resistance without increasing venous return and hence stroke volume. Thirdly, BP was controlled easily during surgery and postoperatively. The very short, effective half life of sodium nitroprusside permitted smooth control of blood pressure when substantial changes were occurring in the factors which maintain blood pressure (such as the renin-angiotensin system). We suggest that sodium nitroprusside is the agent of choice in such circumstances.

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Meningitis in Whipple's disease

Neurological features are well recognised in Whipple's disease but clinical meningeal inflammation is rare.¹ We report a further case which was unaccompanied by any other clinical neurological abnormality.

Case report

A 51-year-old Caucasian man was referred with a three-year history of increasing polyarthritides, fever, muscle weakness, and steatorrhoea partially responsive to prednisolone, 15 mg/day. Histologically the jejunal biopsy specimen was characteristic of Whipple's disease showing infiltration with

periodic acid Schiff (PAS)-positive foamy macrophages containing rod-shaped bacilliform bodies on electron microscopy. Similar appearances were seen in a muscle biopsy specimen.² Addition of oxytetracycline, 250 mg four times daily, induced complete symptomatic remission and the steroid dosage was slowly reduced. Eight weeks later a repeat biopsy specimen still showed PAS-positive macrophages but there was evidence of improvement on electron microscopy with replacement of the bacteria-like rods by intracellular membranous material.³ Three months after diagnosis, however, when taking 5 mg of prednisolone daily together with oxytetracycline, weakness and fever recurred, accompanied by severe headache and vomiting. He was drowsy and febrile (39°C) with neck stiffness but no focal neurological signs and normal fundi.

The results of investigations on admission were as follows: leucocyte count $12 \times 10^9/l$ (80% neutrophils); blood culture no growth; throat swab no growth; and cerebrospinal fluid (CSF)—pressure 360 mm, protein 0.9 g/l, microscopy 900 polymorphs/mm³, Gram stain no organisms, and no growth on culture. Countercurrent immunoelectrophoresis (CIE) indicated presence of *Neisseria meningitidis* type-Z antigen. Benzylpenicillin, 1 megaunit six-hourly, was substituted for the oxytetracycline and the prednisolone increased to 30 mg/day. Rapid recovery ensued, with return of CSF to normal. Two weeks later when the dose of prednisolone had been reduced to 10 mg daily meningism recurred, again responding to penicillin and an increased dose of prednisolone. In the next three weeks he had several further episodes of clinical meningitis. Brain scan, electroencephalography, and skull and sinus x-ray investigations were performed but failed to show evidence of any focal intracranial abnormality.

In view of these relapses cephaloridine was substituted for penicillin and metronidazole added to cover the possibility of anaerobic infection but a further recurrence occurred. No evidence of infection was found during any of these episodes and CIE failed to demonstrate further antigen. Cytological examination of CSF, however, showed the typical PAS-positive macrophages of Whipple's disease (figure). In the fifth attack of meningitis no antibiotic was given but the dose of prednisolone was raised and the effect of the increased dose observed. A rapid clinical response occurred within 24 hours with disappearance of the CSF and blood leucocytosis as before.

Prednisolone, 20 mg/day, with reintroduction of oxytetracycline prevented further symptoms until five months later, when another attempt at steroid reduction resulted in meningitis, which again resolved completely on returning the prednisolone dose to 20 mg/day without additional antibiotic.

Course of the disease showing changes in CSF and peripheral blood leucocytes with treatment.

Discussion

Widespread foci of PAS-positive macrophages have been found in the brain and meninges of patients with Whipple's disease at necropsy, even in the absence of clinical neurological abnormality.⁴

Our inability to show organisms in the CSF; the failure of antibiotics to influence the course of the illness; the complete clinical resolution on increased prednisolone dosage alone; and the finding of typical PAS-positive phagocytes in the CSF must strongly support a diagnosis of Whipple's meningitis. Whether this was induced by an

immune response to viable intracerebral Whipple's organisms or to intracellular PAS-positive material alone in the meninges is unknown.

The importance of discovering type-Z meningococcal antigen on one occasion is uncertain and may be a spurious result. It may, however, indicate the presence of an antigen common to type Z meningococcus and to the Whipple's organism, as has been noted with *Shigella*, *Streptococcus*, and *Escherichia coli* antigens.⁵

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Anorexia nervosa and Turner's syndrome

The concurrence of anorexia nervosa and Turner's syndrome (gonadal dysgenesis) was first reported in 1963.¹ Nine cases of XO gonadal dysgenesis and two of XO/XX (mosaic) gonadal dysgenesis have now been reported.² It is debatable whether this association occurs with a greater than chance frequency and, if so, what the relation between these disorders is. We describe the first reported British case of anorexia nervosa in association with Turner's syndrome.

Case report

A 23-year-old postgraduate student had developed a close relationship with her mother after her father died when she was 8. Turner's syndrome was diagnosed when she was a child, karyotyping showing a 45,XO chromosome pattern. She had the typical features of short stature (147 cm), webbing of the neck, low hairline, "shield-like" chest, absent breast development, sparse pubic hair, primary amenorrhoea, a rudimentary uterus, and skeletal abnormalities. At age 18 she began treatment with oestrogens to induce a more feminine appearance and to induce withdrawal bleeding. Two and a half years later the oestrogens were stopped because she developed jaundice.

She was overweight and was frequently teased by children at school about her appearance. At 18 years she weighed 57.5 kg, but in her first year at university she became a vegetarian and began to diet, largely eliminating fats and carbohydrates. Within six months she had lost 15 kg. This was followed by short periods of overeating, for which she had guilt feelings. Dieting continued for four years and she considered the weight loss as desirable and not excessive. On admission to hospital she weighed 35 kg. Self-induced vomiting to avoid weight gain was suspected. Anorexia nervosa was diagnosed according to Feighner's criteria.³

Comment

One theory of anorexia nervosa is that it results from a wish to regress to an asexual prepubertal state to avoid mature biological sexuality.⁴ This seems an unlikely explanation for a girl with Turner's syndrome, especially of the 45,XO variety, who remains largely prepubertal. Although patients with Turner's syndrome may pass through a rudimentary puberty, our patient could not remember either physical or psychological changes, indicating that adolescent sexual conflict was unlikely. But she developed anorexia nervosa while undergoing an artificial puberty in her late teens and stated that she was not enthusiastic about the changes induced by oestrogens. The regression theory might therefore still apply.

Alternatively, an association between Turner's syndrome and anorexia nervosa might result from the low self-esteem associated with the genetically determined physical defects. Our patient was self-conscious about her appearance, and her illness may have been