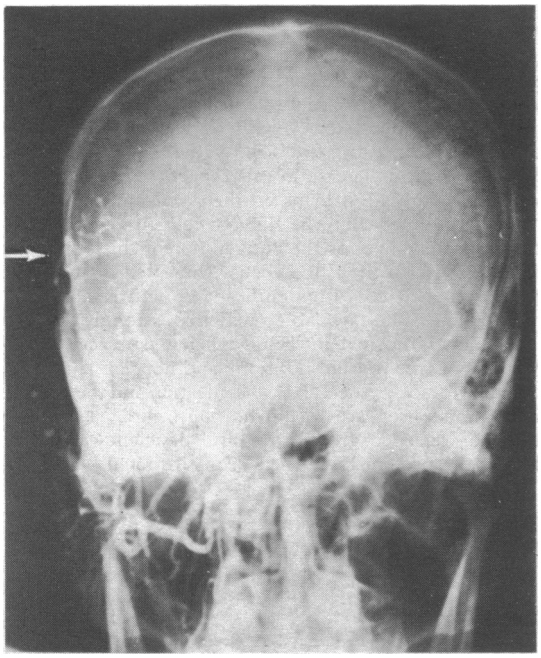
minutes and afterwards made no spontaneous conversation, was uninterested in his surroundings, and refused to feed himself. He was doubly incontinent. On admission to hospital, he was found to be emaciated; he ignored questions or answered them slowly and inaccurately. Lack of co-operation made examination of the central nervous system difficult but no abnormal neurological signs were found. Carotid pulsations were present in the right side of the neck but not on the left.

Plain *x*-ray films showed calcification in the region of both carotid bifurcations. Psychological assessment showed that the patient was functioning at a severely subnormal level, his mental age being between 2 and 6 and his IQ between 20 and 49. Arch aortography showed occlusion of the left common and right internal carotid arteries; the right vertebral artery was patent, but the left one failed to opacify intracranially. The basilar artery and its branches filled from the right vertebral artery, and delayed filling of the anterior and middle cerebral arteries occurred via the posterior communicating arteries.

On 21 January 1977 the right superficial temporal artery was anastomosed to a cortical branch of the middle cerebral artery, as described.[1] Postoperative right carotid arteriography (figure) showed a patent anastomosis through which early filling of the middle cerebral branches occurred. Timed serial films showed that filling through the anastomosis was more rapid and produced better opacification than the reflux filling of the intracranial vessels via the right vertebral artery.



Postoperative right common carotid arteriogram showing excellent filling of the branches of the middle cerebral artery via the patent anastomosis (arrowed) between the superficial temporal artery and a branch of the middle cerebral artery. Outlining of the basilar, both posterior cerebral, and the left middle cerebral arteries has occurred through reflux filling of the right vertebral artery.

Within a week of operation the patient was taking an interest in his surroundings. After two weeks he was orientated and could recall that day's events. By the third week he was actively occupied in the occupational therapy department. He was continent, except occasionally at night. He went home four weeks after operation. Six weeks later he attended the outpatient department smartly dressed and was able to give a good account of himself and his activities. Psychological assessment showed that his IQ was between 85 and 90.

Comment

The calcification seen radiologically indicates that this patient's arterial disease was longstanding. Probably the occlusion of two major arteries supplying blood to his brain had occurred long before the onset of the illness which caused loss of consciousness and dementia. Closure of the third major artery reduced the cerebral perfusion level below the critical level causing ischaemia and failure of function of the frontal lobe.

Donaghy and Yasargil in 1967 showed that it was possible to make an extracranial-intracranial anastomosis and appreciably increase the cerebral blood supply. Several series since have shown[2 4 5] the value of the procedure in patients with transient ischaemic attacks, prolonged reversible ischaemic episodes, and generalised low perfusion states. Chater and Peerless[2] had six patients with dementia and multiple major artery occlusions, one with occlusion of both internal carotid and both vertebral arteries and a clinical picture similar to our patient. The dementia of all six patients improved after extracranial-intracranial anastomosis.

Before extracranial-intracranial anastomoses were performed it was usually assumed that patients with dementia and multiple major artery occlusions inevitably had cerebral infarction, but this is not so. Low perfusion levels may cause ischaemia and cessation of function of neurones. These cells, though inactive, are alive and capable of regaining function after revascularisation of the cerebral hemispheres as is clearly shown in our patient's case.

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Pasteurella multocida meningitis in an infant with recovery

Pasteurella multocida is reported to be the most common infecting organism in patients studied bacteriologically after dog bites.[1] The effects are usually mild, manifesting as acute cellulitis, regional adenopathy, and low-grade fever. The rare complications are septicaemia and meningitis. A review has shown that only 18 cases of *P multocida* meningitis have been reported.[2-5] Of these, five cases have been in infants, of whom two have survived.[4 5] We report the occurrence of septicaemia and meningitis in a 7-week-old infant with recovery.

Case report

A 7-week-old baby boy was admitted for scrotal swelling, "whining," and not feeding well for about one day. He had had no vomiting or fits. He looked ill and irritable, the temperature was 37·5°C, pulse 180/minute, respiratory rate 20/minute, weight 5·5 kg. The anterior fontanelle was full and neck stiffness was present. The left side of the scrotum was warm, red, and tender with oedema of the skin. The testis and epididymis were enlarged and the cord was thickened. The results of the rest of the examination were normal. The cerebrospinal fluid was turbid and contained protein 1·2 mg/l; sugar 1·1 mmol/l (19·8 mg/100 ml); cells $3·0 \times 10^9$/l (3000/mm^3), polymorphs 95%, lymphocytes 3%. A smear of the deposit showed many pleomorphic Gram-negative rods. Blood urea and electrolyte concentrations were normal; the haemoglobin was 11·5 g/dl; WBCs $5·8 \times 10^9$/l (5800/mm^3), neutrophils 50%, lymphocytes 46%, monocytes 4%. From the spinal fluid and blood a Gram-negative coccobacillus was isolated, sensitive to penicillin, ampicillin, gentamicin, chloramphenicol. It was later identified as *P multocida;* Frederiksen biotype 4.

Treatment was begun with gentamicin, 8 mg intravenously every eight hours, and ampicillin, 200 mg intravenously every four hours, for five days. He was also given from the second day thrice daily intrathecal injections of gentamicin, 1 mg, with hydrocortisone, 10 mg. On the fourth day the scrotal skin was less inflamed, but the testis was still enlarged and his fever persisted at 38°C. Aspiration of the scrotum produced 0·5 ml of viscid yellow pus which was sterile on culture. The exact location of the pus was difficult to determine because of the oedema of the skin, but it was most likely present in the tunica vaginalis. On the fifth day, because his fever persisted at 39·3°C and the fontanelle remained full, a search for subdural effusions was made by bilateral subdural tapping but none were found. Treatment was continued with the

gentamicin intramuscularly and ampicillin by mouth. After the 10th day the dose of ampicillin was changed to 250 mg six-hourly, and after the 14th day the dose of gentamicin was changed to 10 mg twice daily. Treatment was discontinued after the 19th day. The baby became afebrile on the 10th day and his subsequent progress was satisfactory. At follow-up eight weeks after the onset of the illness he was thriving and appeared normal.

Comment

P multocida is a common inhabitant of the oral cavities of healthy dogs. Three dogs were frequently present in the baby's home, one belonging to the family and two to neighbours. The saliva of all three was cultured, and from the household pet *P multocida* biotype 4 was isolated. In the absence of any bites or scratch marks, probably the baby acquired the organism from infected saliva after being licked by the dog. The exact nature and mode of development of the scrotal lesion remained undetermined.

Treatment with gentamicin and ampicillin was chosen empirically to give wide cover after Gram-negative rods had been seen in the direct smear of the cerebrospinal fluid. It was continued after the organism was found sensitive to both antibiotics.

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Growth retardation and familial thyroxine-binding globulin deficiency

The association of growth retardation and absence of thyroxine-binding globulin (TBG) was first observed by Nikolai,[1] and similar cases with absent or reduced levels of TBG have subsequently been described. We report a case of growth retardation and delayed bone age in a TBG-deficient patient both of whose siblings were also TBG-deficient, and one of whom also had a retarded bone age.

Case report

An 11-year-old Indian boy was referred for investigation of short stature. He had been born by vaginal delivery at 38 weeks' gestation with a birth weight of 1932 g. Parental heights were: father 156 cm, mother 144 cm. He had had no history of serious childhood illness. He was an alert boy, 122 cm in height (6 cm below the third centile) and 22 kg in weight (3·5 kg below the third centile). No goitre was palpable and he was clinically euthyroid. Examination of the other systems was normal as was his mental development. Radiological examination of the left wrist showed a bone age of 5 years (Greulich and Pyle). A skull radiograph was normal. The results of numerous relevant investigations were normal. In response to insulin-induced hypoglycaemia, peak outputs of 37 μ units of growth hormone per ml and 700 nmol of cortisol per litre (25·4 μg/100 ml) were obtained. The total serum thyroxine concentration was 23 nmol/l (1·79 μg/100 ml), (normal: 60-130 nmol/l (4·7-10 μg/100 ml)); Thyopac-3 was 63% (normal: 92-117%); free thyroxine index was 37 (normal: 61-132); serum triiodothyronine concentration was 1·5 nmol/l (0·97 μg/l) (normal: 1·6-3·6 nmol/l (1·0-2·34 μg/l)) and serum TSH was 2·0 mU/l (normal: 0-6·0 mU/l). Serum TBG measured by the method of Bradwell *et al*[2] was not detectable.

Two brothers, one aged 15 and the other aged 9 years were also studied. Their heights and weights were between the tenth and twenty-fifth centiles, and the elder had a bone age corresponding to chronological age. The younger, however, had a bone age of 5-6 years. Neither had a palpable thyroid and both were clinically euthyroid. Neither however had any detectable TBG in the serum. The results of their thyroid function tests and those of their parents are shown in the table. All members of the family were positive for Xg^a red cell antigen.

Comment

The pattern of transmission of the TBG-deficient state is consistent with sex-linked dominant inheritance. Affected males have complete absence of TBG and hetrozygous females usually have intermediate levels.[3] In some instances, however, the carrier state in females cannot be clearly identified because the TBG value may approximate to the normal range. Such is the case in this report.

Localisation of the TBG locus on the X chromosome was studied using a known X-linked trait, the Xg^a red cell antigen. As all members of the family are positive for the antigen no conclusion can be drawn about its proximity to the TBG locus. Inherited TBG deficiency is not invariably associated with significant growth retardation, as this report illustrates. The available evidence strongly suggests that thyroid function is normal in TBG-deficient subjects, with normal concentrations of free thyroid hormone and normal daily degradation of thyroxine.[4] Moreover, the TSH concentration and the TSH response to TRH administration are within normal limits.[5] The mechanisms responsible for retarded growth in TBG deficiency remain obscure and must for the present be termed "constitutional."

Requests for reprints to J M Barragry.

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Thyroid function studies in patient's family

Study (units and normal range)	Total T4 (nmol/l) (60–130)	Thyopac-3 (%) (92–117)	Free thyroxine index (61–132)	T3 (nmol/l) (1·6–3·6)	TSH (mU/l (0–6)	TBG (mg/l) (6–16)
Father	81	106	76	2·5	1·0	10·7
Mother	71	100	71	3·1	1·0	9·5
Elder brother	17	64	27	1·8	1·6	ND*
Younger brother	23	63	37	1·8	6·0	ND*

*ND = Not detectable—that is, <100 μg/l.
Conversion: SI to traditional units—T4: 1 nmol/l ≈ 0·08 μg/100 ml. T3: 1 nmol/l ≈ 0·65 μg/l.