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.