was 7.0 mmol/l. The following morning she admitted to having drunk a cocktail of alcoholic beverages before the episode.

Case 4. A 12 year old girl was admitted with a three hour history of vomiting followed by loss of consciousness. The possibility of alcohol or drug ingestion was denied. She was drowsy but responded to verbal commands. No alcohol related smell was detected on her breath. Her axillary temperature was 35.1°C. A toxicology screen was negative. Her serum alcohol concentration was 44.5 mmol/l and plasma glucose was 5.7 mmol/l. After the laboratory results were available she admitted to having drunk a 'small gin and tonic' with friends.

Discussion

Our experience shows the importance of considering alcohol intoxication in children with unexplained drowsiness, hypoglycaemia, or hypothermia. The mode of presentation may be confused with other conditions such as head injury or post-ictal state. It was characteristic that parents were often reluctant to admit even the possibility of alcohol ingestion by their children, who in turn rarely volunteered the information. Detection of an alcohol related smell on the breath was an unreliable sign and emphasises the value of blood alcohol measurements. The importance of diagnosis arises from the potentially serious complication of alcohol induced hypoglycaemia³ and the necessity to monitor blood glucose concentrations. In adults the blood alcohol concentration is also a guide to the severity of the intoxication, values over 30 mmol/l being associated with increasing confusion while concentrations

greater than 85 mmol/l may be fatal.⁴ It is uncertain, however, whether these adult blood concentrations have the same clinical importance in children, where age related variations in alcohol susceptibility and metabolic clearance may occur.⁵

In our patients alcoholic beverages were the most commonly identified source of ethanol. The American Academy of Pediatrics Committee on Drugs, however, has recently emphasised the potentially toxic concentrations of ethanol in some paediatric pharmaceutical preparations.⁶ In the United Kingdom alcohol is used similarly in many paediatric preparations—for example paracetamol elixir BP contains 10% v/v and chlorpheniramine elixir BP 6.3% v/v. Although the harmful effect of ethanol in these concentrations remains to be established it could have an additive effect in fasting related hypoglycaemia or cause problems in accidental ingestion if its presence went unrecognised.

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Hypertrichosis due to primary hypothyroidism

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SUMMARY A 10 year old girl with hypertrichosis associated with primary hypothyroidism that resolved after 6 months' replacement treatment with thyroxine is reported. It is important to consider the diagnosis of hypothyroidism in children with abnormal hairiness or distribution of body hair.

Hypertrichosis and hirsutism are common reasons for seeking medical advice, particularly in young women. Only a small proportion of subjects have a diagnosable condition, and treatment is often unsatisfactory, especially in childhood.¹ Hypothyroidism is usually associated with loss of hair.² We report a 10 year old girl whose hypertrichosis was associated with primary hypothyroidism and re-

solved after replacement treatment with thyroxine for six months.

Case report

A 10 year old girl was referred for assessment with a history of poor growth for about three years, with increasingly severe asthma and eczema, lethargy, and growth of body hair over the previous six months. Serum thyroxine concentrations found at the referring hospital were normal (110 nmol/l). Thyroid stimulating hormone and thyroid antibodies were not measured. On examination she had dry skin with patchy excoriated eczema and cold extremities. Hypertrichosis was particularly noticeable over the lateral aspects of the limbs, the back, and the forehead (Fig. 1) and was cosmetically disturbing to her and her family. She had Tanner stage 2 pubic hair with stage 1 breasts and axillary hair³ and normal female external genitalia. Standing height was 130.5 cm (third to 10th centile; two standard deviations below mean parental height) with normal body proportions. Height at entry to school had

been 108 cm (50th centile). Blood pressure was 90/25 mm Hg. Tendon reflexes were normal. Skin-fold thickness at triceps and subscapular sites were 25th and 50th centile, respectively.

Results

Investigations showed normal diurnal cortisol, oestradiol, testosterone, prolactin, 17 α hydroxy progesterone, androstenedione, dehydroepiandrosterone, and sex hormone binding globulin concentrations. The responses of growth hormone to insulin hypoglycaemia and gonadotrophins to luteinising hormone releasing hormone were both normal. Basal thyroxine and thyroid stimulating hormone were <55 nmol/l and 511 mU/l respectively. After treatment with thyrotrophin (200 μ g intravenously) thyroid stimulating hormone concentration rose further (> 640 mU/l). Karyotype was 46,XX. Bone age was 8.3 years at a chronological age of 10.4 years. An x-ray film of the skull suggested rounding of the sella turcica but a computed tomogram of the head was normal. Full

Fig. 1 *Distribution of hair at diagnosis.*

Fig. 2 Distribution of hair after six months' treatment with thyroxine.

blood count showed an eosinophilia ($806 \times 10^9/l$) and radioallergosorbent tests yielded strongly positive results for grass pollen. Abdominal ultrasound showed no adrenal abnormality but cystic enlargement of the left ovary.

After receiving appropriate replacement with thyroxine ($0.1 \text{ mg/m}^2/24 \text{ hours}$) the girl grew at a normal rate with appropriate advance in bone age. Thyroxine and thyroid stimulating hormone concentrations became normal. She lost 4 kg in weight, skinfold thicknesses reached the 10th centile, and performance at school and wellbeing improved dramatically. During six months her hypertrichosis resolved progressively (Fig. 2).

Discussion

It is unusual to find a cause for hypertrichosis or hirsutism and treatment is often unsatisfactory.¹ The causal association between hypothyroidism and hypertrichosis has not been reported in paediatric studies, though congenitally hypothyroid infants often have a low hairline. Perloff reported four cases

of what he called hirsutism in children, with body distribution of hair similar to ours, who responded to replacement treatment (thyroid extract),⁴ contrasting with the case reported by Maekawa *et al* in which an underlying abnormality of keratinisation was thought to lead to hair retention.⁵ The pathophysiology of the relation is unknown. The increased body hair in our patient was not generally in areas sensitive to androgen (hirsutism) despite some pubic hair growth. Clearly, stimulation of the growth of hair by hyperprolactinaemia mediated by thyroid releasing hormone, either directly or via adrenal androgens, was not the mechanism in our patient.

Cystic ovarian enlargement in untreated hypothyroidism is well recognised.⁶ In most cases, as in ours, ovarian size returns to normal with treatment.

It is difficult to explain the normal plasma thyroxine concentration obtained at the referring hospital, particularly in the absence of a simultaneous measurement of thyroid stimulating hormone. In view of the length of the history of impairment of growth, compensated hypothyroid-

ism seems unlikely, and an error in handling or assay is more probable. Measurement of thyroid stimulating hormone might have enabled the diagnosis considerably earlier.

We suggest that full assessment of thyroid function be carried out in all cases of hypertrichosis or abnormal distribution of body hair. If hypothyroidism is noted and treated the loss of body hair may be dramatic and, to the patient, extremely rewarding.

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Congenital hypothyroidism with hereditary, raised thyroxine binding globulin

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SUMMARY A boy with congenital hypothyroidism and hereditary raised thyroxine binding globulin is described. This hitherto unreported combination resulted in under treatment of the thyroid deficiency until serum thyroid stimulating hormone measurement became routinely available. Inadequate L-thyroxine replacement treatment between 2 and 7 years of age caused retarded bone maturation, poor growth velocity, and probably added to his educational difficulties.

Abnormalities in serum thyroxine binding globulin may be inherited or acquired,¹ and result in raised or depressed total thyroid hormone concentrations even in euthyroid subjects. If true thyroid dysfunction occurs in the presence of thyroxine binding globulin derangement, diagnosis and management will be unsatisfactory unless the existence of both abnormalities is recognised. We report a patient with congenital hypothyroidism in association with hereditary, raised thyroxine binding globulin, a combination not previously described.

Case history

A boy (birthweight 3180 g) was born at term in 1970. Perinatal and family medical histories were normal. A clinical diagnosis of hypothyroidism was made at 3 months of age when he presented with an

umbilical hernia, and this was confirmed by a protein bound iodine value of 3 µg/100 ml (normal range 4.0 to 8.4 µg/100 ml). Treatment with oral L-thyroxine (150 µg daily) was begun at 14 weeks of age. He smiled at 3 months, sat unsupported at 8 months, and walked at 20 months. At 22 months, the family moved to a new area, at which time his height was at the 50th centile, three ossification centres had been reported on a wrist radiograph (now unavailable), and the protein bound iodine concentration was 12 µg/100 ml. Apart from his delay in walking, development was considered normal. In view of the high protein bound iodine concentration, thyroxine treatment was gradually reduced. At 2½ years it was stopped completely for five weeks, whereupon the protein bound iodine fell to 1.7 µg/100 ml. L-thyroxine treatment (50 µg orally daily) was begun again, after which the protein bound iodine rose to 15.2 µg/100 ml. Again his thyroxine treatment was reduced to 25 µg daily and he remained on this dose from age 2.8 years until 7.25 years. Over this period his height fell from the 50th centile to the 3rd centile with a correspondingly slow rate of increase. Radiological bone age was reported as 'within normal limits' at chronological age 2 years, and was 6 years (Greulich and Pyle) at a chronological age of 8 years. He entered a normal primary school, but required remedial teaching because of slow progress. Although poor growth caused concern, no alteration was made to the thyroxine dose (25 µg daily) as protein bound iodine