an objective criterion exists for the efficacy of hypolipidaemic agents[7]. Simultaneous regression of atheromatous lesions, particularly coronary, is possible[6].

In the normo-lipidaemic subject, adiposity in the tendo-achilles increases with age, and correlates with serum cholesterol. A direct correlation exists between the intensity of lipid deposition in the tendo-achilles and the coronary arteries, though not with the aorta[6]. Localization of lipid in the tendon has been attributed to ageing, avascularity and wear and tear, and reflects the greater stresses the gastrocnemius experiences compared with the soleus component. Spontaneous rupture at the musculo-tendinous junction corresponds with the area of maximum lipid deposition, although rupture through xanthomatous lesions is rare[9].

In mammals other than man, the paucity of moderate or severe lipid insudation matches the rarity of advanced atherosclerosis. In domestic animals, rupture follows trauma or infection, and usually occurs through the body of the muscle[9].

Xanthomatous lesions can appear on plain X-ray as a sharply defined thickening of the tendo-achilles, with occasional small areas of calcification. Quantifying deposition has been described using a modification of the mammographic technique, echography, xeroradiography, and CT scanning[10-14].

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(Accepted 5 February 1992)

Familial precocious puberty in girls

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Keywords: puberty; precocious; familial; female

Case presented to Section of Paediatrics, 22 November 1991

Familial precocious puberty is a rare condition. It has been more commonly reported in boys, but is very unusual in girls. There are few reports of its occurrence in sisters and to our knowledge only five families have been reported in the English literature since 1922. We wish to report the sixth family of two sisters with familial precocious puberty.

Case reports

Case 1 was referred for evaluation of early puberty at the age of 6.9 years. Puberty started at 4 years, with breast development. When first seen her height was 134.2 cm and weight 36.8 kg. She had breast development stage III and pubic hair stage II with vaginal secretion but no menstruation.

Clinical features and investigations performed at presentation are shown in Table 1. Treatment was started with Cyproterone Acetate. Later a long acting luteinising hormone releasing hormone (LHRH) analogue (buserelin intranasal) was added and the progression of puberty slowed. At 10.5 years she started menstruation.

Case 2, the younger sister of Case 1, started puberty at 5.3 years, with breast development. When evaluated at 5.4 years she was found to have acceleration of growth with breast development and oestrogenization of the vulva. In the next 6-7 months there was rapid growth and progress into puberty and at 6 years she had stage III breast development and pubic hair stage II.

Clinical features and investigations performed at presentation are shown in Table 1. Long acting LHRH analogue, buserelin was started intranasally with no effect. Treatment was changed to subcutaneous goserelin and after two courses, LHRH stimulation test showed excellent suppression of luteinising hormone (LH) but 10 months later when she was 7.1 years of age she had a single menstrual period. Then long acting depot LHRH analogue leuprorelin acetate was started in place of goserelin and this resulted in good suppression of LH in response to LHRH stimulation. She has remained at breast stage III and pubic hair stage II with moderate vulval oestrogenisation and no further menstruation.

Discussion

True precocious puberty is a relatively uncommon condition. Approximate estimates are, four girls each year and one boy every other year in a population of 4.5 million[1]. Female to male ratio is between 4 : 1 and 8 : 1[2]. The incidence of true

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0141-0768/92
080497-02/$02.00/0

Table 1.

	Case 1	*Case 2*
Age at presentation	6.9 years	5.4 years
Height	134.2 cm	118.5 cm
Weight	36.8 kg	26.6 kg
Secondary sexual characteristics	B 111, PH 11, Vulva little mucoid secretion	B 11, PH 1
Bone age (left wrist X-ray)	8 years	8.8 years
Pelvic ultrasound	Uterus adult size, Ovaries both 6 cm^3 containing multiple cysts. No adrenal masses.	Uterus infantile in size and configuration 3.5×0.5 cm Ovaries prepubertal 2 cm^3 with few 1-2 mm follicles
CT scan	Brain and pituitary fossa normal	Brain and pituitary fossa normal
LHRH stimulation test	Pubertal response LH 2.2→52.6 iu/l FSH 6.2→14.1 iu/l	LH 1.9→26.6 iu/l FSH 1.9→8.4 iu/l

*Abbreviations: CT, computerized tomography; LHRH, leutenising hormone releasing hormone; LH, leutenising hormone; FSH, follicle stimulating hormone; B, breast stage; PH, pubic hair stage

familial precocious puberty is even more uncommon, but this may partly be due to under-reporting of cases. Familial early puberty is much more common and there may be some overlap between the two conditions. However, the two cases described here showed pubertal changes at 4 years and 5.3 years, respectively, and therefore cannot be considered as having familial early puberty.

Familial precocious puberty has a marked male predominance. About 25 cases have been reported in males and several of these pedigrees suggest dominant inheritance with father-to-son transmission. In comparison, only four cases of familial precocious puberty in siblings of the opposite sex (brother and sister), and five instances in sisters have been reported in the English literature.

Familial precocious puberty in males seems to be mostly gonadotrophin-independent and is essentially different from the female form of familial precocious puberty, in which there is premature activation of hypothalamic pituitary gonadal axis as shown by increased secretion of LH and follicle stimulating hormone (FSH) and this was the pattern shown in our two cases.

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(Accepted 21 February 1992)

Milk-alkali syndrome due to Caved-S

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Keywords: Caved-S; hypercalcaemia; renal failure

Milk-alkali syndrome is rarely seen nowadays because of the decline in the usage of absorbable alkali in antacid preparations. There is a danger that milk-alkali syndrome may not even be considered, unless the patient is taking large amounts of proprietary medication. This case demonstrates that ingestion of a prescribable antacid in recommended doses together with a high milk intake can result in life-threatening hypercalcaemia and renal failure.

Case report

A 51-year-old man presented with nausea, vomiting, constipation, polyuria and polydipsia. There was a history of intermittent dyspepsia due to a peptic ulcer for which he had been taking Caved-S 6 times daily. He had reddened conjunctivae and there was an early band keratopathy on slit lamp examination.

Table 1. Biochemistry investigations on admission

Serum calcium	3.48 mmol/l
Serum phosphate	1.92 mmol/l
Serum creatinine	703 μmol/l
Serum urea	27.1 mmol/l
Serum magnesium	1.19 mmol/l (NR 0.8-1.1 mmol/l)
Serum 25 OH vitamin D	43 μg/l (NR 4-40 μg/l)
Serum intact PTH	1.5 pmol/l (NR 0.9-5.4 pmol/l)
Urine calcium/creatinine (molar ratio)	0.22

Investigations on admission are shown in Table 1.

The patient was treated with 0.9% NaCl infusion 3 l daily for 5 days. Antacids and milk were withdrawn. The serum calcium returned to normal by 5 days. Serum creatinine fell to 239 μmol/l by 5 days and to 129 μmol/l by 28 days. No other cause for hypercalcaemia or renal failure has been found and he remains well after one year follow-up. Endoscopy showed oedema of the pylorus but no active ulcer. H2 antagonists were recommended only if dyspepsia recurred.

Discussion

Although our patient was taking Caved-S in the recommended dose he was receiving 600 mg aluminium hydroxide,

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0141-0768/92
080498-02/$02.00/0