local insult such as sleeping prone in infancy; careless cutting, which may leave a spicule of nail, which penetrates the nailfold as the nail grows forwards; or compression in shoes. It would be of interest to look for a family history in adults with this condition.

A conservative approach to the condition is preferable in infancy, with simple avulsion of the nail when necessary. The lack of certainty about the prognosis makes destructive surgery of the germinal matrix undesirable.

We thank Mr M N Saad for permission to report case 1. The illustrations were prepared by Mr D Griffin.

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Anterior T wave changes in the ECG of an athlete

Electrocardiographic alterations are well recognised in athletes and may lead to diagnostic difficulties on occasions.¹ Information about the natural history of these changes when the athlete ceases strenuous activity is limited.

Case report

In 1972 a 24-year-old professional footballer who had played in top class competition since the age of 18 was found to have an abnormal ECG on routine examination (figure, A). He had not had a previous ECG. This finding led to a diagnosis of acute myocardial infarction and he was admitted to hospital elsewhere. He was asymptomatic and on examination was thin and muscular with a blood pressure of 110/70 mm Hg and a pulse rate of 65 beats/min; there were no abnormal physical signs. Over 3 weeks' enforced rest resulting from the diagnosis of infarction, the ECG returned to what would be considered normal for a thin, active, young man (figure, B). Cardiac enzyme concentrations and the chest x-ray film were normal and he was afebrile. Three months later he was still inactive and the ECG

The precordial leads from serial electrocardiograms. The limb leads did not alter during this period; the mean frontal QRS vector was $+90^\circ$ and mean frontal T wave vector 0° in all three tracings.

remained normal. He was referred to the Brompton Hospital for further investigation.

Because his livelihood was threatened by the presumptive diagnosis of heart disease (possibly ischaemic), cardiac catheterisation, left ventriculography, and coronary arteriography were performed and all gave normal findings. He then returned to full training and has continued to play competitive football for the last five years. Subsequent yearly ECGs have shown the return of the initial changes (figure, C), which have remained stable. Echocardiography in 1977 showed a left ventricular cavity size of normal dimensions, a normal left ventricular filling pattern, and considerable septal thickening (>2 cm at end diastole).

Comment

Alterations in the electrocardiogram¹ and echocardiogram^{2,3} are common in professional athletes. The electrocardiogram may show increased voltage in both right and left precordial leads as well as T wave inversion and S-T elevation most commonly occurring in leads VI—4. The echocardiogram commonly shows increased left and right ventricular dimensions and septal hypertrophy is often present. Such septal hypertrophy is commonly seen in all forms of left ventricular hypertrophy⁴ and is not characteristic of any particular condition.

This case is presented because the very considerable changes in the ECG of this athlete regressed during only three weeks of enforced inactivity. The initial ECG raised the possibility of organic heart disease and initially the regression of these changes during inactivity was misinterpreted as the evolution of an ischaemic or inflammatory myocardial lesion. Only when the changes reappeared with a return to activity did the nature of the initial changes become clear. The associated septal hypertrophy suggests that the so-called "right ventricular changes" commonly seen in athletes in lead VI—4 may be the result of septal hypertrophy causing repolarisation forces to be directed mainly posteriorly. It is important, particularly in athletes, that changes in the level of physical activity are considered when interpreting the electrocardiogram, and the meaning of serial electrocardiographic changes.

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Hypercalciuria and recurrent urinary stone formation despite successful surgery for primary hyperparathyroidism

Hypercalciuria may persist after surgery for primary hyperparathyroidism despite restoration of the serum calcium concentration to normal. Associated urinary tract stone formation has been described.¹ We describe the first patient with this condition in whom the mechanism of persisting hypercalciuria has been defined.

Case history

A 31-year-old driver presented in 1964 with bilateral radiopaque renal calculi. Hypercalcaemia and hypercalciuria were found. Eventually a benign parathyroid adenoma of mixed cell type was excised in 1966 (Professor G W Taylor). He became normocalcaemic but remained severely hypercalciuric (figure). Dietary calcium intake was about 25–30 mmol daily. Dietary calcium restriction and the addition of oral sodium cellulose phosphate corrected his hypercalciuria when he was under supervision in hospital. He did not adhere to dietary restriction or take medication regularly

Correction of hypercalcaemia by removal of parathyroid adenoma in 1966 with persistence of hypercalcaemia. Note normal calcium excretion during inpatient treatment in 1974. Shaded line represents dietary calcium restriction and sodium cellulose phosphate treatment. Serum albumin concentration ranged from 37-44 g/l.

Conversion: SI to traditional units—Calcium: 1 mmol $\approx$ 40 mg.

when at home. After parathyroidectomy he passed 12 calculi, ureterolithotomy was performed twice, and serial x-ray films showed the development of several new calculi in both kidneys. Recurrent gouty arthropathy began in 1961 but ceased after parathyroidectomy. Throughout, serum uric acid concentration ranged from 0.38 to 0.48 mmol/l (6.4-8.1 mg/100 ml) (upper limit of normal 0.42 mmol/l (7.1 mg/100 ml)) and urinary uric acid from 7.6 to 8.6 mmol/24 h (1.3-1.5 g/24 h) (upper limit of normal 6.0 mmol/24 h (1.0 g/24)). Significant bacteriuria was noted occasionally. Despite this, four analysed calculi contained no uric acid, magnesium, or ammonium, being composed only of calcium oxalate, phosphate, and carbonate. Serum alkaline phosphatase concentration and a radiological skeletal survey were normal. There was no evidence of sarcoidosis, medullary sponge kidney, thyroid disease, myelomatosis, or vitamin D abuse.

METHODS AND RESULTS

Urine calcium was measured with a Perkin-Elmer 30 Atomic Absorption Spectrophotometer.

Parathyroid function—Serum total calcium concentration was measured by a method employing cresolphthalein complexone. Serum ionised calcium concentration (measured by Orion SS 20 calcium electrode) was 1.04 mmol/l (4.2 mg/100 ml) (normal range 0.95-1.12 mmol/l (3.8-4.5 mg/100 ml)). Serum immunoreactive parathyroid hormone (iPTH) concentrations (method of Addison *et al.*²) were 0.8 and 1.0 μ g/l (normal range <1.0 μ g/l). Ionised calcium and iPTH concentrations were measured during 1977 on serum taken without venous stasis and with the patient fasting. Urinary cyclic AMP (measured by competitive protein binding, Amersham) to creatinine (μ mol/g) ratio was 0.245 after an overnight fast and 0.117 after an oral calcium load (see below). Both ratios are normal.³ Serum 25-hydroxycholecalciferol concentration (method of Preece *et al.*⁴) was 8.0 μ g/l (normal range 3.5 to 30.0 μ g/l).

Renal function—Creatinine clearances were 167 and 143 ml/min. ⁵¹Cr-labelled EDTA clearance was 98 ml/min. Tubular function: abnormal glycosuria, proteinuria, and aminoaciduria were absent; plasma potassium, phosphate, and bicarbonate concentrations were normal. Minimum urine pH after oral ammonium chloride (100 mg/kg body weight) was 5.2. Maximum urine osmolality was 871 mosmol/kg.

Effect of calcium restriction and loading—We followed the protocol of C Y Pak *et al.*³ Calcium to creatinine (mg:mg) ratio was measured on a two-hour urine sample after an overnight fast and on a four-hour urine sample after giving 1 g calcium by mouth. The ratio was 0.097 fasting and 0.200 after calcium loading—that is, calcium excretion was normal on fasting and above normal after calcium ingestion.³

Comment

Persisting hypercalcaemia in our patient results from excessive gastrointestinal calcium absorption. Normal serum and ionised calcium and PTH concentrations and normal cyclic AMP excretion exclude hyperparathyroidism. Hypercalcaemia is abolished by calcium restriction and this would not be expected if either excessive resorption of calcium from bone⁵ or a primary renal calcium leak³ was present.

Fasting urinary cyclic AMP excretion is well below all values reported in "renal" hypercalcaemia.³ There is no evidence of kidney damage resulting from previous hypercalcaemia.

The relation, if any, between this "absorptive" hypercalcaemia and previous primary hyperparathyroidism is not clear. Idiopathic "absorptive" hypercalcaemia is not uncommon and chance association is a serious possibility.

Given the chemical composition of the calculi and the severity of the hypercalcaemia, it seems likely that hypercalcaemia is the major cause of recurrent stone formation in our patient. Successful surgery for primary hyperparathyroidism does not invariably ensure correction of a stone-forming tendency. Careful postoperative follow-up is necessary.

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Scombrototoxic fish poisoning

Fish in Britain is generally a safe food; elsewhere poisonous fish are common and dangerous. The occurrence of four cases of scombroid fish poisoning within a short time suggested that the disease may be commoner than is generally recognised. Only one outbreak has been reported in Britain.¹

Scombroid fish are the dark-meated marine fish—mackerel, tuna, bonito, and skipjack. In scombroid poisoning the flesh of the fish is toxic (ichthyosarcotoxic) and neither washing nor cooking appreciably reduces the activity of the toxic principle.²

Case reports

Cases 1 and 2—Mr and Mrs B bought two smoked mackerel. Mr B ate his immediately, but Mrs B decided that hers was too rich (not "off") and after one or two mouthfuls gave the rest to her husband, who finished it. Two hours later Mr B developed severe headache, nausea, and slight diarrhoea and went to bed, where he became hotter and hotter and his scalp began to itch. He felt anxious and noticed that his trunk and arms had turned bright red. Two hours after the onset the redness began to fade and all the symptoms rapidly disappeared. Mrs B suffered only transient slight nausea. Both Mr and Mrs B are healthy, have never experienced these symptoms before, and have eaten many smoked mackerel both before and after this incident.

Cases 3 and 4—Mr and Mrs C bought three fillets of smoked mackerel, which looked, smelt, and felt completely normal. Mr C ate his first and Mrs C hers 20 minutes later. The third fillet was left in the refrigerator. An hour and a quarter after he had finished Mr C began to feel unwell and became conscious of his heart beat, which felt exaggerated and fast. He felt hot and his wife told him he looked red and flushed in the face and hands. A little later he had one episode of diarrhoea. He had neither headache nor nausea. Mrs C became ill about half an hour after her husband, with a moderately severe headache; a feeling of hotness, especially in the face, which flushed bright red; and, a little later, a brief attack of diarrhoea. Both felt slightly itchy. Within three hours their symptoms began to subside and shortly thereafter they felt completely normal. The experience alarmed them sufficiently to contact the local environmental health department. Both had eaten smoked mackerel regularly for several years. Fortunately the third fish was available for examination.

No investigations were carried out on the patients. The fish was divided