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SHORT REPORTS

Hyperphosphataemic rickets in an Asian infant

We have recently shown that hyperphosphataemic rickets is associated with hyporesponsiveness of renal tubules to parathyroid hormone. Patients with this disorder have extremely low basal excretions of phosphate in their urine; urinary phosphate and cyclic adenosine monophosphate increase only marginally after challenge with parathyroid hormone.[1] After treatment with vitamin D plasma phosphate concentration becomes normal and urinary phosphate excretion increases.[2] This hyperphosphataemic variant of rickets has hitherto not been described in an infant. We describe one such infant with severe deficiency of vitamin D. So far as we know this is the first reported case of hyperphosphataemic rickets in an infant.

Case history

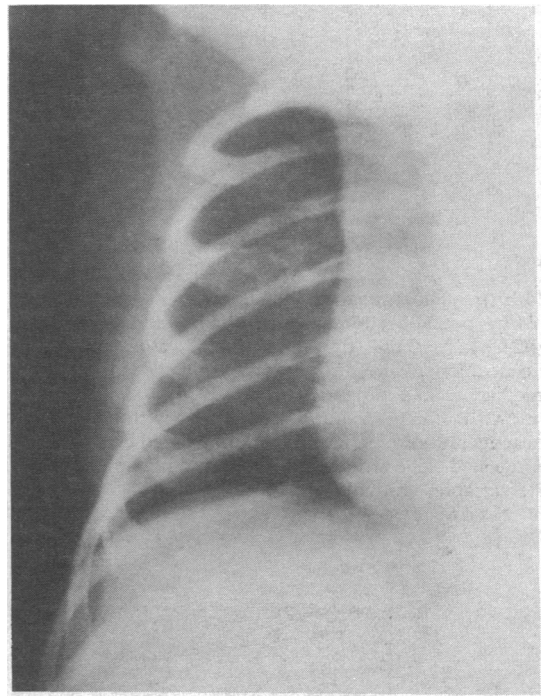
A baby boy was born to healthy unrelated Asian parents after a full term, non-eventful pregnancy. Both parents were vegetarian and the mother had received only folic acid and iron supplements during her pregnancy. The infant was breast fed. At 13 days of age he developed projectile vomiting and shortly after admission to hospital he had a twitching episode lasting four minutes. Results of investigations were: plasma calcium concentration 1·26 mmol/l (5·0 mg/100 ml); phosphate concentration 2·8 mmol/l (8·7 mg/100 ml); alkaline phosphatase activity 361 IU/l; magnesium concentration 0·57 mmol/l (1·4 mg/100 ml); glucose concentration 3·0 mmol/l (54 mg/100 ml). 25-Hydroxyvitamin D was undetectable in serum samples from the baby and the mother. He recovered after intravenous and oral calcium supplements and intramuscular magnesium sulphate and was discharged from hospital six days later taking multivitamin drops (Abidec) 0·6 ml twice daily (800 IU daily).

The patient was readmitted six days later (age 28 days) with a recurrence of twitching. Plasma calcium concentration was 1·56 mmol/l (6·2 mg/100 ml) and phosphate concentration 2·9 mmol/l (9·0 mg/100 ml). Oral calcium supplements and alfacalcidol 1·25 μg twice daily were begun. Three generalised grand mal convulsions then necessitated a further admission (age 40 days). Plasma calcium concentration was 1·76 mmol/l (7·0 mg/100 ml) and alkaline phosphatase activity 268 IU/l. He was treated with oral calcium supplements and phenobarbitone. Electroencephalography, radiography of skull, cranial ultrasound examination, and metabolic and infection screens all gave negative results. The parathyroid hormone concentration on day 52 was raised at 86 pmol/l (8·6 pg/ml). He was discharged from hospital (age 57 days) taking alfacalcidol 1 μg twice daily and multivitamin drops 1·2 ml daily. Plasma calcium concentration rose to 2·05 mmol/l (8·2 mg/100 ml), phosphate was 2·84 mmol/l (8·8 mg/100 ml), and alkaline phosphatase activity was 343 IU/l. Alfacalcidol was discontinued at 108 days of age. At 6 months he was thriving and taking no medication. Calcium concentration was 2·4 mmol/l (9·6 mg/100 ml), phosphate 2·2 mmol/l (6·8 mg/100 ml), alkaline phosphatase activity 360 IU/l, and parathyroid hormone concentration 36 pmol/l (3·6 pg/ml). A chest radiograph on day 40 was reported as normal but on retrospective examination showed severe splaying and rarefaction of the anterior ends of the ribs.

Comment

This infant's biochemical abnormalities were accounted for by severe vitamin D deficiency: the features were low calcium concentration; raised alkaline phosphatase activity; non-detectable 25-hydroxyvitamin D in both the infant and the mother; raised parathyroid hormone concentration; and, finally, response to an antirachitic dose of vitamin D in multivitamin drops, which increased his calcium concentration from 1·26 to 1·76 mmol/l (5·0 to 7·0 mg/100 ml) before the introduction of alfacalcidol. Such a dose of vitamin D is unable to raise the plasma calcium value in patients with hypoparathyroidism and pseudohyperparathyroidism. The only unusual biochemical feature in this patient was the greatly increased plasma phosphate concentration, which initially led to considerable confusion about the diagnosis. It was only in retrospect that we arrived at the diagnosis of "hyperphosphataemic rickets." Review of the chest radiograph at that time showed severe splaying and rarefaction of the anterior ends of the ribs. These changes could have heralded the formation of a rachitic rosary. Hyperphosphataemia in association with rickets has recently been shown by us to be due to severe vitamin D deficiency and a severely diminished end organ (especially renal tubular) response to parathyroid hormone.[1] These patients require large doses of vitamin D, at least initially, since their vitamin D reserves are totally depleted.

These data emphasise that hypovitaminosis D is the commonest cause of hypocalcaemia in Asian neonates.[2 3] Thus even in the presence of the unusual feature of a raised plasma phosphate concentration, vitamin D deficiency must be considered as the primary cause of hypocalcaemia.



Chest *x* ray appearances on day 40 showing splaying and rarefaction of anterior ends of ribs.

The association of neonatal hypocalcaemia with hypovitaminosis D in Asians was described 14 years ago, and it is tragic that we still see pregnancy related complications of hypovitaminosis D: neonatal secondary hyperparathyroidism[4] and maternal pathological fractures.[5]

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Transient urinary tract dilatation associated with hypokalaemia

Pronounced weakness of skeletal or smooth muscle accompanying hypokalaemia of recent onset can result in general paralysis, gastric dilatation, or paralytic ileus.[1] A disturbance of neuromuscular excitation, arising from a change in the ratio of extracellular:intracellular fluid potassium concentrations on which transmembrane electrical potential gradient depends is the cause. We report a case of dilatation of the upper and lower urinary tract, with transitory azotaemia, associated with hypokalaemia, which subsequently resolved when serum potassium concentration returned to normal.

Case report

A 73 year old woman continually suffered bouts of depression and confusion but comprehensive investigation had not provided a reason. No biochemical abnormality had been detected two years earlier. She suddenly lost her appetite for eight weeks and became confused, restless, and weak. Her general practitioner requested serum electrolyte concentrations: sodium (Na^+) concentration was 140 mmol(mEq)/l, potassium (K^+) 2·6 mmol(mEq)/l, chloride (Cl^-) 105 mmol(mEq)/l, bicarbonate (HCO_3^-) 25 mmol(mEq)/l, urea 14·2 mmol/l (86 mg/100 ml), creatinine 196 μmol/l (2·22 mg/100 ml), glucose 6·0 mmol/l (108 mg/100 ml). Encouragement to eat more proved unsuccessful, and she was admitted to hospital eight days later.

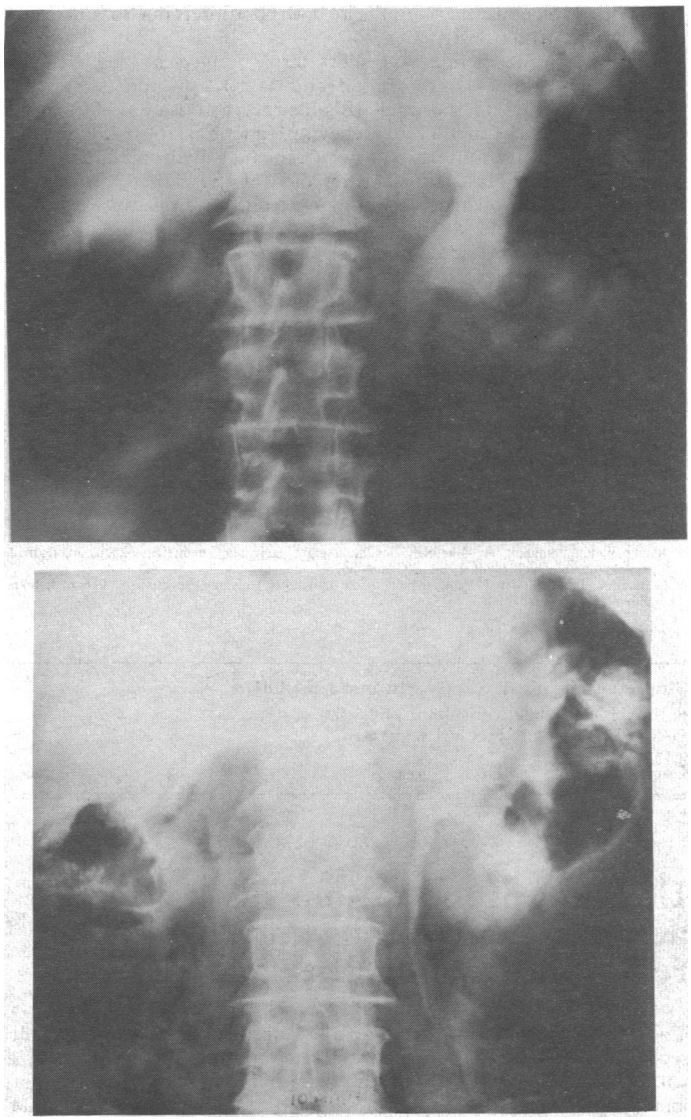
On admission the patient was mildly confused but not dehydrated. Blood pressure was 130/80 mm Hg, serum Na^+ concentration 135 mmol/l, K^+ 3·0 mmol/l, Cl^- 107 mmol/l, HCO_3^- 12 mmol/l, urea 35·6 mmol/l (214 mg/100 ml), creatinine 694 μmol/l (7·9 mg/100 ml), albumin 23 g/l, calcium (corrected) 2·05 mmol/l (8·2 mg/100 ml), inorganic phosphate 1·62 mmol/l (5·01 g/l). Urine volume was 640 ml/24 hours, and K^+ excretion 10 mmol/24 hours.

Haemoglobin concentration was 8·9 g/dl, serum folate 6·9 μg/l, vitamin B_{12} 299 ng/l, ferritin 302 μg/l. Erythrocyte sedimentation rate showed 68 mm fall in the first hour.

Urine microscopy showed 150 leucocytes and 80 red blood cells/high power field. Culture grew *Escherichia coli* with a colony count of $>10^5$ organisms/ml.

Glomerular filtration rate was 11·3 ml/min/1·73 m^2, and effective renal plasma flow 71·6 ml/min/1·73 m^2.

An antibiotic given from the second day sterilised the urine by the sixth day. The erythrocyte sedimentation rate then decreased to a 4 mm fall in one hour. Bladder catheterisation on the second day produced 100 ml urine. Intravenous fluids were given, initially containing potassium, so that urine volume increased to 1·2-1·7 l/24 hours. The catheter was removed on the fifth day but reinserted on the seventh day and 150 ml of urine was removed. Serum creatinine concentration had fallen to 427 μmol/l (4·83 mg/100 ml) by the seventh day; serum K^+ was 3·1 mmol/l, and HCO_3^- 18 mmol/l. Intravenous urography performed on the seventh day showed delayed contrast excretion from bilaterally enlarged hydronephrotic kidneys



Intravenous urograms at seven days (above) showing bilateral dilatation of upper urinary tract and at 30 days (below) showing renal damage on right side but no sign at this stage of urinary tract dilatation.

associated (figure: top) with hydroureters dilated to the bladder, which was also enlarged. Lumbosacral spine *x* ray films were normal.

Cystoscopy (Mr K C Vaughton) showed that the urethra was not tight on a 21 F gauge. The bladder was trabeculated but otherwise normal. Ureteric catheterisation was unsuccessful. Vaginal examination under anaesthesia did not indicate pelvic or faecal masses.

Appetite and food intake gradually improved, her depression abated, and confusion disappeared. The bowels were not opened often, but the stools were not hard and nor were they palpable in the abdomen. Serum creatinine concentration had fallen to 112 μmol/l (12·7 mg/100 ml) by the 14th day, when serum K^+ was 3·3 mmol/l and HCO_3^- 23 mmol/l.

A month later haemoglobin was 11·4 g/dl, serum albumin 35 g/l, serum Na^+ 145 mmol/l, K^+ 3·7 mmol/l, Cl^- 113 mmol/l, HCO_3^- 30 mmol/l, urea 4·2 mmol/l (25·3 mg/100 ml), creatinine 104 μmol/l (1·18 mg/100 ml), glomerular filtration rate 43·8 ml/min/1·73 m^2, effective renal plasma flow 127·3 ml/min/1·73 m^2. A repeat intravenous urogram showed a shrunken pyelonephritic appearance on the right side and a normal outline on the left side with normal pelvicaliceal pattern (figure: bottom).

During several months of follow up serum creatinine and K^+ concentrations remained normal and renography showed no sign of an obstructive excretory pattern.

Comment

Hypokalaemia was probably related to poor intake of food, resulting in depression and confusion and depressed neuromuscular activity throughout the smooth muscle of the whole urinary tract. Consequently the pathophysiology of bilateral hydronephrosis and hydroureters simulated periureteric fibrosis, in which peristaltic ureteric contractions are diminished in the absence of physical ob-