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Correspondence

Phosphate deficiency and rickets

Sir,

Oppenheimer and Snodgrass[1] concluded that phosphate deficiency might be an aetiological factor in neonatal rickets. We agree, and we think also that in some cases of vitamin D deficiency rickets the leaking of phosphate rather than of calcium may be responsible for the bone lesions.

Hurwitz *et al.*[2] observed that rats fed a normal calcium diet without vitamin D developed hypocalcaemia and had a slight decrease in bone ash but had no evidence of rickets, whereas when they were given a low phosphate diet with vitamin D, hypophosphataemia and severe bone lesions developed. These findings led us to investigate the severity of the rachitic process (as shown by the level of serum alkaline phosphatase) and the radiological picture of the bones in relation to the serum calcium and phosphate levels.[3] We studied 100 infants who were thought to have vitamin D deficiency and who had been treated with vitamin D between 1962 and 1974 at Aghia Sophia Children's Hospital in Athens. The diagnosis was based on raised levels of serum alkaline phosphate (>20 KA units), or serum calcium levels <8 mg/100 ml (<2 mmol/l), or serum phosphate levels <4 mg/100 ml (<1·3 mmol/l). A close inverse relationship was found between serum phosphate and serum alkaline phosphatase and the presence of radiological signs of rickets. There was no correlation between serum calcium and the severity of bone lesions.

As hypophosphataemia and rickets can be produced experimentally by phosphate deficiency alone[2] and as the level of plasma phosphate reflects the intake of phosphate, hypophosphataemia in infants with nutritional rickets must either be the result of a low phosphate intake or, as seems more likely, of a discrepancy between phosphate intake and the increased requirements of the growing infant. As vitamin D has been shown to act on both calcium and phosphate transport,[4] it is not surprising that both groups (infants with true vitamin D deficiency and infants with phosphate deficiency) responded to treatment with vitamin D. Moreover in 4 infants with hypophosphataemia and rachitic bone lesions healing took place when increased intakes of phosphate were given without additional vitamin D. It seems that lack of phosphate in some cases is the main aetiological factor responsible for the development of rickets.

References

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Sucrase-isomaltase deficiency: difficulties in diagnosis

Sir,

We report an infant who presented with severe diarrhoea in the neonatal period, and in whom the apparent detection of large amounts of glucose in the stool while on Maxijul led to a delay in diagnosis.

The patient, a girl, had been born at 38 weeks' gestation and had weighed 2·95 kg; pregnancy and delivery had been normal. She was fed Ostermilk complete formula but soon developed persistent diarrhoea. At age 2 weeks her weight was 2·84 kg, and stool chromatography showed large amounts of lactose. Because primary alactasia was suspected her feed was changed to a low-lactose formula, Galactomin 17, but diarrhoea continued with 1% reducing substances. Stool chromatography showed small amounts of lactose, glucose, and maltose. Milk protein intolerance was considered and at 4 weeks she was changed to Prosobee (powder based formula) but there was no improvement. Stool chromatography showed no lactose or glucose, and small amounts of maltose. A further change to Albumaid and Maxijul led to torrential diarrhoea, stools became strongly positive for reducing substances, and stool glucose was 64·4 mmol/l (1·16 g/100 ml) (Beckman glucose analyser). Glucose-galactose malabsorption seemed possible, and so she was changed to a fructose-based formula, Galactomin 19, on which she thrived.

Reinvestigation at age 5 months, when her weight was 6·38 kg, led to a diagnosis of sucrase-isomaltase deficiency. Histology of jejunal mucosa was normal as was the mucosal lactase concentration (5·6 units μmol disaccharide hydrolysed/g tissue per minute; normal >2·5), but maltase was very low (0·57 units; normal >10·0) and sucrase undetectable (normal >6·0). Glucose, galactose, and lactose tolerance tests were normal in that no diarrhoea was precipitated by the test and there was an appreciable rise in monosaccharide in the blood whether