Short reports

Prostatic calcification in a 4-year-old boy

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Summary

The unusual finding of prostatic calcification in a 4-year-old boy is reported.

A 4-year-old boy was admitted to hospital complaining of recurrent abdominal pain. He had not had any overt urinary symptoms. His mother stated that he had been treated for rickets in the past, receiving unknown quantities of vitamin D and calcium.

Case report

Physical examination showed nothing abnormal, except that on rectal examination firm nodules could be felt in the region of the prostate. Urine culture grew *Escherichia coli*. All other investigations were normal; these included serum calcium (2.0, 2.3, 2.3 mmol/l) and phosphorus (1.2, 1.3, 1.0 mmol/l).

A plain x-ray (Fig. 1) showed two opacities; these were thought initially to be calculi lying in the right ureter and in the bladder. A urogram confirmed the ureteric stone, and urethrography (Fig. 2) showed that the lower opacity represented the prostate.

At operation, ureterolithotomy with exploration of the bladder, the prostatic calcification was confirmed.

Postoperative recovery was uneventful and the boy was well on discharge.

Analysis of the ureteric stone showed the following: weight 262 g, volume 0.136 cm³; chemical composition—magnesium, phosphate, ammonium, uric acid, and calcium.

Fig. 1 Plain x-ray showing two radio-opaque stones, one in right ureter and the other in the prostate gland.

Fig. 2 Urethrogram showing the dye in the anterior urethra, the prostatic urethra is outlined by air.
Discussion

In adults prostatic calcification is often found in association with prostatic hypertrophy. Fox1 reviewed 3510 patients who had been examined radiologically and found that 484 (13-8%) had prostatic calculi, the youngest patient being 16 years.

The history of rickets in this patient for which unknown amounts of vitamin D and calcium had been given might explain the prostatic calcification, as this is most unusual in so young a patient. The child was symptom-free when he came to our surgical unit and there was no sign of back pressure from either the prostatic calcification or the ureteric stone. Whether this boy will have any future complications from his prostatic calcification remains to be seen.

I thank Dr Modhafer Saeed for referring the patient, and Dr Issam Al-Hatem for performing the radiology.

Reference


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Late presentation of vitamin D-dependent rickets

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SUMMARY An 8½-year-old girl presented with rickets. This had developed despite a normal diet and in the absence of symptoms, signs, or laboratory evidence of malabsorption, hepatic or renal disease. The rickets healed with physiological doses of 1-α-hydroxy-cholecalciferol. It is suggested that this case provides evidence for genetic and metabolic heterogeneity in vitamin D-dependent rickets.

Vitamin D-dependent rickets (pseudo deficiency rickets) is a rare but well recognised inborn error of cholecalciferol (D₃) metabolism, which presents classically in the first year of life. Prader et al.1 were the first to describe infants with muscular hypotonia, retarded growth and development, hypocalcaemic tetany, and severe rickets. Such patients have low concentrations of serum calcium, low or normal serum inorganic phosphate, raised serum alkaline phosphatase, high serum parathormone, and a generalised aminoaciduria. The underlying defect is thought to be an inability in the kidney to achieve hydroxylation of 25 (OH) cholecalciferol to 1,25 (OH)₂ cholecalciferol. This is inherited as an autosomal recessive. Recently it has been suggested that the condition is heterogeneous and that there may be more than one defect in the metabolism of 25 (OH) D₃ to 1,25 (OH)₂ D₃ and its subsequent action on the gut. The patient reported here presented relatively late and lends some support to the latter hypothesis.

Case report

This 8½-year-old white girl presented with a short history of fever, paraesthesia in her fingers, and tetany. No other symptoms were elicited. Her diet was normal. Her past medical history was normal, except for gross obesity in infancy. Weights recorded at the local welfare clinic include 10·5 kg at 4½ months, 14·5 kg at 10 months, and 16·4 kg at 16 months. The diet consisted of full cream Cow and Gate milk at 5 months and a good mixed diet thereafter. On examination she was febrile, with a red throat, and cervical lymphadenopathy. She exhibited carpopedal spasm but Chvostek's sign was negative. There was no clinical evidence of rickets, enamel hypoplasia, or muscle hypotonia. She was above the 50th centile for height and the 90th centile for weight. Her father is 188 and her mother 156·7 cm tall. No other abnormality could be detected.

Her tetany responded rapidly to 20 ml of 10% calcium gluconate given intravenously. Blood was then taken for analysis (Table). Her urine chromato-gram showed gross generalised aminoaciduria. She showed no proteinuria or glycosuria. X-ray films of the left wrist and hand showed active rickets, with no evidence of hyperparathyroidism.

Further investigation did not show any biochemical evidence of malabsorption, nor any sign of renal or hepatic disease (Table). The patient was started on 10000 units of calciferol BP and 8 g calcium gluconate daily (Figure). She remained asymptomatic.

After 3 weeks of treatment her serum calcium was 2·09 mmol/l (8·4 mg/100 ml) and her serum inorganic phosphate 2·02 mmol/l (6·1 mg/100 ml). The aminoaciduria had resolved and x-ray films showed some bone healing. The calciferol was stopped and her biochemistry promptly deteriorated.