Primary hyperparathyroidism in a child

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ABSTRACT

Primary hyperparathyroidism is rare in children. We report a 12-year-old girl who presented with recurrent renal calculi, muscular weakness and inability to walk; was diagnosed to have parathyroid adenoma and underwent parathyroidectomy.

Key words: Parathyroid adenoma, primary hyperparathyroidism, recurrent renal calculi

Introduction

Hyperparathyroidism is unusual in childhood, occurring in 2–5/100,000 children[1] and may be due to primary hyperparathyroidism, familial hyperparathyroidism associated with multiple endocrine neoplasia or secondary to malignancy. It may be incidentally detected or manifest with recurrent renal stones and osteitis cystica. Adenoma is a common cause and a benign condition that can be surgically corrected.

Case Report

A 12-year-old girl, apparently normal till the age of 9 years, presented with gradually worsening proximal muscle weakness and recurrent renal stone formation. There was waddling gait and over time she was limping and finding it very difficult to walk. She had undergone lithotripsy repeatedly, but the attending physicians had not evaluated her for the cause of her renal stones and muscle weakness. She was referred to Nephrology services for work-up of stone disorder.

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On examination, she was 141 cm tall (expected 146 cm) and weighed 30 kg (expected 42 kg). She was euvolemic, blood pressure was 100/70 mmHg and did not have pallor. Examination of the cardiovascular and respiratory systems and abdomen were normal. Musculoskeletal system examination revealed significant proximal muscle weakness of both the lower limbs, waddling gait, knock knees and she would climb on herself to stand erect (positive Gower's sign). Laboratory investigations showed normal urine examination, normal arterial blood gases and renal functions; serum calcium was 15 mg/dl, serum albumin was 4.0 g/dl, phosphorus was 2.8 mg/dl, alkaline phosphatase was 1619 units/L, parathyroid hormone (PTH) was 1319 pg/ml and the 25 hydroxy Vitamin D level was 9.8 ng/ml. Urinary excretion of calcium was high with random urinary calcium/creatinine ratio of 0.35.

X-ray of the hand [Figure 1] showed subperiosteal resorption and cystic lesions. Long bones showed thinning of the cortex. Hypertrophied left superior parathyroid gland was demonstrated by ultrasound examination of the neck. Sestamibi scan highlighted the left upper parathyroid gland which persisted after 2 h of wash out period [Figure 2].

In view of the parathyroid adenoma, she underwent left upper parathyroidectomy under per-operative radio-guidance. The immediate pre- and post-surgery PTH were 1619 pg/ml and 203 pg/ml. Histological examination of the excised gland confirmed it to be a benign adenoma. Postoperatively, serum calcium reduced to a nadir of 7.0 mg/dl, and she was treated with intravenous calcium gluconate initially followed by oral calcium and Vitamin D.

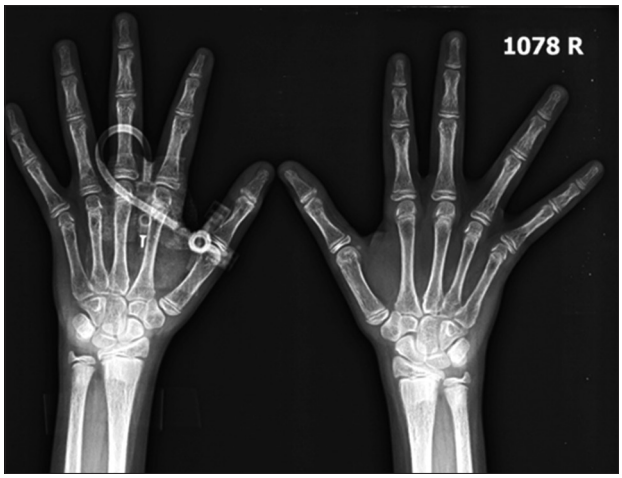

Figure 1: X-ray hands showing subperiosteal erosions and cystic lesions

Three months after the surgery her serum calcium was 10.1 mg/dl, the proximal muscle weakness and waddling had significantly reduced, and her speed of walking had increased.

Discussion

The incidence of kidney stones in children is unknown, but described in 1 out of 1000–7500 pediatric hospital admissions. Most common causes are metabolic abnormalities, anatomic abnormalities leading to urinary stasis and urinary tract infection. Hypercalciuria contributes to 50% of metabolic risk factors; hyperoxaluria, hyperuricosuria and cystinuria are among others. Genetic disorders like Dent's disease, Bartter's syndrome, Wilson's disease, Glycogen storage disease, hereditary distal renal tubular acidosis, familial hypomagnesemia and multiple endocrine neoplasia lead to hypercalciuria. Hyperparathyroidism and chronic metabolic acidosis increase bone resorption and hypercalciuria. Hypocitraturia increases the risk of crystallization.

Unlike adults, predisposing factors can be identified in up to 87% of children; recurrent stones occur in 67% of pediatric patients. Hence all children require a complete evaluation with metabolic work up.

Evaluation includes a detailed history of diet, fluid intake and micturition habits, and family history of renal calculi. Urine should be analyzed for the pH, specific gravity and presence of crystals. Twenty four-hour urine collection is ideal to look for volume, creatinine, calcium, phosphate, oxalate, uric acid, sodium and citrate. If 24 h collection cannot be obtained, at least spot sample should be assessed for these. Serum levels of calcium, phosphorus, bicarbonate, magnesium, uric acid, PTH and alkaline phosphatase complete the work-up for stone. Radiological evaluations for the stones include X-rays and ultrasound. They help in locating the stone and presence of anatomical abnormalities if any, and planning therapy.

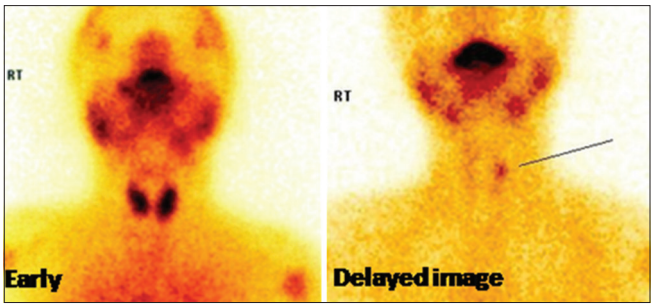

Figure 2: Sestamibi scan showing persisting activity in the left superior parathyroid gland (arrow)

Primary hyperparathyroidism is not uncommon in adults and is found in 0.3% of the general population and is often seen in the third and fourth decades. The incidence is very low in children (2–5/100,000) with very few case reports.[1-4] Solitary benign adenoma is the cause in 80–85% of the cases. Diffuse hyperplasia of all the four glands is seen in 15–20%.[5] The clinical presentation can vary from asymptomatic incidentally detected hypercalcemia to recurrent renal calculi with its consequences. Non-renal manifestations include pathological fractures, bony pain, brown tumors, psychosis, depression, mood swings, muscular weakness, growth retardation and pain in abdomen. In view of the varied manifestations, they can present to different medical specialties. The diagnosis is clinched by the presence of hypercalcemia, increased PTH levels, hypercalciuria, hypophosphatemia and hyperphosphaturia. The extent of demineralization due to hyperparathyroidism will be seen as resorptive areas in the periosteum, lytic lesions and thinning of the cortex. Ultrasound of the parathyroid gland guides the surgeon in locating the gland and its size. ^{99m}Sestamibi scan with single photon emission computed tomography helps in localizing the adenoma. Surgical removal remains the standard therapy. After surgical removal of the gland, hungry bones reclaim calcium requiring large replacements with stringent monitoring.

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