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AN INTERESTING CASE OF KNOCK KNEE

General Medicine

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ABSTRACT

Primary hyperparathyroidism is a generalized disorder of calcium, phosphate, and bone metabolism due to an increased secretion of parathyroid hormone. The manifestations may be subtle, and the disease may have a benign course for many years or a lifetime. If the patients are symptomatic , the most commonly involved system include the renal and the skeletal system. Herein we present a case of 16 year old male who presented with complaints of multiple large joint pain and progressive deformity of both knees over the past 1 year. Physical examination revealed bilateral genu valgum. Investigations taken showed elevated serum calcium levels, parathormone levels, alkaline phosphatase levels and low levels of vitamin D. Skeletal radiograph taken showed evidence of osteitis fibrosa cystica along with salt and pepper appearance of the skull. USG Neck showed evidence of left parathyroid adenoma and a Tc99 Sestamibi parathyroid scintigraphy scan confirmed it. Patient underwent inferior parathyroidectomy. Post procedure, patient's parathormone levels were within the normal limits and hence was discharged with calcium and vitamin D supplements.

KEYWORDS

Primary hyperparathyroidism, Genu valgum, Parathyroid adenoma

INTRODUCTION

Primary hyperparathyroidism (PHPT) is common in adults, with a frequency of around 3 in 1000[1]whereas it is rare in children and young adults . The most common cause is excess parathyroid hormone release secondary to a parathyroid adenoma[2] and the least common cause being multiple endocrine neoplasia. Incidental detection of hypercalcemia in routine blood investigation allows the diagnosis at an asymptomatic stage in adults. In contrast, diagnosis in children is often made after they are symptomatic with target organ involvement. The incidence of juvenile hyperparathyroidism is 2-5cases/million[3]. Almost half of the patients experience moderate neuromuscular symptoms like fatigue and lethargy. Peptic ulcers and pancreatitis are possible side effects of symptomatic hypercalcemia, which primarily impacts the skeletal, renal, and gastrointestinal systems. 25% of individuals have osteopenia as determined by bone mineral density and may develop bone pain or fractures . Around 20% of people will develop the renal stone diseases such as nephrolithiasis and nephrocalcinosis. Less than 5% of the patients might have the skeletal abnormalities associated with osteitis fibrosa cystica, including subperiosteal resorption of the distal phalanges, tapering of the distal clavicles, salt-and-pepper pattern of the skull, bone cysts and brown tumors of the long bones. It is hypothesized that certain skeletal symptoms caused by PHPT may become more obvious during puberty.

CASE REPORT

A 16 year old male presented with pain over multiple large joints for 1 year and progressive difficulty in walking and progressive deforming of knee for 1 year duration. There was no history of trauma, fractures, abdominal pain, vomiting ,constipation or neck swelling. He has undergone tonsillectomy at the age of 9 years .There was no history of similar illness in family members and his siblings were healthy.

On examination, bilateral valgus deformities of the knees without any other clinical features of rickets or dysmorphism were present. No other joint deformities were noted. Height and weight were appropriate for age and he was in Tanner 4 in pubertal development. His height was 183 cm and weight was 71 kg with a BMI- 21.2 kg/m2 and the mid-parental height was 178.5cm. The upper segment was 85 cm and the lower segment was 98 cm with a US/LS ratio of 0.8. The rest of the systemic examination was normal.

Investigations revealed serum calcium levels of 12.41 mg/dl (8.4-10mg/dl) and serum phosphate levels of 3.41mg/dl (2.7-4.9mg/dl). ALP levels in the serum were high at 3031 U/L (50-135 IU/L) . Parathyroid hormone values in the serum were high 804.7pg/ml (14-72 pg/ml) and suggested hyperparathyroidism. Levels of vitamin D were 25 nmol/l (less than 30 nmol/l is indicative of insufficiency). Thyroid function tests , anterior pituitary hormones, baseline cortisol, complete blood count , liver function tests, renal function tests and serum electrolyte profiles were all normal. The X-ray images of lower limbs showed bilateral genu Valgum. A skeletal examination revealed subperiosteal bone resorptions in radial aspect of proximal phalanx of all the digits and expansile lytic lesion in the base of right 4th proximal phalanx suggestive of Brown tumor along with the lateral radiograph of the skull demonstrating a salt and pepper appearance of the skull. There were no radiological signs of rickets, such as metaphyseal enlargement, cupping, or fraying. An ultrasound of the neck revealed 1.6 x 1.7 cm sized well defined cystic lesion with low grade internal echogenicity in the inferolateral aspect of left lobe of thyroid indicating a parathyroid adenoma. Ultrasound scan of the abdomen did not reveal the presence of any renal calculi or nephrocalcinosis. A Tc99 Sestamibi parathyroid scintigraphy scan revealed hypermetabolic well defined soft tissue nodule of size 19 x 20 x 23 mm (ML x AP x CC), posterior to the left lobe of thyroid gland, abutting the esophagus, representing a left inferior parathyroid adenoma. A diagnosis of primary hyperparathyroidism secondary to parathyroid adenoma was made and he underwent a left focused inferior parathyroidectomy surgery in which a sizable 3 x 2 cm parathyroid gland was excised posteroir to left strap muscles. The Histology revealed the presence of adenoma in the excised parathyroid gland. He had chvostek sign positivity following surgery and hence was administered with an infusion of calcium gluconate. PTH levels in the post operative period were 56.14pg/ml. He was discharged on postoperative day 8 with calcium and vitamin D supplements.

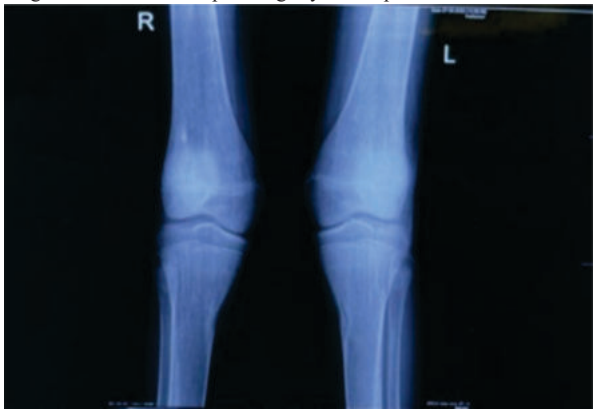
DISCUSSION

Our adolescent patient had bilateral genu valgum when he was first diagnosed with hyperparathyroidism. His age at presentation is in line with people with a comparable abnormality mentioned in literature. Majority of the documented instances appeared during the adolescent era, between the ages of 11 and 17. Uncertainty prevails in the underlying cause of this disorder in this particular age group. It has been suggested that the primary contributing factor may be the direct impact of increased parathyroid hormone on the epiphyseal plate and the subsequent bone remodeling during the pubertal growth spurt.

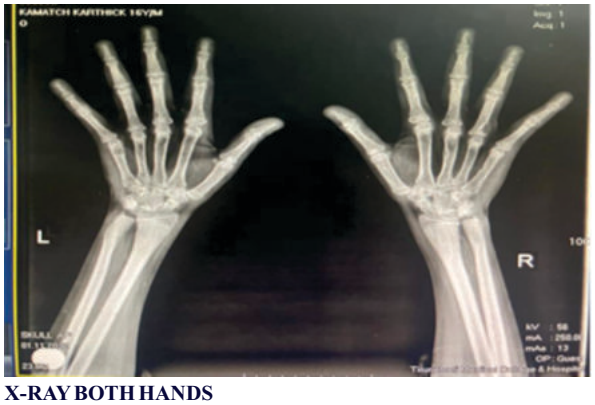
Primary hyperparathyroidism in children and adolescents is brought on by either parathyroid hyperplasia, which can be sporadic or familial, or parathyroid adenoma (single or multiple). FIHPT, HPT-JT, and MEN 1 or MEN 2A are examples of familial causation. Due to the absence of a family history of the above-mentioned illnesses, our patient most likely has sporadic PHPT. Further testing revealed normal structural and functional status of the pituitary gland. All of the published instances, including our patient, appear to be caused by

solitary parathyroid adenoma.Without any other signs or symptoms related to hypercalcemia, our patient only had isolated bilateral genu valgum. Symptoms of hyperparathyroidism that usually affect the gastrointestinal, renal, musculoskeletal, and neurological systems could be neglected as insignificant ones and thus it is essential to test the calcium levels which is less often considered in the standard blood testing of children. Because of this, juvenile age group frequently experiences delayed diagnosis of PHPT with end-organ damage upon presentation, brown tumours, salt-and-pepper radiologic appearance of the skull, and osteopenia. One of the recognised clinical signs of nutritional rickets is genu valgum. The diagnosis of PHPT is subsequently made in such individuals because of the lack of clinical improvement following treatment, the new emergence of hypercalcemia and the sustained increase of parathyroid hormone following vitamin D supplementation. PHPT presentation in juvenile population is severe compared to their adult counterparts despite identical serum PTH levels, presenting with greater hypercalcemia and hypercalciuria. The main distinctions between these two age groups were thought to be enhanced target tissue responsiveness to the effects of parathyroid hormone and decreased parathyroid adenoma susceptibility to negative feedback by calcium in Juvenile PHPT.

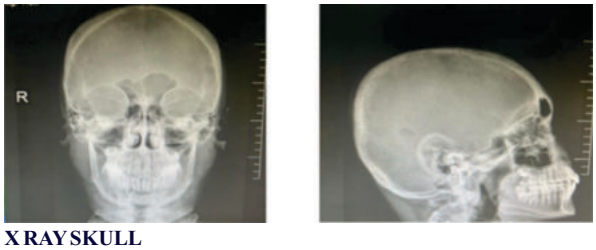
In patients with PHPT, parathyroidectomy is the basic modality of treatment. Goals of treatment include symptom reduction, end-organ damage repair, and an instantaneous and permanent cure for excessive serum calcium and parathyroid hormone levels. Almost all cases that have been recorded in literature, including ours, have had successful parathyroidectomies. Hungry bone syndrome poses a greater risk in this age group as witnessed in our case during the early post-operative period. It was treated immediately and the patient was discharged with calcium and vitamin-D supplements. Osteotomy to treat bilateral genu valgum is needed in the upcoming days in our patient.



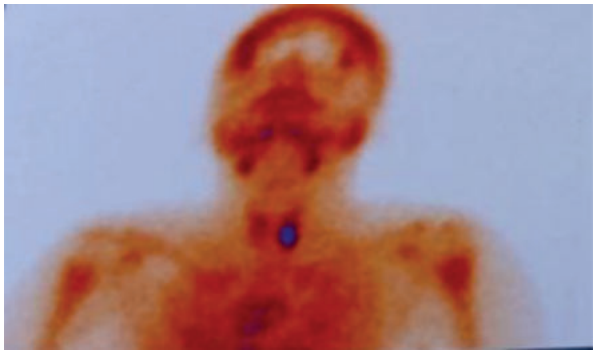
X-RAY LOWER LIMBS – GENU VALGUM



X-RAY BOTH HANDS



X RAY SKULL



99m-Tc-SESTAMIBI PARATHYROID SCINTIGRAPHY

CONCLUSION

Genu valgum is an atypical presentation of primary hyperparathyroidism in children and adolescents, which is uncommon and frequently identified late. However, a high index of suspicion is necessary because disabling consequences may be cured or reversed with timely parathyroidectomy.

REFERENCES

1. Goldman – Cecil Medicine, 25th edition vol-1, by Lee Goldman & Andrew I. Schafer, chapter 245, Hyperparathyroidism: p 1655
2. Kollars J, Zarroug AE, van Heerden J, Lteif A, Stavlo P, Suarez L, et al. Primary Hyperparathyroidism in pediatric patients. Pediatrics 2005;115:974-80.
3. Schmidt H.,Kusser B.,Spelsberg F.:Asymptomatic hypercalcemia due to an ectopic parathyroid adenoma in an 8 year old boy. Exp Clin Endocrinol Diabetes 2001;109:184–6.