

CASE REPORT**PARATHYROID ADENOMA IN AN ETHIOPIAN ADOLESCENT LIVING WITH HUMAN IMMUNODEFICIENCY VIRUS: A RARE ASSOCIATION**Tinsae Alemayehu, MD^{1*}, Workeabeba Abebe, MD¹, MPH**ABSTRACT**

Background: Parathyroid adenomas are rarely diagnosed before adulthood. Diagnosis among HIV infected people has even rarely been described in the medical literature.

The case: We report about a 19 year old female adolescent living with HIV who presented with a chronic body weakness, multiple fractures, and renal stones. Serum electrolytes and parathyroid hormone level measurements, radiographic and imaging findings and a post-operative biopsy confirmed a diagnosis of primary hyperparathyroidism due to a solitary parathyroid adenoma. Symptoms improved after surgical excision of mass and supplementation with calcium, phosphorus and vitamin D.

Conclusion: We describe a rare co-occurrence of primary hyperparathyroidism due to a solitary parathyroid adenoma in an HIV infected adolescent.

Key words: HIV, Hyperparathyroidism, Parathyroid adenoma, Ethiopia

INTRODUCTION

Primary hyperparathyroidism is a rare diagnosis in children and adolescents. Causes range from hyperplasia or adenoma of the parathyroid gland, ectopic parathyroid hormone production, vitamin D excess, prolonged immobilization, hematologic malignancies, and etc. Adenomas are the commonest etiology in older children and adolescents (1). Though pediatric parathyroid adenomas have been reported in literatures as early as the 1930s, the reports are sparse (2).

From post-mortem examinations of HIV infected patients, Cherqaoui et al reported Parathyroid hyperplasia to be the most common cause parathyroid gland histologic abnormality (22.5%) (3). Vitamin D deficiency is also seen in 60% of HIV infected adults, with half being attributed to hyperparathyroidism (4).

We report the presentation of a 19 year old adolescent patient living with HIV who was diagnosed with primary hyperparathyroidism due to a solitary parathyroid adenoma. To our knowledge, the two diagnoses have not been reported in a single patient.

Case Presentation

A 19 year female adolescent from Addis Ababa presented to our hospital with a progressive body weakness and bone pain on multiple sites of 1 year duration. She subsequently developed numbness and tingling over all extremities, leg swelling and weight loss. Eventually, she started to walk with support, to the extent that she unable to walk and finally became bed-ridden prior to presentation.

A right leg injury four months before presentation resulted in a closed proximal tibial fracture, which was treated conservatively with a cast. Closed manipulation of the tibial cast resulted in fracture of the mid shaft of her right femoral mid-shaft. An alleged minor trauma two weeks later resulted in a left closed femoral fracture.

Her past medical history included HIV infection that was diagnosed at the age of 12 years and she had undergone a right sided nephrolithotomy for renal stones at 10 years of age. For the HIV infection, an initial regimen of Stavudine/Lamivudine/Efavirenz was started, and then shifted at age 14 years to Tenofovir/Lamivudine/Efavirenz due to a disproportional weight gain. The second regimen was changed to Abacavir/Lamivudine/Nevirapine at age 18 years because of a major depression disorder attributed to Efavirenz and deranged renal function.

On examination, she appeared chronically sick. Her lower extremities were rotated outwards with posterior and lateral angulations of her bilateral thighs. She had diffuse bony tenderness, scoliosis and bilateral grade 1 pedal edema.

Her lab tests showed the following: Serum alkaline phosphatase values ranged from 2500–4700 IU/L. She had normal liver and renal function studies. She had hypercalcemia (total calcium 11.9 mg/dL and ionized calcium of 3 mg/dl) and hypophosphatemia (0.38 mmol/dl).

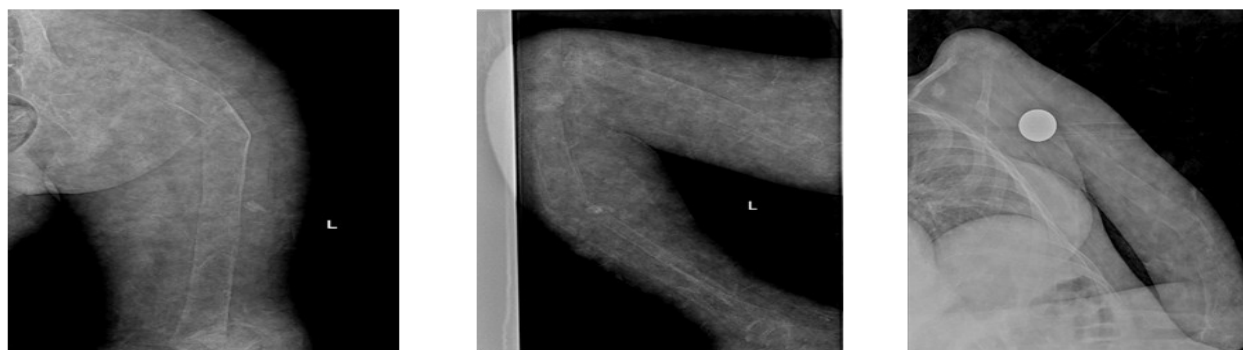
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Her urinalysis showed a pH of 7, specific gravity of 1.02 and calcium oxalate crystals. Serum vitamin D (25-OH-D) level was <3 ng/ml (normal 6.2 – 49 ng/ml) and a serum parathyroid hormone level was 1900 pg/ml (normal 14 – 72 pg/ml). Her ECG was normal.

X-rays of the long bones showed severe bone density loss in all long bones with multiple bone fractures and deformities.

All imaged joints were deformed with a loss of joint space. There were also diffuse soft tissue calcifications (Figures A – C). An abdominal ultrasound revealed bilateral multiple nephrolithiasis (the largest measuring 1.5 cm). A thoracolumbar MRI was normal.



Figures A – C: Proximal and mid-femoral, proximal tibial and left humeral fractures

A CT scan of the neck was not possible for fear of procedure related cervical instability and fractures. Hence, a neck ultrasound was done and showed a hypo-echoic well-defined oval right neck mass measuring 3.1 x 1.2 x 0.6 cm.

She was diagnosed with primary hyperparathyroidism due to a parathyroid adenoma and underwent surgery. A 3 x 4 x 2 cm nodular parathyroid mass from the right upper pole was excised and subjected to biopsy. Biopsy showed a lobulated encapsulated firm tissue, with proliferation of chief cells, diffuse mild pleomorphism, a focal trabecular pattern with no capsular invasion and normal surrounding parathyroid tissue, confirming a parathyroid adenoma. A serum parathyroid hormone level on her 6th post-op day was 202.3 pg/ml with normal serum calcium and phosphate.

Post-operatively, the patient was supplemented with calcium, phosphorus and vitamin D. On subsequent examinations, there was marked improvement of her symptoms (better mobility, weight gain, no extremity numbness/tingling).

DISCUSSION

Parathyroid adenomas do not usually develop in early life. They are benign masses with clinical presentation of anorexia, nausea, vomiting, constipation, polyuria, polydipsia and in advanced cases, nephrolithiasis, bone pain, pancreatitis or rickets (5).

The usually subtle signs lead to delayed diagnosis and treatment (6). Our patient had multiple fractures, bone pain, hypercalcemia, hypophosphatemia and a 100 times elevated serum parathyroid hormone level. Diagnosis is settled by demonstrating elevated parathyroid hormone levels and electrolyte imbalances.

Imaging using ultrasonography or CT scans does not usually detect normal parathyroid glands. Hence, if glands are notable on these studies, they can be considered enlarged (7). The normal parathyroid glands measure 0.5 x 0.3 x 0.1 cm. Adenomas appear homogeneously hypoechoic to the overlying thyroid gland. They are usually solitary and rarely involve two glands (5,8). Our patient's solitary adenoma measured 3 x 4 x 2 cm. No family members with such clinical presentation and diagnosis were reported.

Surgical removal is indicated in all such cases. One must be vigilant towards a rare immediate post-operative complication known as the "hungry bone syndrome" which is treatable by supplementing calcium, phosphorus and vitamin D (9).

In conclusion, we describe a rare diagnosis of primary hyperparathyroidism due to a solitary parathyroid adenoma in an adolescent living with HIV. A delayed diagnosis resulted in many deformities disabling our patient from performing many vital activities.

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