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Case Report

Discovery of Primary Hyperparathyroidism in a Patient with Nephrolithiasis and Multiple Limb Fractures

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Abstract

Primary hyperparathyroidism (PHPT) is an endocrine disorder characterized by excess secretion of parathyroid hormone (PTH), resulting from the hyperfunction of one or more of the parathyroid glands. The leading cause of primary hyperparathyroidism is the solitary parathyroid adenoma in 80% to 85% of cases [1–4]. Secondary causes include parathyroid hyperplasia (10% to 15% of cases), multiple adenomas (5% of cases), and rarely parathyroid cancer (<1% to 5% of cases) [1,2]. Biochemical distinctive features are hypercalcemia and high or inappropriately normal PTH (1-3). The prevalence of primary hyperparathyroidism is approximately 1–7 per 1000 in the general population, affecting predominantly postmenopausal women [4]. Consequences, in this case, are nephrocalcinosis, renal lithiasis, and spontaneous fractures in the field of osteoporosis.

Keywords: Primary Hyperparathyroidism; Parathyroid Adenoma; Renal Calculus; Spontaneous Fracture

Case Report

A 66-year-old female patient presented to the Trauma Hospital Emergency after a light fall in the apartment, with pain in the right leg. She was diagnosed with trochanteric fracture of the right femur and was hospitalized in the Orthopedic department.

In 2008, the patient was diagnosed with Fracture of the right humerus from a very light fall. The fracture was treated with immobilization and after 6 weeks she had 25 radiotherapy sessions for an undetermined mass at the site of the fracture. In 2009, the patient was diagnosed with a fracture of the left femur, which occurred without any stimulus other than shock or radiation, for which intervention was performed. In 2013, in a routine abdominal echography, numerous calculi with different dimensions were observed in both kidneys (Figure 1).

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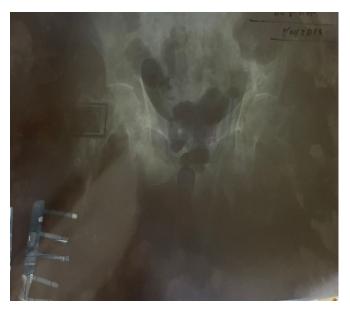


Figure 1: Radiography of the treated fracture of the femur

In 2015 and 2016, the patient performed surgical procedure for the calculi removal. In 2018, the patient was diagnosed with Chronic Renal Disease and was followed regularly by a nephrologist. Meanwhile, the patient during this period was treated also with stage 2 arterial hypertension. In December 2022, the patient was diagnosed with Acute Pancreatitis, for which she was treated in hospital.

Based on the patient's anamnesis, medical and surgical antecedents, radiological laboratory examinations (x-ray, scintigraphy, echography) are performed, resulting in a positive diagnosis of Primary Hyperparathyroidism caused by Parathyroid Adenoma, and manifested with multiply fractures and nephrocalcinosis (Figure 2).

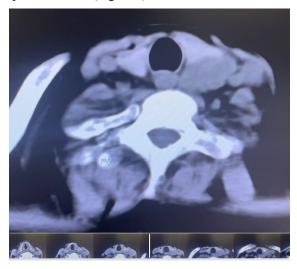


Figure 2: Abdominal CT scan showing nephrolithiasis.

Laboratory examination results

Urea 104 mg/dl, creatinine 2.42 mg/dl, calcium 10.4 mg/dl, ionized calcium 1.37 mmol/l, phosphoremia 3.5 mg/dl, potassium 4.33 mmol/l, total protein 6.7 g/dl, albuminemia 3.5 g/dl.

LDH 106 u/l, ferritin 1023.62 ng/ml, TSH 0.029 mu/l, ft4 0.59 pmol/l (0.7-1.48), ft3 ng/l (1.58-3.91), urine complete; leukocyte fields filled, erythrocyte fields filled. PTH 1492 ng/l (15-65). Eco of the thyroid and parathyroid gland showed an adenoma of the left inferior parathyroid gland measuring 2 x 3 cm.

Scintigraphy of the parathyroid gland resulted in a massive adenoma of the left parathyroid.

Under these conditions, the patient was scheduled for surgical intervention, where she first started treatment with Rocaltrol 0.25 mg 2 times 2 tablets a day and calcium gluconate 500 mg 3 times 1 tablet a day [5].

On the scheduled date, the patient underwent the inferior sinister parathyroidectomy (Figure 3,4)



Figure 3: Thyroid and parathyroid echography. Left parathyroid adenoma.

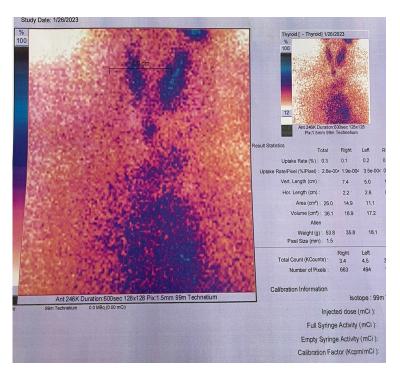


Figure 4: Parathyroid scintigraphy showing fixation of technetium

Post-intervention analyses; ionized Ca 1.13 mmol/l, total calcium 9.8 mg/dl, PTH 27 mg/dl, phosphoremia 4.2 mg/dl. The patient was in a good condition after the surgery [6-10].

Discussion

We presented the case of primary hyperparathyroidism in a patient with spontaneous fractures. The documentation of this case is important in terms of the treatment and management of similar cases, best focusing on:

- 1. Identification of changes in treatment, prevention of osteoporosis, and use of bisphosphonate therapy increasing bone density and preventing spontaneous fractures.
- 2. Early detection of primary hyperparathyroidism caused by parathyroid adenoma, identification of high-risk patients.
- New protocols for these patients and guidelines for the management of patients with HPT P and at high risk for spontaneous fractures and renal calculus.

4. The importance of monitoring calcium and PTH in the blood. Complications caused by Primary HTP in the context of a parathyroid adenoma, in addition to these complications remain to be discussed in the future and the changes that may occur in other organs, knowing that calcium can also be deposited somewhere else.

Conclusion

Primary hyperparathyroidism caused by parathyroid adenoma is an endocrine condition that causes high levels of calcium in the blood and results in spontaneous fractures due to bone weakening. The discussion focuses on the pathophysiological factors that lead to the development of these complications, diagnostic methods, and treatment, as well as the role of preventing spontaneous fractures in these patients. The use of advanced diagnostic methods and effective treatment protocols prevent complications and improve the lives of patients. To understand the importance of evaluating the levels of calcium and PTH in the blood, and promote lifestyle changes to reduce the risk of fractures and other complications.

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