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Primary Hyperparathyroidism Revealed by Impaired Renal Function with Persistent Elevation of Parathormon (PTH) and Normal Calcemia after Surgical Removal of the Parathyroid Adenoma

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Abstract

Introduction: The curative management of primary hyperparathyroidism is based on surgery. We report the case of a patient presenting with elevated parathormone despite surgical removal of the parathyroid adenoma. **Observation:** A 48-year-old patient presented for more than 6 months with significant weight loss, polyuria with polydipsia and impaired renal function. The assessment carried out revealed hypercalcaemia at 155 mg/L with hypophastetaemia at 24 mg/L and an elevation of the parathyroid hormone at 218.9 pg/ml. Cervical ultrasound showed a mass at the expense of the lower left parathyroid gland. These results made it possible to conclude primary hyperparathyroidism by parathyroid adenoma which was supported surgically. The postoperative biological assessment revealed a normocalcemia at 85 mg/L associated with an increase in parathormone (PTH) at 271.9 pg/ml. In front of the normalization of calcemia in the subsequent controls and the amendment of all the signs, monitoring was carried out. 9 months after surgery, spontaneous normalization of PTH was observed at 38.4 pg/ml with normal serum calcium at 90 mg/l. **Discussion:** Primary hyperparathyroidism can be revealed by other manifestations. Post-operative follow-up is generally simple with normalization of PTH and serum calcium. However, there may persist an elevation of PTH with normal self-limiting calcemia.

Keywords

Primary Hyperparathyroidism, Postoperative Follow-Up, Normocalcemic Hyperparathormonemia

1. Introduction

Primary hyperparathyroidism (PHP) is the excessive and inappropriate secretion of parathyroid hormone (PTH) due to hyperplasia of one or more parathyroid glands or the presence of an adenoma. The main metabolic consequence of this excessive production of parathyroid hormone is hypercalcemia [1]. Women are affected 2 to 3 times more often than men. Postmenopausal women seem particularly affected since the prevalence is 21 per 1000 women between 55 and 75 years old, while the overall prevalence is 0.1% [2]. The most reported circumstances of discovery are renal lithiasis and bone pain [3]. Parathyroid surgery (sufficient resection of pathological tissue) is the only therapeutic option to cure PPH. Transient moderate hypocalcemia is often seen after parathyroid surgery, and is considered an important stimulator of healthy inhibited parathyroids. In general, PTH and calcium return to their normal values within 30 hours of surgery [3]. We report a case of primary hyperparathyroidism revealed by impaired renal function with persistent elevation of PTH and normocalcemia after surgical removal of the parathyroid adenoma.

2. Medical Observation

A 48-year-old female patient was referred by a nephrology department for management of hyperparathyroidism. The onset dates back to around 6 months with progressive weight loss (estimated at around 25% in 6 months), asthenia, polyuria with polydipsia, and digestive disorders (nausea, constipation). The patient consults and carries out an assessment which shows a regenerative normochromic normocytic anemia at 9.7 g/dl, fasting venous blood sugar at 0.82 g/l with a glycated hemoglobin at 5.4%, and impaired renal function with a creatinine at 18.1 mg/l (N: 6 - 11 mg/l). The clearance according to MDRD was 31.4 ml/min (N: 90 - 140 ml/min).

Faced with these results, she was referred to nephrology where she carried out a second assessment which confirmed the anemia and impaired renal function. Liver function tests, protein electrophoresis and HIV serology came back unremarkable. Electrolyte balance showed hypercalcemia at 147 mg/l with corrected serum calcium = 147.24 mg/l (N: 81 - 104 mg/l) and hypophastemia at 24 mg/l (N: 26 - 45 mg/l) motivating the parathyroid hormone assay (PTH) coupled to TSH. Intact PTH comes back elevated at 218.90 pg/ml (N: 9 - 45 pg/ml) and TSH is normal at 3.07 pg/ml (N: 2 - 5 pg/ml). Calciuria was also high at 537 mg/24H (N: 100 - 300 mg/24H). The abdominopelvic ultrasound returned normal (absence of renal calcifications, normal kidneys) as did the radiographic assessment (spine, pelvis).

The patient was therefore referred to endocrinology for treatment of this hyperparathyroidism. In view of the clinical and biological picture, we thought of primary hyperparathyroidism. In the absence of scintigraphy with sesta methoxy-isobutyl-isonitrile (sesta MIBI) in our country, we performed a cervical ultrasound which objectified a tissue mass at the expense of the lower left para-

thyroid gland. The search for the other components of a possible multiple endocrine neoplasia came back unremarkable. Bone densitometry was not performed because it was not available. Surgical management is indicated and performed 7 days after diagnosis. Ten days after the surgery, the patient reports improvement of all signs with a weight gain of 5 kg. The postoperative assessment shows a normal calcemia at 85 mg/l, an improvement in renal function with creatinine at 11.7 mg/l (Clearance according to MDRD = 64.3 ml/min). On the other hand, there is a greater increase in PTH at 271.90 pg/ml. Anatomopathological analysis of the surgical specimen was in favor of a parathyroid adenoma with main cells without signs of malignancy. In view of the clinical improvement and normalization of serum calcium, we opted for monitoring. Three months later, the patient reported no recrudescence of the signs, we note a weight gain of 11 kg. high. Six months later, the patient has no complaints. The calcemia is always normal at 90 mg/l with a spontaneous normalization of the PTH at 38.4 pg/ml.

3. Discussion

PHP is observed at any age, from early childhood to the very elderly. However, the incidence of the disease increases gradually in adulthood, with an average age close to 58 years when the diagnosis is established. The female predominance is 2 to 3/1. If the prevalence is close to 1/1000, it is close to 1/500 in women over 45 [4]. Our case concerns a woman in whom PHP was diagnosed at the age of 48.

Renal lithiasis and bone pain are the most revealing clinical manifestations of the disease [3]. However, the clinical picture of osteoporosis or nephrolithiasis has become rare, and 80% of PHP are currently diagnosed at an asymptomatic stage [5]. The case of our patient was clinically revealed by significant weight loss and polyuro-polydipsia, in a context of impaired renal function. The diagnosis is confirmed, biologically, in the presence of high serum calcium values (>110 mg/l or 2.6 mmol/l) with hypercalciuria, inappropriate for high or simply normal PTH values [6].

Parathyroid imaging is the best way to localize the lesion. Cervical ultrasound and sesta MIBI scintigraphy are the examinations of choice for locating the morphological abnormality [7]. In the absence of sestaMIBI scintigraphy in Ivory Coast, the cervical ultrasound was able to make the morphological diagnosis in the patient. Cervical ultrasound can also detect an added thyroid pathology.

The curative management of PHP is based on excision of the pathological parathyroid tissue. Surgical treatment, performed by a minimally invasive technique, is recommended for patients with symptomatic PHP [3]. Indications for this surgical treatment are: age under 50, serum calcium over 110 mg/l, urinary calcium over 400 mg/24H, creatinine clearance under 60 ml/min, Z score < - 2, 5 DS to bone densitometry of the spine and/or femur [5] [8]. The presence of a single criterion is sufficient for surgical management. The patient had all the in-

dications for surgical management except for bone densitometry, which could not be performed due to the technical facilities available.

The pathological analysis of the surgical specimen confirms the histological nature of the morphological abnormality. Parathyroid adenoma (single or multiple) is the main cause of primary hyperparathyroidism, followed by hyperplasia of the parathyroid glands and then parathyroid cancer [9]. Parathyroid adenomas are benign tumors of chief or oncocytic cells. Most of these adenomas develop in normally located parathyroids [3]. The patient had a parathyroid adenoma developed at the expense of the left inferior parathyroid gland.

Effective parathyroid surgery results in normalization of calcium and PTH concentrations. Symptoms and quality of life for most patients are improved [10]. Careful monitoring of serum calcium is essential in the postoperative phase. Transient mild hypocalcemia is often seen after parathyroid surgery, and is considered an important stimulator of healthy inhibited parathyroids. In general, PTH and calcium return to their normal values within 30 hours of the procedure.

PTH decreases after excision of autonomic parathyroid tissue since healthy parathyroids were inhibited in the presence of hypercalcemia and therefore secreted no more or almost no PTH [3].

The follow-up of the biological parameters in particular the calcemia and the PTH must be done for at least 6 months after the surgery. A high level of PTH with normal calcemia after parathyroid surgery has been reported in 12% to 43% of cases [11]. However, the percentage of patients decreased as the time since surgery increased [12] [13].

Research into the preoperative characteristics of patients who presented with elevated PTH with normocalcemia postoperatively revealed higher levels of PTH in the preoperative period of these patients [13] [14]. Carsell *et al.* demonstrated a significant positive correlation ($P < 0.0001$) between the higher level of PTH preoperatively and the persistence of a high level of PTH postoperatively [15]. A preoperative PTH greater than 200 pg/mL could be considered a risk factor [12] [13]. Other factors seem to be incriminated, namely impaired renal function, vitamin D deficiency [11]. The follow-up of these patients over several months, approximately 18 months, has objectified a spontaneous and progressive normalization of the PTH level in patients with postoperative elevation of PTH [12]. It is reported that taking vitamin D postoperatively, even in non-deficient patients, would significantly reduce the incidence of these poorly explained hyper-PTHs [15]. Moreover, the five-year risk of recurrence of PPH in the event of persistent elevation of postoperative PTH was evaluated at 2.5% by Solorzano *et al.* [13].

4. Conclusion

Primary hyperparathyroidism is an uncommon condition that can be revealed by other clinical manifestations (weight loss, polyuria, polydipsia) and biological (impaired renal function). The most common cause is parathyroid adenoma, which can be demonstrated on cervical ultrasound. Curative management is

based on parathyroid surgery. Postoperative normalization of PTH is not always obtained even in the event of disappearance of clinical manifestations and normal calcemia. This postoperative elevation of PTH would be related to higher values of PTH preoperatively and would evolve towards a progressive decrease and spontaneous normalization in a few months.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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