

## Delirium Revealing a Parathyroid Adenoma

Jebbar Nourddine<sup>1,2\*</sup>, Elkaissi Jaber<sup>1,2</sup>, Maaroufi Ayoub<sup>1,2</sup>, Dlai Abdellatif<sup>1,2</sup>, Jbili Nabil<sup>1,2</sup>, Bibiche Lotfi<sup>1,2</sup>, Kechna Hicham<sup>1,2</sup>, Laoutid Jaouad<sup>1,2</sup>

<sup>1</sup>Anesthesiology & intensive care department, Moulay Ismail Military Hospital, Meknes, Morocco

<sup>2</sup>Faculty of Medicine, Sidi Mohamed Ben Abdellah University, Fes, Morocco

DOI: [10.36347/sjmcr.2021.v09i04.045](https://doi.org/10.36347/sjmcr.2021.v09i04.045)

| Received: 18.03.2021 | Accepted: 25.04.2021 | Published: 30.04.2021

\*Corresponding author: Jebbar Nourddine MD

### Abstract

### Case Report

Primary hyperparathyroidism is quite common. The most forms are now seen in women after menopause and appear to have little evolution. Support surgery is imperative for primary hyperparathyroidism occurring before age 50, and in symptomatic forms, especially in cases of bone loss.

**Keywords:** Parathyroid, Delirium Revealing.

Copyright © 2021 The Author(s): This is an open-access article distributed under the terms of the Creative Commons Attribution 4.0 International License (CC BY-NC 4.0) which permits unrestricted use, distribution, and reproduction in any medium for non-commercial use provided the original author and source are credited.

## INTRODUCTION

Primary hyperparathyroidism (HPT) has long been considered like a rare disease, responsible for pain, pathological fractures, and resulting in appalling deformations. The severity of the disease was further aggravated by the nephrological risk complicating recurrent kidney stones.

Primary hyperparathyroidism is now a disease frequent: its incidence has been estimated at 27 new cases per 100,000 inhabitants in New York [1], its prevalence reached 1 in 1,000 subjects [2].

Affection predominates now largely in women after the age of menopause, this explained by the revealing effect of estrogen deficiency on osteoblastic activity.

In this population in particular, the majority of the forms observed are asymptomatic, fortuitously discovered by the measurement of calcium. Surgery, on the other hand, remains essential in young subjects and in symptomatic and progressive forms.

It is in these circumstances particularly justified by a double investigation etiological and morphological. Recognition of predisposition genetics and the exact location of pathological parathyroids now modulate the practical modalities of intervention and monitoring.

## CASE REPORT

This is a 69-year-old patient with a history of type 2 diabetes on metformin and gout on allopurinol, admitted on February 25<sup>th</sup> 2021 in intensive care for afebrile disorder of consciousness. The onset of symptoms dates back to February 15<sup>th</sup> 2021 with the onset of abdominal pain with incoercible vomiting. The evolution was marked by the installation of a disturbance of consciousness and the patient was admitted to us for treatment. On admission the patient was confused with a Glasgow score of 10/15, brainstem reflexes present with equal and responsive pupils. Hemodynamically, the patient had a blood pressure of 120/76 with a heart rate of 89bpm. The patient was eupneic and 98% saturated with ambient air. A brain scan was performed returning normal. A biological assessment objectified a malignant hypercalcemia at 177mg / l, a parathyroid hormone level at 899pg / ml, natremia at 144mmol / l and renal failure with creatinine at 25mg / dl. An electrocardiogram showed a shortening of the QT space. The patient was put under rehydration with 9% saline serum (500CC / 4 hours), bisphosphonate, furosemide (20mg / 12H), gastric protection, anticoagulation preventive dose, corticosteroids and vitamin D. The evolution was marked by a slight improvement on the neurological level with decrease in the corrected calcium level (81 mg / l) and the level of parathyroid hormone (147 pg / ml) with the appearance of hypernatremia at 160 mmol / l; the patient was placed under tap water with correction of the natremia at the end of 48 hours. A cervical ultrasound was performed objectifying a large right parathyroid nodule measuring 24mm for which the patient was operated. Pathological examination revealed that it was a parathyroid adenoma (Figure 1).

## DISCUSSION

### Pathophysiology

Parathyroid hormone elevates serum calcium, increasing the bone remodeling and predominantly osteolysis, by promoting renal tubular reabsorption of calcium and increasing intestinal absorption of calcium; this last action is indirect, is carried out by activating the 1 $\alpha$ ,25-(OH)<sup>2</sup> cholecalciferol. Parathyroid hormone is phosphodiuretic and reduces renal elimination of H ions<sup>+</sup>. Hyperparathyroidism hypercalcemia primary coincides with hypophosphatemia, a tendency to hyperchloraemic acidosis and urine alkalization. Despite increased tubular reabsorption of calcium, high calciuria, due to the increased fraction filtered by the

glomerulus. The severity of primary hyperparathyroidism is not simply linked to increased functional parenchymal mass. It is also a function of the sensitivity of the parathyroid cells to feedback control by calcemia, and the degree at this level calcium receptor expression [2]. At the initial stage, hypercalcemia is primarily the result of increased reabsorption tubular calcium, and not resorption, as during other situations of hypercalcemia of non-parathyroid origin [2]. Finally, an important factor in the expression of hyperparathyroidism primary is constituted by the vitamin load D. The relative deficiency of vitamin D indeed leads to defect absorption of calcium from the intestine, promotes increased PTH levels, and overall increases the severity of bone damage [3].

Clinical manifestations: table I

**Table-I: Primary hyperparathyroidism: revealing manifestations (In JL Wemeau Horm Res 1989) in a Lille series of 622 operated patients (4)**

symptoms	Number of cases	%
Renal lithiasis	160	25,7
Bone pain	57	9,2
Fractures	4	0,6
Peptic ulcers	8	1,3
Acute pancreatitis	7	1,1
Arterial hypertension	10	1,6
Rythm disturbances	3	0,6
Renal failure	8	1,3
Asthenia	30	4,8
Polyuria	4	0,6
Acute hypercalcemia	40	6,4
Other signs : determination of calcemia	57	9,2
Preoperative assessment of a goiter	62	10
Systematic dosage	168	27

Causes: table II (5)

**Table-II: Causes of hypercalcemia**

**Primary hyperparathyroidism (45% of cases) :high or normal PTH**

**Hypercalcemia linked to cancers (45% of cases):reduced PTH**

Metastase of osteophilic cancers or bone localizations of hemopathies (myeloma).

Malignant humoral hypercalcemia : production of hypercalcemic factors: PTHrP, interleukin and prostaglandin

**Other causes**

Sarcoidosis

Hyperthroids, especially in children at the start of treatment

Acute adrenal insufficiency

Prolonged immobilization

Syndrome of milk and alkali drinkers

Poisoning by vitamin D and vitamin A

Syndrome of Williams et Beuren ( children)

## Treatment

In any patient suspected of primary HPT, before any investigation morphological in search of the location and before any project surgical. It seems important to assess in all patients the vitamin D load, by the systematic measurement of 25-hydroxycholecalciferol. Vitamin D deficiency, especially by default intake and sunshine, particularly sensitive in patients with pigmented skin and in the northern regions, in fact profoundly modifies the diagnostic presentation and progressive disease.

Patients are exposed to elevation higher PTH level, at a high risk of demineralization and also to severe and prolonged hypocalcemic crises after surgical cure of the disease. It appeared that supplementation of these subjects deficient by physiological doses of vitamin D under biological monitoring, reduces the rates of PTH, improves bone mass, with little risk of increase calcemia or calciuria. Finally we must discuss with patients the merits either intervention in principle or abstention immediate surgery, at the cost of strict monitoring [5].



**Fig-1**

## REFERENCES

1. Heath H 3rd, Hodgson SF, Kennedy MA. Primary hyperparathyroidism. Incidence, morbidity, and potential economic impact in a community. *N Engl J Med.* 1980; 302:189-93.
2. Houillier P, Maruani G, Eladari D, Paillard M. Hyperparathyroïdie primitive. *Encl Méd Chir* (Editions Scientifiques et Médicales Elsevier SAS, tous droits réservés), *Endocrinologie-Nutrition*, 10-012-B-10, 2002, 9 p.
3. Silverberg SJ, Shane E, Dempster DW, Bilezikian JP. The effects of vitamin D insufficiency in patients with primary hyperparathyroidism. *Am J Med.* 1999;107:561-7.
4. Wémeau JL, Gilliot-Valtille E, Bizard JP. Current concepts in primary hyperparathyroidism. *Horm Res.* 1989; 32:93-6.
5. Clinique Endocrinologique Marc Linquette, CHU de Lille, 6 rue du Professeur Laguesse, 59037 Lille cedex, France. Correspondance : J-L Wémeau,