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Secondary Hyper Parathyroidism, a Typical Mineral and Bone Disorder in a Chronic Hemodialysis Patient in Ziguinchor, Southern Senegal

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Abstract

Mineral bone disorders (MBD) are almost constant complications in chronic hemodialysis patients. They cause an impairment in the quality of life and increase in cardiovascular morbidity and mortality. We are introducing the case of a 54-year-old woman, who was chronically on hemodialysis for 6 years, with a radial arteriovenous fistula as vascular access. Initially, the patient was reported having chronic tubulointerstitial nephritis. She was on hemodialysis three times a week. She developed secondary hyperparathyroidism. Clinically, she had diffuse arthralgias, bone pain mainly in the pelvis impeding walk; all this in a context of relative functional limitation of the lower limbs. As a result of paraclinical examination, serum calcium was 72 mg/l, phosphatemia was 42 mg/l. PTH returned to 2358 µg/ml and vitamin D tested using 25-OH-D was 20 mg/ml. Standard radiographs showed multiple geodes at the shoulder, lower extremity of the radius, trapezius, scaphoid, proximal phalanx head, spine and bilateral fracture lines of the femoral neck. Our patient was treated with calcium carbonate (e.g. Calcidia, in sachet), calcium-free phosphate binders (e.g. Renagel) and calcimimetics (e.g. Mimpara). Under medical treatment, there was a normalization of PTH and a decrease or even disappearance of the symptoms. This case shows that medical treatment for secondary hyperparathyroidism on hemodialysis patients, especially with calcimimetics, the use of which in our context is limited due to lack of availability.

Keywords: *Secondary hyperparathyroidism, hemodialysis, calcimimetics, Ziguinchor ,sub-Saharan Africa.*

Introduction

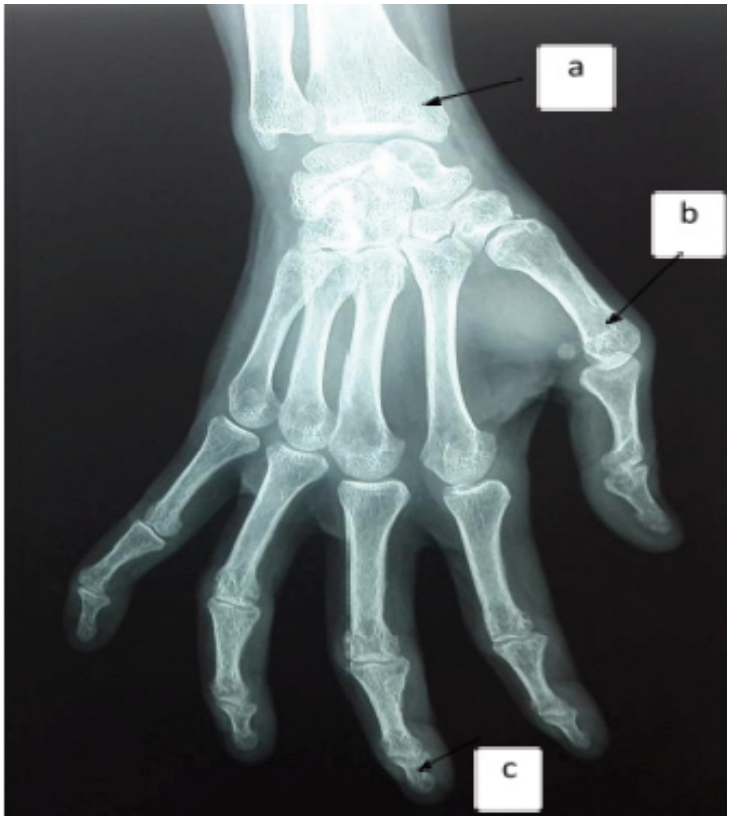
In chronic hemodialysis (CHD), mineral and bone metabolism disorders (BMD) are early in chronic kidney disease, and progressively get worseover time [1]. Impaired phosphate excretion, reduced vitamin D activation, and compensatory increase in PTH secretion define secondary hyperparathyroidism, observable in most patients with advanced chronic renal failure [2]. KDIGO (Kidney Disease: improving Global Outcomes) has recently suggestedamore broadly defined term, i.e. "chronic kidney disease-mineral disorders" (CKD-MDB). [3]. Secondary hyperparathyroidism (PTH II) affected by hyperphosphoremia is part of renal osteodystrophy and is associated with an increased risk of cardiovascular mortality [4]. This clinical case highlights the difficulty of managing BMD in the situation of our developing country where biological and radiological explorations are expensive and not subsidized, in addition to the products used for

treatment such as calcimimetics,hardly available on the local market. Our patient had the financial means that allowed her to take paraclinical examinations and get the therapeutic molecules, most often imported.

Clinical Observation

A 54-year-old woman received chronic kidney dialysis treatment at the hemodialysis center of the Ziguinchor Regional Hospital, 450 km away from Dakar, for 6 years, with a radial arteriovenous fistula for vascular access. Initially, the renal diseasewas supposed to be chronic tubulointerstitial nephritis. She was on a three sessionsper week. She had mainly a story of heart disease with features of left heart failure. There was no previous aluminum toxicity. The diagnosis of hyperparathyroidism was established within 5 years after starting dialysis.

The blood pressure was stable (120/85 mmHg)when the clinical examination was carried out. There had no fever. She had arthralgia in her hips, knees, and right shoulder. This was associated with widespread bone pain mainly at the level of the hip which restricted walk performance with the effect of relative periodical loss of limb function. The osteo-articular examination evidenced a limitation of movements and bone damage in the right wrist and pelvis. The biological assessment: serum calcium level was 82 mg/l (normal serum level between 85 and 105 mg/l), phosphatemia was 42 mg/l (normal level between25 and 45mg/l) The intact PTH 1-84 (parathyroid hormone) decreased to 2358 pg/ml (normal level between is 6 and 50 pg/ml) and the25 (OH) vitamin D concentration was 20 mg/ml (normal level is between 30 and 40 ng/ml). Hemoglobin was 11 g/dl erythropoietin 15,000 IU per 15 days. C reactive protein (CRP) was negative. Standard radiographs showed multiple bone cysts at the shoulder, lower extremity of radius, trapezoid, scaphoid, head of the thumb proximal phalanx and at the level of the spine, and bilateral stress fracture of the femoral necks (figure 1,2).



a.*Cystic bone lesion at the lower end of the radius*

b. *Geode in the proximalphalanx of the thumbProximal phalanx geode of the thumb*

c. *Sub-periosteal resorption of distal phalanges*

Fig 1. *Standard radio of our patient'sright hand, showing macrogeodes at the lower extremity of the radius, trapezius, scaphoid and proximal phalanx of the dystrophic thumb.*

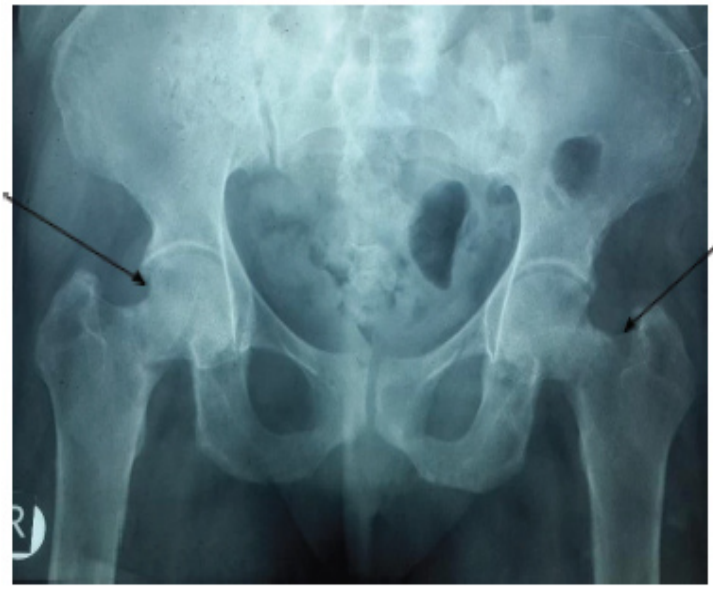


Fig 2. *Standard radiographic view of a pathological bilateral fracture of the femoral necks in our patient.*

The cervical ultrasound was eventless. Parathyroid scintigraphy was back to normal again.

Therapeutically, our patient was treated with calcium carbonate (Calcidia *: 1 sachet in the morning), a non-calcium-based phosphatebinder sevelamer (Renagel * 800 mg: 1 tablet three times a day) and cinacalcet, acalcimimetic agent (Mimpara * 60 mg: 1 tablet twice daily and then dose adjustment according to the effects of PTH 1-84). In dialysis, she was submitted tohigh dialysate calcium concentration. Under medical treatment, there was a gradual decrease in PTH after 4-monthmonitoring. At 10 months of uninterrupted treatment, the biological parameters were returning to normal, with a decrease or even disappearance of the symptoms.

Comment

Secondary hyperparathyroidism can threaten life and functional prognosis in hemodialysispatients as the results ofcardiovascular and osteoarticular complications [5,6].The prevalence of secondary hyperparathyroidism (HPT) is significant in developing countries, in the Ndiaye's series with HPT in 45.5% of cases, in the different series of the literatureas well, where the levels were between 30% and 60% on average. Studies by Llach et al [7], Sherrad et al. [8], Jebrane et al. [9], Seck et al. [10] and Benabdellah et al. [11] found a prevalence of 47.88%, 32.47%, 48.71%, 48% and 56.7% respectively (Table I).

High occurrence of the disease in our countries could easily be explained by the low socioeconomic level of our patients, who lack social coverage in most of cases. Thismakes a therapeutic treatment based on non-calcium phosphate binders and/or calcimimetics often difficult, or even unthinkable, given their high cost. Our patient had a high living standard which explained exceptional good care.

Table 1. *Prevalence of HPT II in dialysis patientsas part of the literature series*

Authors	Years	Number of dialysis patients	HPT II	Prevalence
Llach et al. [7]	1986	142	68	47.88 %
Sherrard et al. [8]	1993	117	38	32.47 %
Ndiaye [12]	2008	40	14	45,5%
Guillaume et al. [13]	2010	502	158	30 %
Jebrane et al. [9]	2012	39	19	48.71 %
Seck et al. [10]	2012	118	57	48 %
Benabdellah et al. [11]	2013	83	47	56.7 %

Our patient was 54 years old, which was consistent with the results found by Jebrane et al. (40.4 ± 10.6 years) [9], Montasser et al. (46 ± 15 years) [14], and Cherkaoui et al. (45 ± 10 years) [15].In our case, HPT was diagnosed 5 years after onset of hemodialysis. This delay was similar to the finding by Montasser et al. (4 ± 2 years) [14] and Cherkaoui with 5 ± 4 years [15].

Traore et al. [16] found that 81.7% of patients were asymptomatic and 7.5% had bone pain and 5.3% muscle pain.However,in a recent series by Haddam et al. in Algeria (2015) [17], all patients had a clinical bone syndrome.Fibrous osteitis remains asymptomatic for a long time, bone pain usually occurs late. In our patient, we observed a high frequency of joint and bone pain, probably due to severity of the infections as well as the delay in these patients' care.

We found a very high levels of PTH, up to 2358 pg/ml (36 N), indicating a lack of adjustment and down-regulation in our work. These figures are significantly higher than those found in the Traoré et al. [16], Benabdellah et al. [11], Haddam et al. [17] series, with respectively 436.11 ± 200 µg/mL, 508 + -380 µg/ml and 680 µg/mlof PTH on average.

The high parathyroid hormone (PTH) concentration in our patient could be explained by the fact that there are currently different kits for determining PTH levelavailable on the market, with a wide range in the values obtained, as well as variationin defining hyperparathyroidism,depending on the series.

The dialysis reference values are 2 to 9 times the upper limit of the kit used (approximately 130-585 pg/ml for a 2ndgeneration assay).

Drug therapy for secondary hyperparathyroidism involves calcium, phosphate binders, calcitriol derivatives, and calcimimetics (cinacalcet Mimpara®). Our patient receivedthe same type of treatment with a favorable evolution. In France, in the series by Guillaume et al., 51% of patients were treated with cinacalcet, of which 10% could be weaned after 12 months [13].

The modest use of calcimimetics in our center (in a patient), due to cost and lack of medical insurance in most of our patients, explains the fact that only our case had acontrol of hyperparathyroidism with PTH kept within the recommended targets for hemodialysis.

Conclusion

Secondary hyperparathyroidism is a common complication of chronic kidney failure. The care in sub-Saharan Africa is very restrictive, sometimes expensive. However, it still represents a challenge despite the recommendations of learned societies and knowledge of physiopathological mechanisms.

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