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### Early-Onset Craniofacial Brown Tumor Progressing to Multifocal Skeletal Lesions in a Peritoneal Dialysis Patient: A Case Report and Literature Review

(Tumeur brune cranio-faciale d'apparition précoce évoluant vers des lésions squelettiques multifocales chez un patient sous dialyse péritonéale : rapport de cas et revue de la littérature)

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#### Summary

**Background:** Brown tumors (BTs) are rare skeletal manifestations of osteitis fibrosa cystica caused by prolonged hyperparathyroidism. They are most commonly reported in patients with advanced secondary hyperparathyroidism undergoing hemodialysis. Conversely, early presentation in peritoneal dialysis (PD) remains uncommon, and craniofacial involvement is especially rare.

**Case Presentation:** We report a case of a 27-year-old woman with end-stage renal disease on PD who developed a craniofacial BT shortly after dialysis initiation. A biochemical evaluation revealed markedly elevated parathyroid hormone (PTH) levels (3032 pg/mL). Despite initial partial regression, the BT rapidly progressed to multifocal skeletal lesions, including on the clavicle, humeral head, ribs, and ischiopubic ramus, associated with fractures and tumoral calcinosis. Medical management was initiated, and parathyroidectomy was planned.

**Conclusion:** This case emphasizes the potential for early craniofacial BT development and rapid multifocal skeletal progression in PD patients with uncontrolled hyperparathyroidism. It further demonstrates that early detection and prompt surgical referral may be vital in preventing severe skeletal complications in PD patients with uncontrolled hyperparathyroidism.

**Keywords:** brown tumor; peritoneal dialysis; tertiary hyperparathyroidism; Chronic kidney disease–mineral and bone disorder (CKD-MBD); renal osteodystrophy; craniofacial bone lesions

#### Résumé

**Contexte :** Les tumeurs brunes (TB) sont des manifestations squelettiques rares de l'ostéite fibrosante kystique causée par un hyperparathyroïdisme prolongé. Elles sont le plus souvent rapportées chez des patients atteints d'hyperparathyroïdisme secondaire avancé sous hémodialyse, tandis que leur apparition précoce chez les patients sous dialyse péritonéale (DP) reste rare. L'atteinte cranio-faciale est particulièrement rare.

**Présentation du cas :** Nous rapportons le cas d'une femme de 27 ans atteinte d'une insuffisance rénale terminale sous DP qui a développé une TB cranio-faciale peu après le début de la dialyse. L'évaluation biochimique a révélé des taux de parathormone (PTH) nettement élevés (3 032 pg/mL). Malgré une régression partielle initiale, la tumeur brune a rapidement évolué vers des lésions squelettiques multifocales, touchant notamment la clavicule, la tête humérale, les côtes et la branche ischiopubienne, associées à des fractures et à une calcinose tumorale. Un traitement médical a été instauré et une parathyroïdectomie a été programmée.

**Conclusion :** Ce cas souligne le risque de développement précoce d'une tumeur brune cranio-faciale et de progression squelettique multifocale rapide chez les patients sous dialyse péritonéale présentant une hyperparathyroïdie non contrôlée. Une détection précoce et une orientation chirurgicale rapide peuvent être vitales pour prévenir les complications squelettiques graves chez les patients sous DP atteints d'hyperparathyroïdie non contrôlée.

**Mots-clés :** tumeur brune ; hialyse péritonéale ; hyperparathyroïdie tertiaire ; maladie rénale chronique – troubles minéraux et osseux (CKD-MBD) ; ostéodystrophie rénale ; lésions osseuses cranio-faciales



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## Introduction

Osteitis fibrosa cystica (OFC) is a rare but clinically important bone disorder marked by significant skeletal changes caused by excessive parathyroid hormone (PTH) activity [1,2]. Brown tumors (BTs), which occur in 0.1% of all hyperparathyroidism cases, are the classic histological feature of OFC and represent localized disease [3]. This condition is characterized by increased osteoclastic activity, leading to bone resorption, fibrosis, and the formation of osteoclastic clusters [4].

Typical BT skeletal sites include the ribs, pelvis, long bones, and rarely, craniofacial bones [2]. Craniofacial BTs are particularly notable due to their aesthetic, functional, and psychological implications [5]. Although reports of BTs are more common in hemodialysis patients, their occurrence in patients on peritoneal dialysis (PD), especially with craniofacial involvement, remains rare and underreported [2,5–9].

A review of the literature shows that most reported BTs typically occur after prolonged hyperparathyroidism, often several years into renal replacement therapy, and that a subset of patients exhibits multifocal skeletal involvement [3,11–17]. Early-onset BTs, particularly within months of starting PD, are exceedingly rare. Management strategies range from treating hyperparathyroidism with medical therapies such as calcimimetics, vitamin D analogs, and phosphate binders to performing surgical parathyroidectomy in refractory cases [2].

Here, we report a case of craniofacial BT occurring shortly after the initiation of PD in a patient with tertiary hyperparathyroidism (THPT). In our patient, the craniofacial BT rapidly progressed to multifocal skeletal lesions, illustrating the aggressive nature of chronic kidney disease–mineral and bone disorder (CKD–MBD) soon after starting PD.

Through an analysis of this case, this report highlights the importance of early detection, prompt biochemical monitoring, and intervention to prevent serious skeletal complications. It also references relevant literature that emphasizes the rarity and clinical importance of this presentation.

## Case Presentation

A 27-year-old woman was admitted to our department with end-stage CKD due to an unspecified chronic nephropathy and started on PD. Baseline biochemical tests before initiating dialysis showed severe secondary hyperparathyroidism. Her serum calcium was 2.3 mmol/L, serum phosphorus was 2.55 mmol/L, parathyroid hormone (PTH) was 3032 pg/mL, and 25-hydroxyvitamin D was 4.25 ng/mL. In response to these findings, she was prescribed calcium carbonate with meals. Her serum phosphorus remained high, so we were unable to use alfacalcidol to suppress her PTH levels. Six months later, she presented with worsening frontal pain and localized swelling. An ensuing physical exam revealed short stature and mild facial asymmetry without neurological deficits, and a cone-beam computed tomography showed an expansile osteolytic lesion in the frontal bone with internal trabeculation and multiple bone lacunae, consistent with a BT.

Laboratory evaluation at that time showed a serum calcium of 2.33 mmol/L, serum phosphorus of 2.85 mmol/L, and PTH of 2,600 pg/mL. Cervical ultrasonography revealed bilateral parathyroid

nodular enlargement, consistent with progression to THPT. The frontal lesion experienced spontaneous clinical and radiological regression over the following months without surgical intervention.

Approximately 18 months after starting dialysis, the patient developed acute pain and swelling in the right acromioclavicular area. A CT scan showed a pathological fracture of the distal right clavicle on a lytic bone lesion, suggestive of a BT. Additional osteolytic lesions were found in the humeral head and ribs, along with multiple rib fractures.

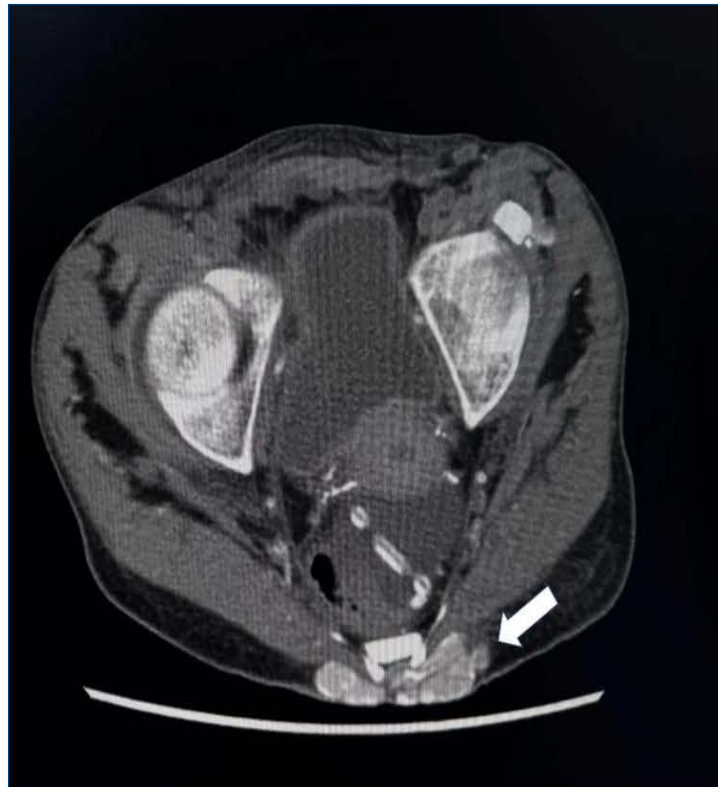
At 24 months, she presented with painless pelvic swelling. Imaging showed a pathological fracture of the left ischiopubic ramus on weakened bone, along with multiple periarticular multilobulated calcified masses consistent with tumoral calcinosis (*Figures 1, 2*).



↑ *Figure 1. Pelvic radiograph showing diffuse osteopenia and pathological fracture in the setting of advanced renal osteodystrophy*

These findings were consistent with advanced renal osteodystrophy complicated by multiple BTs and extra-skeletal calcifications. Throughout follow-up, the patient's PTH levels remained significantly elevated despite medical treatment.

Treatment options were limited because of the scarce availability of calcimimetics and non-calcium phosphate binders and because low-calcium dialysate solutions were not practical in the local PD setting. Parathyroidectomy was therefore seen as the only definitive treatment, although this was postponed due to recurrent infections and temporary surgical ineligibility.



↑ Figure 2. Axial pelvic CT showing a pathological fracture of the left ischio pubic ramus on weakened bone, associated with multilobulated periarticular calcified masses consistent with tumoral calcinosis

## Discussion

OFC is a bone disorder resulting from hyperparathyroidism, most commonly secondary hyperparathyroidism (SHPT) in patients with CKD. In SHPT, prolonged PTH elevation leads to increased osteoclastic activity, resulting in bone resorption, fibrosis, and the formation of osteoclastic aggregates, commonly known as BTs. These tumors represent a severe manifestation of OFC, arising due to excessive bone turnover driven by elevated PTH levels.

In contrast, THPT develops when SHPT persists for a prolonged period, resulting in autonomous PTH secretion by the parathyroid glands [2]. This causes ongoing hyperparathyroidism, often with hypercalcemia, which worsens bone complications, including the formation of BTs. Moreover, in patients with THPT, the chronic elevation of PTH worsens bone resorption, leading to both common and less typical tumor locations [4,18].

As noted earlier, BTs are most commonly found in the ribs, clavicles, extremities, and pelvic girdle, but they can also affect rarer sites, such as craniofacial bones. These craniofacial BTs can cause significant facial disfigurement, as seen in our patient, emphasizing the impact of hyperparathyroidism on the facial skeleton [5,19–21].

In our case, the patient developed a BT after a relatively short period on PD, making it one of the fastest-documented cases in the literature. This rapid progression likely stemmed from a long history of uncontrolled hyperparathyroidism, already established during the stage of CKD and unrelated to the dialysis treatment method. Consequently, this case emphasizes the need for

careful monitoring of CKD in order to avoid severe complications such as hyperparathyroidism, especially in patients with elevated PTH levels. It further emphasizes the importance of early intervention to prevent further complications, including BTs and other skeletal abnormalities. Additionally, considering the limited availability of the full range of treatments for hyperparathyroidism, management should be adapted to a patient's local context. For instance, in our case, non-calcium-based phosphate binders and calcimimetics were not available, and PD was limited by the availability of only one dialysate calcium concentration.

A recent systematic review of BTs showed that facial disfigurement resulting from craniofacial BTs, although rare, is a significant clinical sign. Out of 127 reported cases of severe OFC, only 27.6% appeared in nephrology journals, with most cases noted in hemodialysis patients [10]. While these tumors are uncommon, they carry substantial physical and psychological impacts. As illustrated by our patient, craniofacial BTs can cause notable facial disfigurement, having profound psychological effects in addition to affecting one's appearance. This consequences underscores the need for early diagnosis and treatment to prevent permanent damage. In our patient, although the frontal lesion initially showed spontaneous clinical and radiological regression, this early craniofacial manifestation indicated the severity of her SHPT even before PD initiation.

Although rare, our patient's lesion reinforces the importance of considering BTs in the differential diagnosis when SHPT patients show craniofacial symptoms. While this complication was not directly caused by PD, it was worsened by PD's limitations and the lack of optimal local treatment options. In our case, the lesion's increased activity reflects an osteoclastic response to uncontrolled PTH, even with dialysis. This indicates that our patient's SHPT had already become severe and autonomous before starting PD, making her management more complex.

Interestingly, Verma et al. (2014) reported a similar case of spontaneous resorption involving a 31-year-old woman with secondary hyperparathyroidism who developed a craniofacial BT. Swelling on one side of her jaw resolved spontaneously, suggesting that BTs may initially exhibit dynamic, reversible changes before becoming more destructive, as seen in our patient. This case further underscores the importance of early detection and treatment in preventing permanent bone damage.

The diagnosis of OFC and BTs is mainly based on radiological findings, supported by the clinical context of hyperparathyroidism, whether secondary or tertiary. Early detection of these complications is essential to ensure prompt treatment and to reduce their impact on the patient's quality of life [22,23] [24].

Additionally, differentiating BTs from other bone lesions, such as giant cell tumors, giant cell granulomas, and aneurysmal bone cysts, is crucial due to their similar imaging and histological features [2,19,25]. In particular, a detailed clinical history, including the presence of hyperparathyroidism and CKD, is essential for distinguishing between these conditions.

The medical management of BTs involves treating the underlying SHPT. Specifically, medical treatments, such as phosphate binders, vitamin D analogs, and calcimimetics, are used to control PTH levels [2,26]. In our case, therapeutic options were limited due to the unavailability of calcimimetics, non-calcium phosphate binders, and low-calcium dialysate solutions in the local

PD setting, which contributed to the worsening of the patient's condition.

Should the above medical management prove ineffective, surgical options, such as total or subtotal parathyroidectomy, can be considered [27]. In this case, parathyroidectomy was ultimately viewed as the only definitive treatment for the patient's severe SHPT, as it would likely cause the tumors to regress. Delaying parathyroidectomy in cases of severe uncontrolled hyperparathyroidism can lead to aggressive and irreversible skeletal complications.

*Table 1* below summarizes key cases of BTs in PD patients from the literature, illustrating variations in patient demographics, tumor locations, and management strategies. As shown, BTs can affect various regions of the body, and the duration of PD and PTH levels are critical factors influencing disease progression.

↓ *Table 1: Reported cases of brown tumors in patients undergoing peritoneal dialysis*

Author (Year)	Age (years)	Gender	PD (years)	Brown Tumor Location	HPT Type	PTH (pg/mL)	Management and Outcome
Dursun et al. (2005) [16]	8	F	5	Ribs	Secondary	3965	Intensified dialysis and IV calcitriol; parathyroidectomy refused
Kaya et al. (2007) [15]	72	M	4	T1 vertebra	Secondary	NR	Surgical decompression and spinal reconstruction; neurological recovery at 1 year
Raubenheimer et al. (2015) [12]	31	F	13	Mandible	Secondary	2026	Medical stabilization; planned mandibular recontouring; awaiting transplantation
Fernandes et al. (2016) [14]	22	F	9	Proximal femur; distal femur; proximal tibia	Secondary	1118.9	Prophylactic femoral fixation; medical HPT management
Stârcea et al. (2018) [17]	13	M	5	Hard palate; craniofacial bones	Secondary	1602	Medical therapy; parathyroidectomy refused; fatal outcome
Medina-Castillo et al. (2021) [3]	29	F	1	Finger; knee; clavicles; supraclavicular region; wrist; elbow; sternum	Tertiary	2347.6	Parathyroidectomy; marked biochemical improvement with radiological regression
Luzuriaga et al. (2021) [11]	65	M	NR	Proximal ulna; multifocal axial and appendicular skeleton	Secondary	900	Medical therapy; significant PTH reduction; persistent inactive lesions
Campinho Ferreira et al. (2025) [13]	42	F	4	Cervicothoracic spine	Tertiary	NR	Parathyroidectomy with surgical tumor resection
Present case	27	F	0.5	Frontal bone; clavicle; humeral head; ribs; ischiopubic ramus	Tertiary	3032	Initial spontaneous regression followed by multifocal progression with fractures and tumoral calcinosis; persistent severe HPT; parathyroidectomy planned

In our patient, the initial frontal BT was followed by progressive multifocal bone involvement, including a pathological clavicular fracture, rib fractures, and tumoral calcinosis, further exhibiting the destructive potential of BTs when a patient's PTH levels remain uncontrolled despite medical treatment. Notably, the frontal lesion initially showed spontaneous clinical and radiological regression, indicating a dynamic phase before progressing to more damaging lesions, as evidenced by subsequent skeletal complications.

### **Conclusion**

Although BTs represent a rare and often delayed complication in PD patients, this case highlights their potential for early appearance and rapid progression, especially when a patient's PTH levels are uncontrolled. The literature review provided here further enhances our understanding of the development and management of BTs in dialysis patients. In conjunction, this literature review and presented case demonstrated BTs' clinical significance and the importance of early management of CKD to avoid severe complications, which is crucial to limit end-organ damage and prevent further issues such as OFC and the challenges posed by limited treatment options in certain settings.

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### **Authors' Contributions**

- Conceptualization: *Dr hanène Gaied and Dr Sarra Hadded*
- Methodology: *Dr Hanene Gaied and Dr Marwa Trabelsi*
- Writing - Original Draft: *Dr Marwa Trabelsi and Dr Yasmine Thabti*
- Writing - Review & Editing: *Dr Mariem Khadhar and Dr Rim Goucha*
- Supervision: *Dr Hanène Gaied*

### **Ethical considerations**

*This study was conducted in accordance with the ethical principles of the Declaration of Helsinki.*

### **Artificial intelligence**

*The authors declare that this manuscript is the result of their own original work. No artificial intelligence tools or applications were used for data analysis, the generation of results, or the creation and drafting of the text.*

### **Patient consent**

*Informed consent was obtained from the patient for the publication of this case report and all accompanying images.*

### **Conflicts of Interest**

The authors declare no conflict of interest.

### **Data availability**

The datasets generated and analysed during this study are not publicly available due to institutional regulations and legal restrictions protecting patient privacy and data confidentiality.

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