Hypercalcemia and Suppressed Intact PTH in a Hemodialysis Patient

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Case Description

A 54-year-old woman, with a past medical history of hypertension, obesity, multinodular goiter, and hemorrhagic stroke with persistent left-sided hemiparesis, was evaluated at our nephrology outpatient clinic due to stage 5 CKD. She was treated with calcitriol due to severe hyperparathyroidism (intact parathyroid hormone [iPTH] 2147 pg/ml), with serum calcium (9.1 mg/dl) and phosphorus (3.8 mg/dl) within the normal range.

Two months later, she presented to the hospital with uremic syndrome and thus started hemodialysis, remaining dialysis dependent thereafter. At admission, calcitriol treatment was continued. This hospital admission was prolonged due to various complications, including fever of unknown origin. One month later, she developed severe hypercalcemia (ionized calcium 2.2 mmol/L, serum calcium 15.1 mg/dl), which did not improve, even after discontinuing calcitriol. At that time, iPTH was suppressed (71 pg/ml), whereas serum phosphorus and alkaline phosphatase remained within the normal range. Common causes of hypercalcemia (with and without fever), including multiple myeloma, sarcoidosis, tuberculosis, and hyper-vitaminosis D (calcitriol levels were low: 2.6 pg/ml), were ruled out.

Bone scintigraphy was performed to exclude osteolytic lesions, and it disclosed exuberant multiple soft-tissue calcifications (Figure 1). The patient later developed painful skin ulcerations on the lower extremities, and a skin biopsy revealed calcific uremic arteriolopathy (CUA) (Figure 2). The possibility of undertaking a bone biopsy was considered, but the patient refused it.

Serum calcium remained elevated, despite increased frequency of hemodialysis and low dialysate calcium concentration. Although we were unable to exclude adynamic bone disease, the patient was treated with monthly pamidronate due to refractory hypercalcemia, resulting in progressive calcium normalization. We speculate that hypercalcemia may have been caused by prolonged immobilization in a patient with previous motor impairment who had been hospitalized and mostly bedridden for a long period of time. In this setting, the bisphosphonate may have helped reverse the rapid bone reabsorption enhanced by immobilization. iPTH suppression was probably secondary to severe hypercalcemia.

Bone scintigraphy was repeated 1 year later, revealing significant improvement in the soft-tissue calcifications (Figure 3). By then, the skin lesions were also fully epithelized.

Figure 1. | First bone scintigraphy. Exuberant soft-tissue calcifications and diffuse slight bone uptake.

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In this case, extensive soft-tissue calcification was an unexpected finding diagnosed by the bone scintigraphy. The tracer principle of nuclear medicine can be used in advanced CKD to evaluate hyperparathyroidism by detecting soft-tissue calcifications. This permits detection of CUA by taking advantage of the uptake of the bone-seeking radiopharmaceutical by the soft-tissue calcifications (1,2).

CUA is a rare but potentially devastating condition characterized by painful skin lesions and caused by cutaneous arteriolar calcification. It leads to tissue ischemia and infarction, and it is associated with increased cardiovascular morbidity and mortality (3–5). Usually, the diagnosis is confirmed through a skin biopsy. Yet, it may be contraindicated in some cases (e.g., when a superimposed infection is suspected). Thus, scintigraphy can be an alternative test to detect and quantify tissue calcifications in patients with suspected or confirmed CUA.

In sum, we illustrate the importance of its diagnosis and treatment. This case stands out for the rare use of scintigraphy to identify soft-tissue calcification in CKD patients with suspected CUA.

**Teaching Points**

- Scintigraphy is rarely used to identify and quantify soft-tissue calcifications in patients with suspected CUA.
- Dialysis optimization, sodium thiosulfate, and control of calcium, phosphorus, and iPTH levels (avoiding calcium-based phosphate binders) are the cornerstones of CUA treatment.

**Disclosures**

M. Raimundo holds stocks in Abbvie, Alpine Income Property Trust, Energy Transfer, Essential Properties Realty Trust, Inc., Micron Technologies, Omega Healthcare Investors, and STAG Industrial, Inc.; and reports an advisory or leadership role as a member of Hospital Beatriz Angelo’s scientific commission, a member of the Portuguese Society of Nephrology scientific commission, and a member of the nephrology committee of the “Ordem dos Médicos.” All remaining authors have nothing to disclose.

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**Author Contributions**

B. Donato wrote the original draft of the manuscript, and M. Raimundo and R. Veiga reviewed and edited the manuscript.

**References**


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