

Calcified Clues: Incidental Discovery of Secondary Hyperparathyroidism

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Background

Hyperparathyroidism (HPT) results from excessive secretion of parathyroid hormone (PTH). It is diagnosed and categorized as either primary, secondary, or tertiary based on characteristic patterns in PTH, calcium, vitamin D, and phosphate levels (Table 1). Secondary hyperparathyroidism (SHPT) is characterized by hypocalcemia, hyperphosphatemia, and low vitamin D.¹ SHPT commonly occurs with chronic kidney disease (CKD), with prevalence increasing as kidney function declines.^{3,6} CKD is classified into five stages, which are determined by estimated glomerular filtration rate (eGFR), with higher stages corresponding to progressively lower eGFR and more advanced kidney disease. Given the established association between CKD and SHPT, current guidelines recommend monitoring patients with CKD stage III and above for electrolyte abnormalities and bone mineral disorders. This involves obtaining a basic metabolic panel, serum albumin, phosphate, and PTH levels at varying intervals depending on stage.¹ Generally, laboratory evaluations are necessary every 6-12 months for CKD stage III, every 3-5 months for CKD stage IV, and every 1-3 months for CKD stage V.¹ Screening for low vitamin D levels has also been suggested, as vitamin D deficiency in CKD may be related to albuminuria, progression

of CKD, and increased mortality.³ This report presents a unique case in which a hospitalization for gastrointestinal bleeding prompted further work up, ultimately leading to the incidental discovery of extensive metabolic derangements in a patient with a history of CKD stage IV.

Case Presentation

A 57-year-old woman with a history of CKD stage IV, right staghorn calculus status post nephrostomy tube placement, recurrent urinary tract infections, chronic hyponatremia, and chronic back pain presented with generalized weakness and melena. Her initial workup revealed a duodenal erosion, which her care team treated appropriately. During her inpatient admission, she experienced significant constipation and nephrostomy site pain, prolonging her recovery.

Investigation

A non-contrast CT of her abdomen and pelvis revealed a right staghorn calculus, multiple non-obstructing gallstones, healing rib and sacral fractures, a chronically calcified pancreas, extensive diffuse vascular calcifications, moder-

Table 1. Typical Laboratory Patterns in Hyperparathyroidism (HPT)

	Primary HPT	Secondary HPT	Tertiary HPT
PTH	High	High	High
Calcium	High	Low	High
Vitamin D	Normal	Low	Low
Phosphate	Normal	High	High

ate stool burden, and calcium-phosphate deposition in the soft tissue of her right buttock consistent with tumoral calcinosis. These findings, in conjunction with her history of CKD, prompted review of prior laboratory data (Table 2). Several months earlier, the patient's lab results displayed various abnormalities, including a significantly elevated PTH, low vitamin D, and elevated phosphate. At that time, the patient was lost to follow-up. Repeat testing during the current admission revealed that the patient's PTH had continued to increase, now reaching approximately 50 times the upper limit of normal. Her calcium, vitamin D, and phosphate had all since declined, although the patient's phosphate level remained above the reference value. A sestamibi scan to study the patient's parathyroid glands identified a nonfunctional parathyroid adenoma.



Figure 1. Abdominopelvic CT Coronal View. This image reveals stones present within the gallbladder. Calcifications are present within the pancreas and throughout the vasculature.



Figure 2. Pelvic CT Transverse View. In this image you can appreciate the tumoral calcinosis present in the patient's right gluteal region.

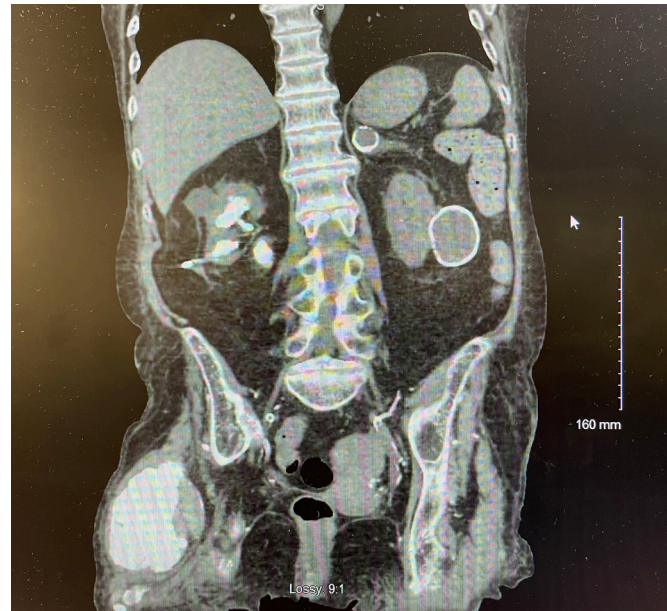


Figure 3. Abdominopelvic CT Coronal View. This image displays calcifications present within the right kidney and throughout the vasculature. Right gluteal region reveals tumoral calcinosis.

Table 2. Patient Laboratory Values

	Prior Admission	Current Admission	Reference Range
PTH (pg/mL)	2271	3261	10 – 65
Calcium (mg/dL)	9.2	8.3	8.6 – 10.3
Vitamin D (ng/mL)	15	< 8	20 – 40
Phosphate (mg/dL)	8.9	6.7	2.5 – 4.5

Treatment

The endocrinology team diagnosed the patient with secondary hyperparathyroidism due to combined CKD and vitamin D deficiency. They initiated sevelamer, a phosphate-binding medication, and vitamin D supplementation to correct her profound disturbances in calcium-phosphate homeostasis.

Discussion

An estimated 74.7-80% of patients with CKD stage IV patients have concomitant SHPT, contributing to bone demineralization, high bone turnover, and extraskeletal calcification.⁶ These manifestations are reflected in this patient's vascular, pancreatic, and soft tissue calcifications. Chronic derangements in calcium and vitamin D associated with CKD trigger sustained secretion of PTH.⁵ This continued overproduction, combined with impaired renal clearance, may lead to a progressive rise in serum PTH. Typically, the gradual escalation of this hormone level takes months to develop.⁵ In the case of this patient with PTH levels greater than 3000 pg/mL, this metabolic dysregulation likely progressed unchecked for several years. Initial management involves treating vitamin D deficiency with either nutritional supplementation or active analogs and controlling hyperphosphatemia through dietary phosphate restriction and phosphate binder therapy.^{2,4}

Vitamin D analogs, such as paricalcitol, are reserved for patients with CKD stages IV-V whose hyperparathyroidism continues to worsen.² If patients progress to CKD stage V, the goal is to maintain PTH levels between 130-585 pg/mL.¹ For those on dialysis who are unable to control PTH levels, treatment with calcimimetics, such as cinacalcet, can be initiated.^{2,4} This pharmacotherapy can significantly reduce PTH levels, with a mean decrease of 68%.² However, phosphate binders and calcimimetics have not shown significant impact regarding cardiovascular disease or mortality, particularly in non-dialysis patients.⁵ The presence of refractory symptoms may necessitate parathyroid gland removal, which has been associated with 15-57% increased survival.⁵ Uncontrolled SHPT

negatively impacts patients' quality of life and is associated with increased cardiovascular morbidity and mortality, greater fracture risk, and progression of CKD.^{4,6} This case highlights the importance of routine monitoring in patients with CKD, allowing for early intervention and hopefully increased quality of life.

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