Journal of Neonatal Surgery
ISSN(Online): 2226-0439
Vol. 14, Issue 26s (2025)
https://www.jneonatalsurg.com

OPEN ACCESS

Secondary Hyperparathyroidism Due To Hypocalcaemia and Vitamin D Deficiency in A Young Adult with Chronic Kidney Disease: A Case Report

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Cite this paper as: Francesca Melissa Marsha Pareira, Sony Wibisono Mudjanarko, (2025) Secondary Hyperparathyroidism Due To Hypocalcaemia and Vitamin D Deficiency in A Young Adult with Chronic Kidney Disease: A Case Report, *Journal of Neonatal Surgery*, 14 (26s), 751-755

ABSTRACT

Background: Secondary hyperparathyroidism (SHPT) is a common and significant complication in patients with chronic kidney disease (CKD), especially in advanced stages. It is triggered by disturbances in calcium, phosphate, and vitamin D metabolism, leading to parathyroid hyperplasia and overproduction of parathyroid hormone (PTH) [1]. This case report describes an 18-year-old female with stage V CKD, who developed SHPT associated with hypocalcaemia and vitamin D deficiency. In addition to her kidney disease, the patient had growth hormone (GH) deficiency, which complicated the clinical picture.

Case Presentation: The patient, an 18-years-old high school student, presented with symptoms of fatigue, difficulty walking, and persistent lower back pain. Laboratory findings revealed severe hypocalcaemia, elevated PTH, and vitamin D deficiency. Radiological imaging confirmed the presence of bilateral parathyroid adenomas and evidence of renal osteodystrophy. Growth hormone deficiency was diagnosed based on laboratory values, which may have contributed to her worsening kidney function. Initial therapy with calcium supplementation and vitamin D analogs resulted in marked improvement in biochemical markers and symptoms.

Conclusion: This case highlights the importance of early diagnosis and comprehensive management of secondary hyperparathyroidism in patients with CKD, especially in young adults. Additionally, the potential interplay between CKD and growth hormone deficiency requires further research to guide optimal treatment strategies.

Keywords: *Secondary hyperparathyroidism; chronic kidney disease; growth hormone deficiency; hypocalcemia; vitamin D deficiency; parathyroid hormone; renal osteodystrophy; mineral bone disorder*

1. INTRODUCTION

Chronic kidney disease (CKD) is a progressive condition characterized by a gradual decline in kidney function over time [2]. As kidney function deteriorates, patients experience various metabolic disturbances, including imbalances in calcium, phosphate, and vitamin D metabolism [1]. One of the most common and clinically significant complications of CKD, particularly in its advanced stages, is secondary hyperparathyroidism (SHPT). This condition is caused by elevated levels of parathyroid hormone (PTH) in response to abnormalities in calcium and phosphate levels, often exacerbated by insufficient activation of vitamin D due to impaired renal function [3].

The consequences of SHPT are far-reaching, contributing to renal osteodystrophy, vascular calcification, and other metabolic disturbances associated with CKD [2]. One of the key factors complicating CKD is the co-occurrence of growth hormone deficiency (GHD), which can further exacerbate the metabolic dysfunction seen in CKD patients due to its role in adapting

calcium homeostasis with bone growth in adolescents [4].

This report focuses on an 18-year-old female patient with stage V CKD who developed SHPT due to hypocalcaemia and vitamin D deficiency, compounded by growth hormone deficiency (GHD). The interplay between CKD and GHD, and how one may influence the other, is explored in this case

Case Report

An 18-year-old female high school student from Sidoarjo, Indonesia, presented to Dr. Soetomo General Hospital in Surabaya, East Java, Indonesia, with complaints of fatigue, difficulty walking, and persistent lower back pain. These symptoms had been gradually worsening for several months. She first noticed epigastric pain and nausea, which persisted despite outpatient treatment. Over the subsequent months, her symptoms escalated, and she developed difficulty walking, general weakness, and muscle pain. Her medical history was significant for chronic kidney disease (CKD) diagnosed around a year prior to admission after laboratory results revealed elevated serum creatinine levels (6-9 mg/dL). She was diagnosed with stage V CKD but had not yet begun hemodialysis. Over the following months, she was treated with erythropoietin (EPO) every two weeks, but her symptoms continued to progress.

As part of her workup, laboratory tests revealed that the patient had growth hormone deficiency (GHD). Her growth hormone (GH) level was measured at 0.343 ng/mL, significantly below the normal reference range for an 18-year-old female. This confirmed the diagnosis of GHD, which could explain her short stature (140 cm at 18 years of age).

On admission, the patient's laboratory investigations revealed hemoglobin 9.3 g/dL (anemia), serum creatinine 5.7 mg/dL (renal impairment), serum calcium 6.8 mg/dL (hypocalcaemia), intact parathyroid hormone (iPTH) 1126 pg/mL (severely increased), vitamin D (25-OH-D) level 6.2 ng/mL (severely deficient), phosphate: 2.56 mg/dL (normal range). The patient was also subjected to radiological imaging to assess her thyroid and parathyroid glands, as well as the bones. A neck ultrasound was performed, revealing bilateral parathyroid adenomas. Additionally, there were small, non-specific lymph nodes in the upper and mid jugular regions, which were not indicative of malignancy. These findings confirmed the diagnosis of SHPT, as parathyroid hyperplasia is commonly associated with CKD.

An MRI of the neck revealed solid lesions on both the right and left posterior thyroid lobes. The right lesion measured approximately 1.0 x 0.8 cm, and the left lesion measured 0.4 x 0.6 cm. The MRI did not suggest malignancy, and these findings were consistent with bilateral parathyroid adenomas. A pelvic X-ray showed osteopenia and bone sclerosis at the sacrum, suggestive of renal osteodystrophy. A follow-up bone survey revealed multiple lytic lesions in the calvaria, iliac bones, pubic bones, and both femoral trochanters, consistent with brown tumors—a hallmark of renal osteodystrophy associated with SHPT.

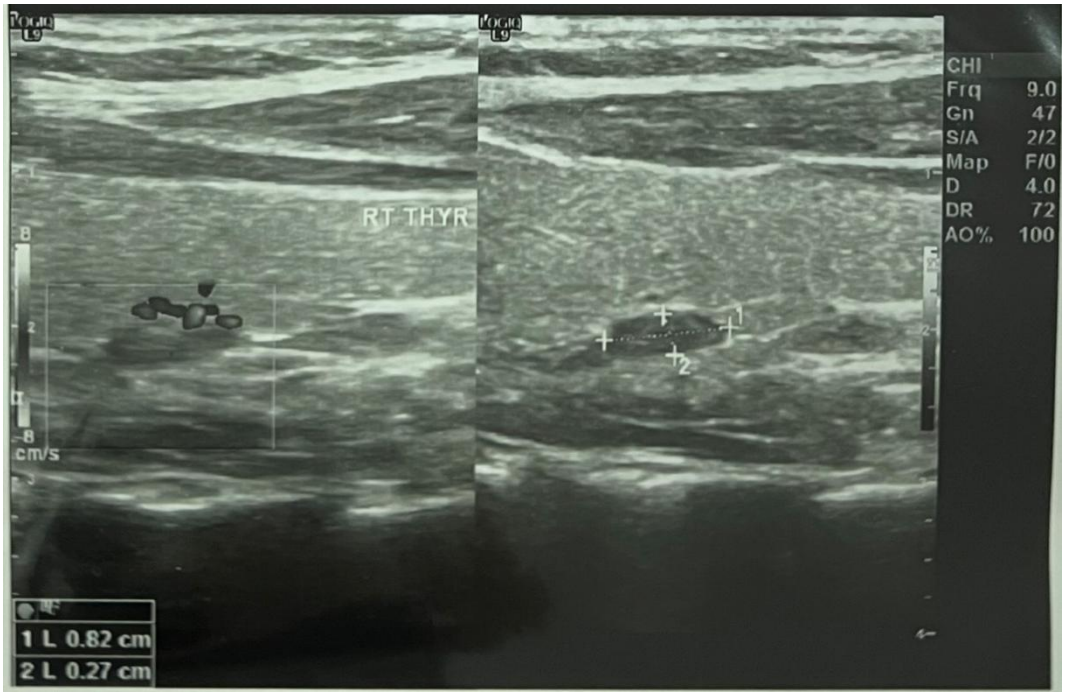

Figure 1. Neck (thyroid) ultrasound, right lobe

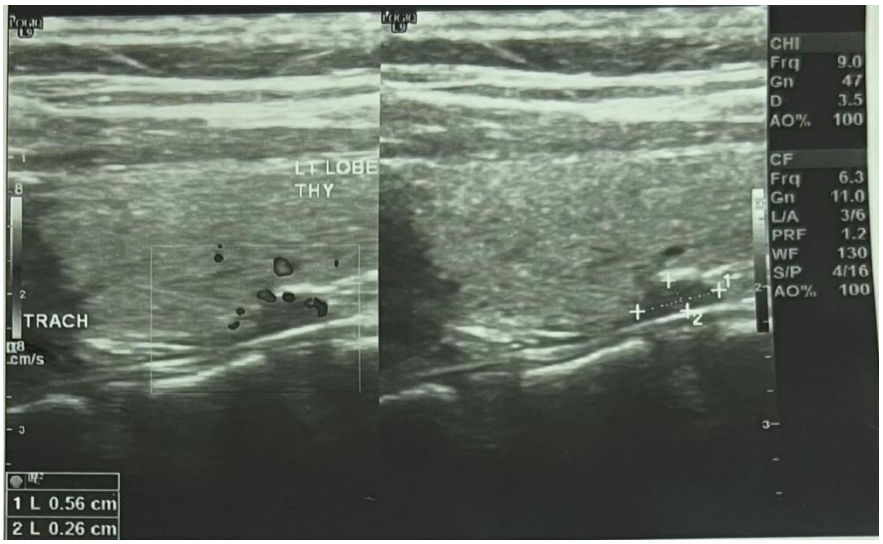

Figure 2. Neck (thyroid) ultrasound, left lobe

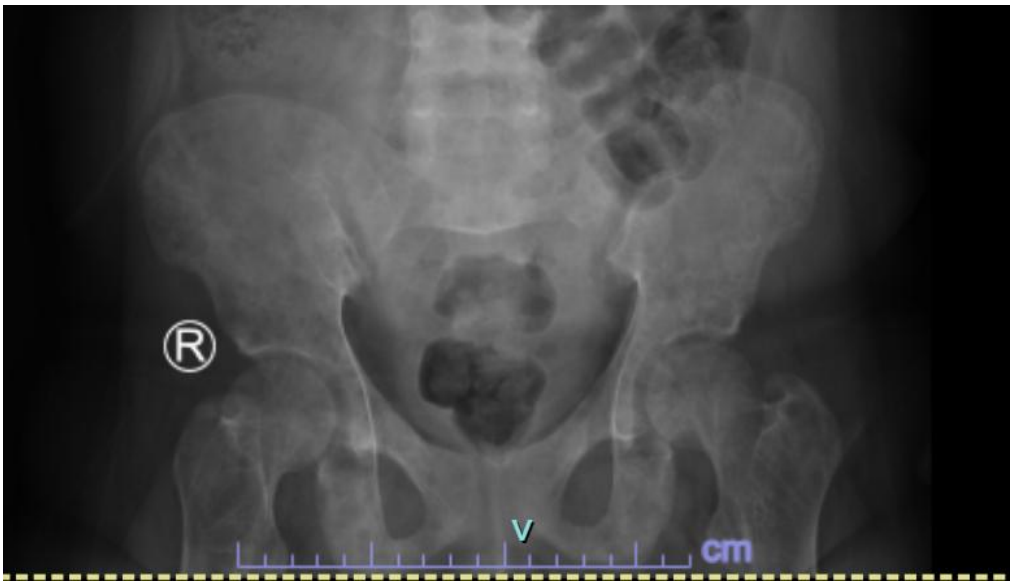

Figure 3. Pelvic x-ray showing multiple lytic lesions in iliac and pubic bones, also both of femoral trochanters

Upon diagnosis, the patient was started on a comprehensive treatment regimen that included calcium supplementation (500 mg calcium carbonate every 8 hours), which helped correct hypocalcaemia and decreased the stimulus for excessive PTH secretion, and vitamin D supplementation with calcitriol (0.5 mcg every 24 hours), which helped to increase calcium absorption from the intestines and suppress parathyroid hormone release. After two weeks, follow-up laboratory tests showed a significant reduction in iPTH levels to 107.6 pg/mL, indicating a positive response to the treatment. The patient's symptoms improved, and the need for surgical intervention was postponed.

2. DISCUSSION

This case highlights the complex interplay between secondary hyperparathyroidism (SHPT) and chronic kidney disease (CKD), with additional complicating factors such as growth hormone deficiency (GHD). The patient's clinical course exemplifies the metabolic disturbances seen in advanced CKD and the associated mineral bone disorder (CKD-MBD), particularly the role of vitamin D deficiency and hypocalcemia in the development of SHPT.

Secondary hyperparathyroidism (SHPT) is a common complication in patients with CKD, particularly in advanced stages. It

arises due to disturbances in the balance of calcium, phosphate, and vitamin D, all of which are regulated by the kidneys [1]. In patients with CKD, the kidneys lose their ability to activate vitamin D to its active form, calcitriol, which is necessary for the proper absorption of calcium from the gut and the regulation of calcium levels in the blood [5]. The resulting deficiency of calcitriol leads to hypocalcemia, a key stimulus for the overproduction of parathyroid hormone (PTH), as the parathyroid glands attempt to compensate for the low calcium levels by increasing PTH secretion. The chronically elevated PTH levels lead to parathyroid hyperplasia and the typical skeletal abnormalities seen in renal osteodystrophy, a hallmark of CKD-MBD. [1]. In this patient, laboratory findings of elevated iPTH, combined with low calcium and vitamin D levels, confirmed the diagnosis of secondary hyperparathyroidism (SHPT).

In this case, the patient's severe vitamin D deficiency (measured at 6.2 ng/mL) contributed to the hypocalcemia observed (serum calcium level of 6.8 mg/dL) and played a major role in the overproduction of PTH. Moreover, radiological findings of osteopenia and bone sclerosis in the sacrum, as well as lytic lesions in the skull and pelvis, were consistent with renal osteodystrophy, further supporting the diagnosis of SHPT and its complications. The treatment of vitamin D deficiency with calcitriol (active vitamin D) was essential to correct this imbalance and manage the patient's SHPT. The correction of hypocalcemia through calcium carbonate supplementation helped normalize calcium levels and alleviate some of the triggers for excessive PTH secretion [1]. This underscores the importance of monitoring vitamin D levels in CKD patients and ensuring appropriate supplementation to mitigate the effects of SHPT and prevent bone and mineral complications.

A key component of the patient's workup included evaluating growth hormone (GH) levels. Laboratory investigations revealed a GH level of 0.343 ng/mL, which is below the reference range for an 18-year-old female. This value indicated the presence of growth hormone deficiency (GHD), contributing to her short stature (140 cm) and possibly influencing the progression of her kidney disease.

The dilemma that arises in this case is whether CKD-induced growth hormone deficiency is the primary cause, or whether growth hormone deficiency itself contributed to the onset and progression of CKD. Given that growth hormone (GH) is involved in kidney function and mineral metabolism, it is critical to explore whether GHD exacerbated the metabolic disturbances in CKD, including calcium and phosphate imbalances, or whether CKD itself contributed to impaired GH secretion through disrupted hypothalamic-pituitary feedback mechanisms [6].

This duality is seen in many patients with CKD, where GH deficiency may both be a result of the chronic disease (due to altered pituitary function) and a contributing factor to the progression of renal dysfunction. The relationship between GHD and CKD is complex and not fully understood, but studies suggest that long-term kidney dysfunction can lead to hormonal dysregulation, including GHD. On the other hand, evidence also indicates that untreated GHD may impair kidney structure and function, potentially accelerating CKD progression [4]. Thus, it is challenging to pinpoint whether the GH deficiency occurred due to the CKD or whether the GHD has accelerated the patient's kidney disease.

In this patient, the diagnosis of GHD was confirmed through laboratory testing, which revealed a growth hormone level of 0.343 ng/mL, well below the normal range. GHD likely contributed to the patient's short stature (140 cm at 18 years of age) and may have also played a role in the worsening of her CKD and SHPT. Growth hormone influences renal function and bone mineral density through its impact on the IGF-1 axis and calcium-phosphate homeostasis [4].

Radiological examination for localizing parathyroid adenomas is standard in the evaluation of hyperparathyroidism. If a mass is detected in the parathyroid glands, it is essential to determine whether the mass is benign or malignant. Parathyroid carcinoma is most commonly diagnosed after parathyroidectomy, making it a critical step in tracking malignancy. Initial radiological evaluations for hyperparathyroidism include neck ultrasound, followed by four-dimensional computed tomography (CT) if the lesion cannot be localized by ultrasound. This imaging modality is useful for determining whether the tumor is benign or malignant; however, it lacks sufficient sensitivity and specificity to assess malignancy potential [7].

The management of SHPT in CKD patients involves addressing the underlying calcium and phosphate imbalances, as well as vitamin D deficiency and other hormonal deficiencies like GHD [1]. The patient's treatment regimen consist of calcium supplementation (500 mg calcium carbonate every 8 hours), which helped correct hypocalcaemia and decreased the stimulus for excessive PTH secretion, and vitamin D supplementation with calcitriol (0.5 mcg every 24 hours), which helped to increase calcium absorption from the intestines and suppress parathyroid hormone release. Phosphate binders such as aluminum hydroxide, sevelamer hydrochloride, sevelamer carbonate, and lanthanum carbonate are often used to control phosphate levels when hyperphosphatemia is present to prevent further complications related to mineral metabolism [1]. Calcimimetics are agents that enhance the sensitivity of the calcium-sensing receptor (CaSR) in the parathyroid glands, leading to a reduction in parathyroid hormone (PTH) production. Cinacalcet is a commercially available calcimimetic widely used in dialysis patients. This agent helps regulate calcium metabolism and is particularly beneficial in controlling PTH levels in patients with secondary hyperparathyroidism (SHPT), especially those undergoing hemodialysis [1].

Parathyroidectomy is a surgical option for patients with secondary hyperparathyroidism (SHPT) when medical therapy fails or when it is difficult to achieve adequate control of the disease [1]. Additional indications for surgery include calciphylaxis,

refractory pruritus, severe hypercalcemia (serum calcium levels above 10.2 mg/dL), or hyperphosphatemia (serum phosphate levels greater than 5.5 mg/dL), erythropoietin-resistant anemia, parathyroid hormone (PTH) levels exceeding 800 pg/mL for more than six months despite medical treatment, and the presence of extra-skeletal calcifications [1]. Parathyroid glands in SHPT are characterized by asymmetric enlargement and nodular hyperplasia. Assessment of parathyroid mass is crucial in predicting the response to medical management. Glands larger than 1 cm or those measuring more than 500 mm^3 on ultrasound typically suggest glandular hyperplasia and are often resistant to medical treatment. It is estimated that surgery may be necessary in approximately 15% of patients within 10 years and 38% within 20 years after the initiation of dialysis. With the introduction of calcimimetic agents, there appears to be a reduction in the need for parathyroidectomy [1].

Given the patient's GHD, consideration of growth hormone therapy could be beneficial to further improve bone health and possibly slow the progression of kidney dysfunction, although more evidence is needed to support its routine use in CKD patients. In future cases, patients with CKD and SHPT should be closely monitored for growth hormone deficiency, as it can complicate the management of bone and mineral disorders. A multi-disciplinary approach, including endocrinologists, nephrologists, and dietitians, is crucial in managing such complex cases to prevent the need for surgical interventions and improve patient outcomes.

3. CONCLUSION

This case report illustrates the complexities of managing secondary hyperparathyroidism (SHPT) in a young patient with stage V chronic kidney disease (CKD), complicated by growth hormone deficiency (GHD). The patient's clinical presentation, including hypocalcaemia, vitamin D deficiency, and elevated PTH levels, was effectively managed with calcium supplementation and vitamin D therapy. Her positive response to treatment suggests that early diagnosis and multi-faceted management of SHPT can prevent the need for surgical intervention in many cases. The role of growth hormone deficiency in CKD requires further investigation to determine its impact on renal function and mineral metabolism. This case underscores the importance of addressing mineral imbalances, hormonal deficiencies, and growth disorders in the management of CKD to improve patient outcomes

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