



# Prolactinoma management in chronic kidney disease: Diagnostic and therapeutic challenges - A case report

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## Abstract

Hyperprolactinemia, a common endocrine disorder, often occurs in patients with Chronic Kidney Disease (CKD). This case report discusses the management of prolactinoma in a CKD patient at Dr. Soetomo Hospital. A 41-year-old man presented with nausea, vomiting, weakness, and sore throat. He had a long-standing history of dizziness, hearing loss, difficulty walking, and decreased appetite. Radiological imaging suggested a pituitary adenoma consistent with prolactinoma. Treatment with bromocriptine 2.5 mg resulted in effective suppression of prolactin levels. This case highlights the importance of early recognition and appropriate endocrine management in CKD patients.

**Keywords:** Bromocriptine, Chronic kidney disease, Prolactin, Prolactinoma, Radiological imaging

## Introduction

Chronic Kidney Disease (CKD) represents a global health challenge characterized by nonspecific early-stage symptoms, limited patient awareness, and accelerated progression in advanced stages. A 2017 Global Burden of Disease study revealed significant increases in CKD incidence (31.6%), prevalence (27.0%), and mortality (34.0%) compared to the previous decade [1].

Hyperprolactinemia frequently accompanies kidney failure as a common endocrine complication [2,11]. Research demonstrates that uremia alters the metabolic milieu and reduces glomerular filtration rates. These pathophysiological changes disrupt extrarenal metabolic processes and impair feedback mechanisms governing peptide hormone production and release. Prolactin (PRL), traditionally recognized for its role in lactation and gonadal luteinizing hormone receptor regulation, serves multiple physiological functions including osmoregulation, immune modulation, and angiogenesis [3]. In CKD, elevated prolactin levels result from both diminished renal clearance and enhanced hormone secretion [4].

## Case report

A 41-year-old male was referred to the emergency department of Dr. Soetomo Hospital from the

Neurosurgery Clinic with a diagnosis of prolactinoma and Acute Kidney Injury (AKI), suspected to be Acute On Chronic Kidney Disease (ACKD). The patient presented with nausea and vomiting occurring 2-4 times daily for the past week, accompanied by generalized weakness and sore throat. His initial referral from Lumajang Hospital was prompted by a 10-year history of vertigo that had significantly worsened over the previous two months, along with accompanying hearing loss and gait disturbance. Diagnostic imaging in June 2023 revealed a 1.1 x 1.4 cm pituitary adenoma consistent with prolactinoma. The patient also reported significant weight loss of approximately 15 kg over the past year, decreased appetite, reduced bowel movements, and low urine output of about 600 mL/day.

His medical history was notable for previous alcohol consumption and high-fat dietary habits during his residence in Makassar, though he denied any history of chronic diseases such as diabetes, hypertension, or kidney disorders. Physical examination revealed an overweight patient (BMI 27.3 kg/m<sup>2</sup>) with pale conjunctivae, normal cardiopulmonary findings, abdominal striae, and mild pedal edema. Laboratory results demonstrated severe microcytic hypochromic anemia (Hb 5.7 g/dL), azotemia (creatinine 21.7 mg/dL), hyperkalemia (7.0 mmol/L), hyponatremia (121 mmol/L), metabolic acidosis (pH 7.28), and hypoalbuminemia (2.79 g/dL), along with significant proteinuria and hematuria. Endocrine evaluation

confirmed hyperprolactinemia (169.98 ng/mL) and secondary hypothyroidism (FT4 0.47 ng/dL). Imaging studies confirmed the pituitary adenoma and revealed right paracardial pulmonary infiltrates suggestive of pneumonia.

The patient was ultimately diagnosed with AKI (possibly ACKD), high-risk community-acquired pneumonia, prolactinoma-related hypothyroidism, multiple electrolyte imbalances, and severe anemia. He required emergency hemodialysis upon admission on August 1, 2023, for management of his uremic state.

On day 4 (4/8), the patient reported persistent weakness, right ear tinnitus, and generalized pruritus. Vital signs were stable (BP 117/97 mmHg, SpO<sub>2</sub> 99%), with pale conjunctivae and bilateral leg edema. Urine output remained low (<500 mL/24h), prompting hemodialysis. Diagnoses included AKI (suspected ACKD), severe CAP, metabolic acidosis, hyperkalemia, hyponatremia, microcytic anemia, and prolactinoma-induced hypothyroidism. Management included IV fluids, antibiotics (moxifloxacin), electrolyte correction, and hormone replacement (levothyroxine).

By day 7 (7/8), symptoms improved: tinnitus and pruritus decreased, edema subsided, and urine output rose to ~700 mL/24h. Lab results showed resolving hyperkalemia (K<sup>+</sup> 5.6 mmol/L) and azotemia post-hemodialysis (2 sessions). Treatment continued with adjusted electrolyte supplements and ongoing antibiotics.

On day 11 (11/8), weakness further improved, edema resolved, and labs indicated recovery (Hb 11.5 g/dL, K<sup>+</sup> 3.5 mmol/L, creatinine 11.4 mg/dL). Prolactin levels normalized (5.7 ng/mL), confirming treatment efficacy. The diagnosis shifted to CKD requiring regular hemodialysis (2x/week), with outpatient nephrology follow-up scheduled.

During follow-up (15/8–18/8), the patient was cleared for hemodialysis in Lumajang. A non-contrast head CT was planned, and an ophthalmologic exam revealed no visual field deficits, confirming intact pituitary adenoma-related optic pathways.

Final Status: Stable on maintenance hemodialysis and hormonal therapy, with resolved acute complications

and scheduled monitoring.

## Discussion

The kidneys serve as crucial endocrine organs, not only producing hormones like erythropoietin and renin but also participating in the metabolism of various hormones including prolactin, insulin, and cortisol. Hyperprolactinemia represents one of the most frequent endocrine disturbances in patients with chronic kidney disease (CKD), with reported prevalence as high as 56% in this population [5]. This condition arises through multiple mechanisms, including decreased renal clearance of prolactin due to reduced glomerular filtration rate (GFR), alterations in dopaminergic tone, and potential pituitary adenoma formation [6]. The case of this 41-year-old male illustrates the complex interaction between renal failure and endocrine dysfunction, presenting with both severe AKI/CKD (creatinine 21.7 mg/dL) and marked hyperprolactinemia (169.98 ng/mL).

The pathophysiology of hyperprolactinemia in renal failure involves both renal and extrarenal mechanisms. The kidney normally clears about 33% of circulating prolactin, and this clearance is markedly impaired in renal failure. Additionally, uremia appears to disrupt hypothalamic dopaminergic tone, the primary inhibitory control of prolactin secretion. This is evidenced by blunted prolactin responses to thyrotropin-releasing hormone stimulation and resistance to single-dose dopamine or bromocriptine challenges, though prolonged bromocriptine administration (6 weeks) can achieve suppression [6]. The patient's MRI findings of a 1.1 × 1.4 cm pituitary adenoma further complicated this picture, as prolactinomas represent the most common pituitary adenoma subtype, accounting for 57% of cases [7].

Clinical manifestations in this patient reflected both his renal failure and endocrine dysfunction. The anemia (Hb 5.7 g/dL) resulted from combined CKD-related erythropoietin deficiency and hyperprolactinemia-induced hypogonadism, which impairs erythropoiesis [7]. His metabolic acidosis (pH 7.28, HCO<sub>3</sub> 14.1 mmol/L) and hyperkalemia (7.0 mmol/L) represented typical complications of advanced renal failure, while his hypoalbuminemia (2.79 g/dL) and peripheral edema reflected the

nephrotic-range proteinuria (4+) and salt retention characteristic of CKD. Notably, the physical findings of striae and gynecomastia suggested additional cortisol and sex hormone disturbances frequently seen in CKD [4, 8].

Management of such cases requires addressing both the renal and endocrine aspects. While hemodialysis effectively managed the azotemia, it had minimal impact on prolactin levels, as prolactin's molecular size (23 kDa) limits its dialyzability [9]. The patient responded well to bromocriptine (2.5 mg), a dopamine agonist that directly suppresses prolactin secretion. This treatment choice aligns with current evidence showing dopamine agonists as safe and effective for hyperprolactinemia in CKD patients when clinically indicated [9, 10]. However, the persistence of his pituitary adenoma may eventually require neurosurgical intervention if medical management proves insufficient.

This case underscores the importance of comprehensive endocrine evaluation in CKD patients, particularly when symptoms exceed typical uremic manifestations. The bidirectional relationship between renal function and prolactin metabolism creates a vicious cycle where CKD exacerbates hyperprolactinemia, which in turn worsens complications like anemia and bone disease. Clinicians must maintain high suspicion for prolactin disorders in CKD patients presenting with hypogonadism, unexplained anemia, or pituitary mass effects, as timely diagnosis and treatment can significantly impact quality of life and long-term outcomes.

## Conclusion

This case illustrates the bidirectional relationship between chronic kidney disease (CKD) and hyperprolactinemia, where renal dysfunction reduces prolactin clearance while elevated prolactin exacerbates CKD complications. A 41-year-old male patient was referred to the emergency department of Dr. Soetomo Hospital with complaints of nausea, vomiting, and weakness, accompanied by a diagnosis of severe AKI/CKD and a 1.4 cm macroprolactinoma. Key findings include: (1) hemodialysis is ineffective in lowering prolactin levels due to its molecular size; (2) dopamine agonists (bromocriptine 2.5 mg) successfully suppress prolactin even in CKD; and (3)

pituitary imaging (non-contrast MRI, for renal safety) is essential when prolactin levels exceed the expected range for kidney failure. This case underscores the importance of endocrine evaluation in CKD patients with disproportionate symptoms, such as galactorrhea or visual disturbances suggestive of prolactinoma, as well as the need for a multidisciplinary approach (nephrology and endocrinology) for optimal management.

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