EJPMR, 2026, 13(1), 426-428 **Case Study** **Impact Factor 7.065**

EUROPEAN JOURNAL OF PHARMACEUTICAL AND MEDICAL RESEARCH

www.ejpmr.com

ISSN (O): 2394-3211
ISSN (P): 3051-2573
Coden USA: EJPMAG

REFRACTORY HYPOCALCEMIA AND HYPOMAGNESEMIA: A CASE OF CARBOPLATIN-INDUCED RENAL TUBULOPATHY IN METASTATIC BREAST CANCER

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DOI: https://doi.org/10.5281/zenodo.18151569

How to cite this Article: Rathnakumar G.[2], Sivasubramanian B.[1], Vinayaka B.[3]* (2026). Refractory Hypocalcemia And Hypomagnesemia: A Case Of Carboplatin-Induced Renal Tubulopathy In Metastatic Breast Cancer. European Journal of Pharmaceutical and Medical Research, 13(1), 426–428.

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Article Received on 05/12/2025 **Article Revised on 25/12/2025** **Article Published on 05/01/2026**

ABSTRACT

Platinum-based chemotherapies, notably cisplatin, are well-documented causes of renal magnesium wasting. Carboplatin, while considered less nephrotoxic, can cause similar electrolyte disturbances, though this is less frequently reported. We present a case of severe, symptomatic hypocalcemia secondary to carboplatin-induced hypomagnesemia in a patient with metastatic breast cancer. A 48-year-old woman with metastatic breast cancer, status post-modified radical mastectomy and palliative carboplatin chemotherapy completed 2nd cycle 2 months back, and stopped due to development of recurrent hypocalcemia, presented with a 2-day history of painful involuntary muscle twitching in her hands and feet. Physical examination showed positive Trousseau's and Chvostek's signs. Laboratory investigations revealed severe hypocalcemia (serum calcium 4.5 mg/dL), hypomagnesemia (0.8 mg/dL), hypokalemia (2.4 mEq/L), and Metabolic alkalosis. Parathyroid hormone (PTH) level was inappropriately normal (31 pg/mL) despite hypocalcemia. Renal function indicated acute kidney injury (AKI). Urinary fractional excretion of magnesium was elevated at 24%, confirming renal magnesium wasting. Other causes of hypocalcemia, such as hypoparathyroidism and sepsis and tumor lysis syndrome were excluded. The patient was diagnosed with carboplatin-induced renal tubulopathy. She was managed with intravenous and oral replacements of magnesium, calcium, and potassium, alongside cholecalciferol, leading to clinical improvement. This case highlights that carboplatin, though safer than cisplatin, can cause significant distal tubular damage, leading to profound magnesium wasting.

KEYWORDS: Carboplatin • Hypomagnesemia • Hypocalcemia • Renal Tubulopathy • Chemotoxicity • Breast Cancer.

BACKGROUND

Electrolyte disturbances are a common complication of chemotherapy. Cisplatin is notoriously associated with nephrotoxicity, particularly renal magnesium wasting, which occurs in 50 -90% of patients.[1] Carboplatin was developed to have a more favorable toxicity profile, with reduced nephrotoxicity. However, cases of significant electrolyte imbalance secondary to carboplatin, though less frequent, are reported and can be diagnostically challenging. The presentation can mimic other disorders like hypoparathyroidism, Vitamin D deficiency Pseudohypoparathyroidism, Mineral bone disease in Chronic renal failure patients necessitating a high index of suspicion in oncology patients.

CASE PRESENTATION

A 48-year-old woman with a history of metastatic left breast carcinoma, treated with modified radical mastectomy and palliative carboplatin chemotherapy completed 2nd cycle 2 months back and stopped due to development of hypocalcemia, presented to the hospital with a 2-day history of intermittent, painful involuntary twitching of both hands and feet. She also reported weight loss and anorexia. She had no history of diarrhea, vomiting, polyuria, or seizure.

Clinical Findings: On examination, she was conscious and oriented. Vital signs were stable. Pallor was present. Notably, Trousseau's(Fig 1) and Chvostek's signs were positive, indicating neuromuscular irritability. Systemic examinations of the cardiovascular, respiratory, and central nervous systems were unremarkable.

Fig 2: Eliciting Trousseau's sign.

Investigations: Laboratory results were critical for diagnosis.

Test	Result	Interpretation
S.Calcium	4.5mg/dl	Severe hypocalcemia
S.Pottasium	2.4mEq/L	Hypokalemia
S.Magnesium	0.8mg/L	Severe Hypomagnesemia
PH	7.52	Metabolic alkalosis
HCO3 / pCO2	35 / 45	
S.Creatine Urea	1.89 35	Acute Kidney injury
	Nill	—-
S. PTH	31pg/ml	Inappropriately normal
Vit D3	<11mg/ml	Very low
Hematology	Pancytopenia	Chemotherapy induced
24hr Urine K+	17 mEq/L	No urinary electrolyte loss
24 hr Urine Na	74 mEq/L	No urinary electrolyte loss
Fractional excretion of Mg in urine	24%	Mg wasting is present in urine

ECG revealed prolonged QT interval (Fig 2)

There was no glycosuria or significant aminoaciduria, ruling out full Fanconi syndrome. There is no electrolyte loss in urine and hence salt wasting syndromes similar to Gitelman can be excluded.

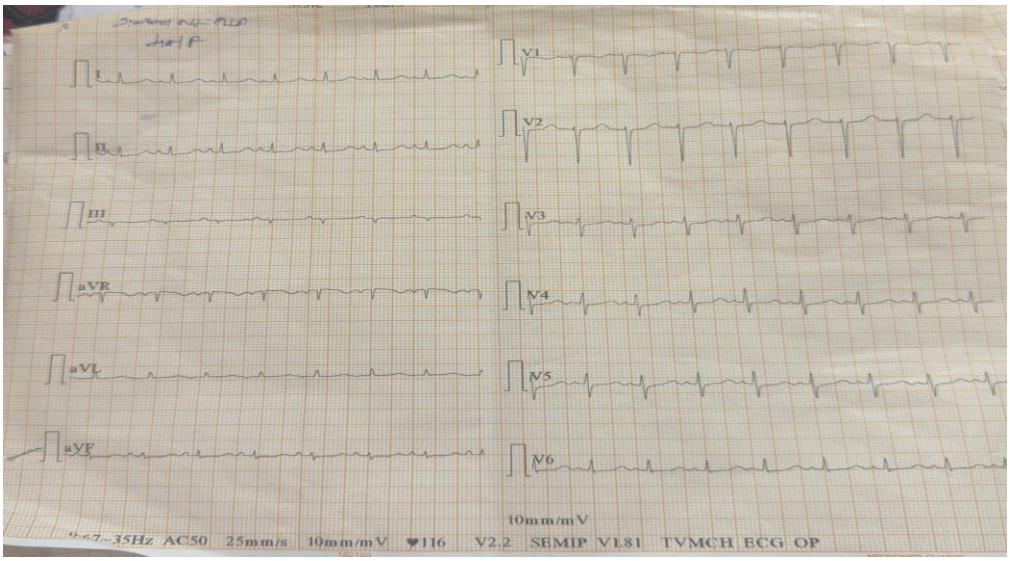

Fig 2: showing prolonged QT interval.

The patient was treated with intravenous magnesium sulfate, potassium chloride, and calcium gluconate, leading to the resolution of her neuromuscular symptoms. Oral calcium, magnesium, and cholecalciferol supplements were initiated. Despite clinical improvement, serum magnesium levels remained persistently low at discharge, consistent with the refractory nature of chemotherapy-induced tubulopathy.

DISCUSSION

This case illustrates a classic but often overlooked complication of platinum-based chemotherapy: renal magnesium wasting. While carboplatin is less tubulotoxic than cisplatin, it shares the potential to cause damage to the distal convoluted tubule, where magnesium reabsorption is primarily regulated.

The pathophysiology involves direct tubular toxicity, leading to an inability to conserve magnesium. The ensuing hypomagnesemia has two critical effects on calcium homeostasis.

- Impaired PTH Secretion: Magnesium is a cofactor for PTH release; severe deficiency leads to suppressed PTH secretion.
- PTH Resistance: Magnesium is required for the peripheral action of PTH on bone and kidney.

This explains the biochemical paradox seen in our patient: severe hypocalcemia with an inappropriately normal PTH level. The hypokalemia and metabolic alkalosis further support a distal tubular defect. The differential diagnosis included Gitelman syndrome-like presentation. No history of polyuria or hypovolumic state on examination and urinary electrolyte loss is absent by investigation too, hence unlikely.

Cisplatin induced nephropathy has got various presentation such as.[2]

a. AKI (20-30%)
b. Hypomagnesemia(50 -90%)
c. Salt wasting (Gitelman like)
d. Urine Concentration defect
e. Fanconi syndrome
f. Distal RTA
g. CKD

Hypomagnesemia caused by Magnesium wasting via renal tubule is as common as 50 to 90% of patients receiving Cisplatin.[3] Though Carboplatin is less tubulotoxic, it still can cause as in literature.[4]

A key learning point is the refractory nature of this condition. Magnesium wasting can persist for years after discontinuing platinum therapy, requiring long-term, often high-dose, oral supplementation. Vigilant monitoring of magnesium and calcium levels before, during, and after platinum-based chemotherapy is essential for prevention and early intervention. The resultant hypomagnesemia impairs PTH secretion and action, leading to refractory hypocalcemia. The triad of hypomagnesemia, hypokalemia, and metabolic alkalosis points to a Renal tubulopathy. This case underscores the need for vigilant monitoring of electrolytes in patients receiving platinum-based agents, even after chemotherapy cessation, as tubulopathy can be persistent and refractory. Administration of isotonic saline supplemented with potassium chloride and magnesium sulfate before and after cisplatin administration can decrease tubulotoxicity.[5]

CONCLUSION

This case highlights that carboplatin, despite its reputation for reduced nephrotoxicity, can cause severe and persistent renal magnesium wasting, leading to symptomatic hypocalcemia. Clinicians must maintain a high index of suspicion for this entity in patients who are receiving chemotherapy presenting with tetany or electrolyte imbalances. The cornerstone of management is aggressive repletion of magnesium, which is necessary to correct the associated hypocalcemia.

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