Case Report

Gallbladder carcinoma with hypoparathyroidism-induced hypocalcemia: A rare presentation

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ABSTRACT

Gallbladder carcinoma (Ca GB) is an aggressive malignancy usually diagnosed at advanced stages, with poor prognosis. Hypocalcemia is a known complication in oncology, most often secondary to tumor lysis syndrome, bisphosphonate therapy, or Vitamin D deficiency. Hypoparathyroidism-induced hypocalcemia, however, is exceedingly rare and has not been documented in gallbladder cancer. We report a 37-year-old male who presented with abdominal pain, jaundice, anorexia, and weight loss. Imaging revealed irregular gallbladder wall thickening extending into the extrahepatic bile duct with partial encasement of the right hepatic artery, consistent with stage T4 Ca GB. He underwent percutaneous transhepatic biliary drainage and received gemcitabine-based chemotherapy. Following eight cycles, he presented with acute bilateral lower limb weakness. Laboratory evaluation demonstrated severe hypocalcemia (serum calcium 4.3 mg/dL), low parathyroid hormone (13 pg/mL), and low Vitamin D (14 ng/mL), consistent with hypoparathyroidism-induced hypocalcemia. He was treated with calcitriol and calcium supplementation, with marked symptomatic improvement. This case represents a rare association of Ca GB with hypoparathyroidism-induced hypocalcemia. Early recognition and management are essential to prevent morbidity. Reporting such unique presentations expands understanding of paraneoplastic manifestations in gastrointestinal malignancies.

Key words: Gallbladder carcinoma, Hypocalcemia, Hypoparathyroidism, Paraneoplastic syndrome

Gallbladder carcinoma (Ca GB) is the most common biliary tract malignancy, accounting for nearly 80–95% of biliary cancers worldwide. Its incidence is highest in parts of India, Chile, and Eastern Europe, with a particularly heavy burden in North and Northeast India [1]. The disease is highly aggressive, and most patients present with advanced-stage disease due to non-specific early symptoms. Prognosis is poor, with median survival rarely exceeding 1 year in advanced stages [2]. Hypocalcemia is a well-recognized complication in oncology, typically caused by tumor lysis syndrome, sepsis, bisphosphonate therapy, or Vitamin D deficiency [3]. In contrast, hypocalcemia due to hypoparathyroidism is rare in malignancy, usually observed after thyroid or parathyroid surgery, autoimmune syndromes, or, very rarely, as a paraneoplastic manifestation [4].

To the best of our knowledge, no prior case report has documented hypoparathyroidism-induced hypocalcemia in Ca GB. We present a rare case of advanced Ca GB complicated by persistent hypocalcemia due to hypoparathyroidism, with a focus on its clinical relevance, diagnostic challenges, and therapeutic implications.

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CASE PRESENTATION

A 37-year-old male, previously healthy, presented in July 2024 with abdominal pain, jaundice, anorexia, and weight loss for 2 months. There was no significant past medical or surgical history, family history of cancer, or history of tobacco or alcohol consumption.

Magnetic resonance cholangiopancreatography (July 24, 2024) revealed a contracted gallbladder with calculi, irregular wall thickening involving the fundus, neck, and cystic duct, extending into the extrahepatic bile duct. Soft-tissue thickening caused stricture of the common hepatic duct and proximal common bile duct. The right hepatic artery was partially encased, with significant abdominal lymphadenopathy and no ascites. Computed tomography abdomen confirmed the findings and incidentally revealed a horseshoe kidney. Laboratory evaluation showed total bilirubin of 13 mg/dL. Histopathological examination confirmed it as well-differentiated adenocarcinoma. The patient underwent percutaneous transhepatic biliary

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drainage for decompression. Based on imaging, he was staged as T4, node-positive Ca GB, deemed inoperable, and referred for systemic therapy.

He was initiated on palliative systemic therapy; cycle 1 included gemcitabine monotherapy due to persistent hyperbilirubinemia. From cycle 2 onward, he received gemcitabine–cisplatin (Gem Cis) as bilirubin had come to normal limits by then. A total of eight cycles were administered, with the last cycle on June 27, 2025.

On July 7, 2025, the patient presented with acute bilateral lower limb weakness and paresthesias. Examination revealed muscle weakness (Grade 4/5), flaccid type in bilateral lower limbs, with diminished tone and diminished deep tendon jerks in the ankle and knee, facial twitching, and a positive Trousseau's sign. No bladder and bowel involvement was noted. His vitals were stable, and the patient was conscious, oriented to time, place, and person. Laboratory workup showed serum calcium 4.3 mg/dL, albumin 2.8 g/dL, corrected calcium 4.7 mg/dL, and serum magnesium 2.1 mg/dL with normal renal function tests. Ionized calcium was not estimated. He was started on intravenous calcium supplementation with routine monitoring as an inpatient. However, his hypocalcaemia persisted with periodic worsening of lower limb weakness. On further evaluation, serum parathyroid hormone (PTH) was found to be 13 pg/mL (which is below the normal lower limit), serum phosphate levels 6 mg/dL (above the normal upper limit), and Vitamin D 14 ng/mL (low). Findings were suggestive of hypoparathyroidism-induced hypocalcemia. A repeat PTH level was not available; therefore, the biochemical findings were considered suggestive of hypoparathyroidism rather than fulfilling strict criteria for chronic hypoparathyroidism. Magnetic resonance imaging of the brain and spine was not done at that time because the neurological symptoms improved following correction of severe hypocalcemia.

He was treated with calcitriol, Vitamin D3, and oral calcium supplementation. The calcitriol dose was gradually titrated up, and the patient showed significant clinical improvement and restoration of ambulation. His serum calcium values also gradually stabilized over a period of 2–3 weeks, and calcitriol supplementation with oral calcium was continued as a maintenance dose.

DISCUSSION

Ca GB is often diagnosed in advanced stages due to non-specific symptoms such as abdominal pain and jaundice. Our patient presented with typical features but subsequently developed an unusual complication, persistent hypocalcemia, which was later found to be due to hypoparathyroidism.

Hypocalcemia in oncology patients is a common problem, often encountered as an oncological emergency. Causes include tumor lysis syndrome, bisphosphonate therapy, sepsis, magnesium deficiency, and Vitamin D deficiency [3]. In contrast, hypoparathyroidism as the cause of hypocalcemia in cancer patients is rare [4].

Although the patient had concomitant Vitamin D deficiency, with a 25-hydroxyvitamin D concentration of 14 ng/mL, Vitamin D deficiency alone was unlikely to account for the entire biochemical profile. In isolated Vitamin D deficiency, reduced calcium absorption generally stimulates a compensatory increase in PTH secretion, resulting in secondary hyperparathyroidism. PTH also promotes renal phosphate excretion, and serum phosphate is therefore generally normal or reduced rather than elevated.

In the present patient, however, profound hypocalcemia was accompanied by an inappropriately low PTH level of 13 pg/mL and hyperphosphatemia of 6 mg/dL. This pattern is more compatible with impaired PTH secretion than with isolated Vitamin D deficiency. Vitamin D deficiency was, therefore, considered a potentially aggravating factor rather than the sole explanation for the severe hypocalcemia.

Hypoparathyroidism is characterized biochemically by hypocalcemia associated with an undetectable, low, or inappropriately normal PTH level. Hyperphosphatemia further supports impaired PTH activity because loss of the phosphaturic action of PTH results in increased renal phosphate retention.

Hypoparathyroidism is characterized by inadequate PTH secretion, leading to hypocalcemia and hyperphosphatemia [4]. It is most often seen post-thyroidectomy, autoimmune conditions, or as part of congenital syndromes. Reports of hypoparathyroidism in solid tumors are exceedingly rare. A few cases have been reported in lung cancer and thyroid carcinoma, where autoimmune mechanisms or paraneoplastic cytokines were implicated [5].

To the best of our knowledge, no published literature has linked Ca GB with hypoparathyroidism-induced hypocalcemia. Our case therefore represents a unique probable paraneoplastic association.

Proposed mechanisms include: Paraneoplastic autoimmunity – tumor antigens may trigger immune-mediated parathyroid destruction. Cytokine dysregulation – advanced tumors secrete cytokines that may interfere with the calcium-PTH axis. Nutritional factors – coexistent Vitamin D deficiency, common in Indian patients, may worsen the biochemical profile [6].

Clinical implications include: Symptom recognition: Neuromuscular weakness in advanced cancer patients should prompt calcium evaluation. Therapeutic awareness: Severe hypocalcemia may affect chemotherapy tolerance, cardiac function, and quality of life. The clinical implications of this case include the need for prompt recognition of neuromuscular symptoms in patients with advanced malignancy, which should trigger evaluation for electrolyte abnormalities, particularly hypocalcemia. Severe hypocalcemia may adversely

affect chemotherapy tolerance, cardiac function, and quality of life. This case also highlights the importance of considering uncommon endocrine abnormalities in patients with malignancy.

Our case expands the spectrum of Ca GB complications by adding a rare paraneoplastic endocrine presentation.

CONCLUSION

This case illustrates a rare presentation of Ca GB with hypoparathyroidism-induced hypocalcemia. In oncology practice, persistent hypocalcemia should not always be attributed to Vitamin D deficiency or tumor lysis syndrome; hypoparathyroidism should also be considered. Early diagnosis and management with calcitriol and calcium can significantly improve patient outcomes. Reporting such rare associations is important to enhance clinical awareness and expand understanding of paraneoplastic syndromes in gastrointestinal cancers.

AUTHORS' CONTRIBUTIONS

KM, RB: Conceptualization, case management, data collection, and manuscript writing. PSR: Clinical supervision, manuscript editing, and final approval. All authors read and approved the final manuscript.

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