Case Report

Delayed Hypocalcemia Post-Parathyroidectomy in a Dog with Parathyroid Carcinoma

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Abstract

Hypocalcemia is considered a common electrolyte abnormality in dogs after parathyroidectomy and usually occurs within the first 2–4 days postoperatively. The authors describe a case involving an 11-year-old spayed female Chow Chow dog that presented with decreased appetite and ongoing hypercalcaemia, with an ionized calcium (iCa) level of 2.08 mmol/L. Computed tomography (CT) revealed enlargement of the left external cranial pole and right internal caudal pole of the parathyroid glands. Abdominal ultrasound identified multiple small hyperechoic mineralizations in both renal diverticula. These findings were consistent with primary hyperparathyroidism. Left cranial and right caudal parathyroidectomies were performed. Histopathological examination revealed proliferation of neoplastic neuroendocrine cells, highly suggestive of parathyroid carcinoma. ICa levels were monitored daily for 5 days, and no hypocalcemia was detected. However, on the 12th postoperative day, the dog developed signs of hypocalcemia, including muscle twitching and tetany, and the iCa concentration dropped from 2.08 mmol/L to 0.6 mmol/L. The patient was treated with 10% calcium gluconate intravenously (1 ml/kg) for 3 consecutive days, followed by oral calcitriol (0.01 µg/kg twice daily) for 7 days. Clinical signs resolved, and ionized calcium levels increased to the normal range (1.45 mmol/L) within 4 days after initial oral calcitriol treatment. Subsequently, the calcitriol dose was tapered to once daily for 7 days, then every other day for the next 14 days, and ultimately discontinued after a total treatment duration of 4 weeks. Two weeks after discontinuation of calcitriol, the ionized calcium level remained within normal limits (1.26 mmol/L), and no signs of hypocalcemia were observed.

Keywords: Hypercalcemia, Hypocalcemia, Parathyroidectomy, Parathyroid carcinoma

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ภาวะแคลเซียมต่ำที่เกิดขึ้นล่าช้าหลังการผ่าตัดต่อมพาราไทรอยด์ในสุนัขที่เป็นมะเร็งต่อมพาราไทรอยด์

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บทคัดย่อ

ภาวะแคลเซียมในเลือดต่ำ (hypocalcemia) เป็นความผิดปกติของอิเล็กโทรไลต์ที่พบได้บ่อยในสุนัขภายหลังจากการผ่าตัดนำต่อมพาราไทรอยด์ออก (parathyroidectomy) ซึ่งมักพบภายในระยะเวลา 2 ถึง 4 วันภายหลังการผ่าตัด รายงานสัตว์ป่วยฉบับนี้นำเสนอสุนัขพันธุ์เชาเชา เพศเมีย ทำหมันแล้ว อายุ 11 ปี ที่เข้ารับการวินิจฉัยหาสาเหตุของภาวะเบื่ออาหารและตรวจพบภาวะแคลเซียมในเลือดสูงแบบต่อเนื่อง โดยมีระดับแคลเซียมอิสระในเลือด (Ionized calcium) เท่ากับ 2.08 มิลลิโมลต่อลิตร การตรวจเอกซเรย์คอมพิวเตอร์ (CT) พบการขยายขนาดของต่อมพาราไทรอยด์บริเวณขั้วบนภายนอกฝั่งซ้ายและขั้วล่างภายในฝั่งขวา ผลอัลตราซาวนด์ช่องท้องพบแร่ธาตุสะสมขนาดเล็กหลายตำแหน่งในไตทั้งสองข้าง โดยความผิดปกติเหล่านี้สอดคล้องกับภาวะต่อมพาราไทรอยด์ทำงานเกินแบบปฐมภูมิ (primary hyperparathyroidism) จึงได้ทำการผ่าตัดนำต่อมพาราไทรอยด์บริเวณขั้วบนภายนอกฝั่งซ้ายและขั้วล่างภายในฝั่งขวาออก ผลการตรวจชิ้นเนื้อทางพยาธิวิทยาพบการเพิ่มจำนวนของเซลล์ประสาทต่อมไร้ท่อที่มีลักษณะนิวเคลียสไม่สม่ำเสมอ ซึ่งเป็นลักษณะของมะเร็งของต่อมพาราไทรอยด์ (parathyroid carcinoma) การติดตามระดับแคลเซียมอิสระในเลือดในซีรั่มต่อเนื่องเป็นเวลา 5 วันหลังผ่าตัด ไม่พบภาวะแคลเซียมต่ำ แต่ในวันที่ 12 หลังผ่าตัด ระดับแคลเซียมอิสระในเลือดลดลงจาก 2.08 มิลลิโมลต่อลิตร เหลือ 0.6 มิลลิโมลต่อลิตร โดยสุนัขแสดงอาการทางคลินิกของภาวะแคลเซียมต่ำ ได้แก่ กล้ามเนื้อกระตุกและชักเกร็ง จึงทำการรักษาด้วยแคลเซียมกลูโคเนตความเข้มข้นร้อยละ 10 ทางหลอดเลือดดำ ขนาดยา 1 มิลลิลิตรต่อกิโลกรัม ต่อเนื่อง 3 วัน จากนั้นให้แคลซิไตรออลทางการกิน ขนาดยา 0.01 ไมโครกรัมต่อกิโลกรัม วันละ 2 ครั้ง เป็นระยะเวลา 7 วัน ภายใน 4 วันหลังการรักษาด้วยแคลซิไตรออล ไม่พบอาการผิดปกติทางคลินิก และระดับแคลเซียมอิสระในเลือดเพิ่มขึ้นสู่ระดับปกติ (1.45 มิลลิโมลต่อลิตร) จึงทำการปรับลดขนาดยาแคลซิไตรออล ลงเป็นวันละครั้ง 7 วัน และ วันเว้นวันอีก 14 วัน และหยุดยารวมระยะเวลาในการรักษาด้วยยาแคลซิไตรออลทั้งหมดเป็นระยะเวลา 4 สัปดาห์ หลังหยุดการรักษา 2 สัปดาห์ ระดับแคลเซียมอิสระในเลือดยังคงอยู่ในช่วงปกติ (1.26 มิลลิโมลต่อลิตร) และไม่พบอาการของภาวะแคลเซียมต่ำ

คำสำคัญ: ภาวะแคลเซียมในเลือดต่ำ ภาวะแคลเซียมในเลือดสูง การตัดต่อมพาราไทรอยด์ มะเร็งของต่อมพาราไทรอยด์

Introduction

Hypercalcemia is a clinically important metabolic abnormality in dogs, most commonly associated with neoplasia, hypoadrenocorticism, chronic kidney disease, or primary hyperparathyroidism (PHPT) (Coady et al., 2019). Among these, PHPT is considered an uncommon cause of persistent hypercalcemia, but it should not be overlooked. PHPT results from excessive secretion of parathyroid hormone (PTH) by functional neoplastic or hyperplastic parathyroid tissue, leading to sustained elevations in ionized calcium (Berger and Feldman 1987).

Persistent hypercalcemia contributes to multisystemic complications, including polyuria and polydipsia, neuromuscular weakness, and gastrointestinal upset (Strumpf et al., 2023). Severe hypercalcemia can lead to cardiac arrhythmias, including conduction disturbances such as third-degree heart block, and may contribute to sudden death in rare cases (Wilson et al., 1974; Dear et al., 2017). Hypercalcemia in dogs can cause widespread soft tissue mineralization (Hilbe et al., 2000). These changes can be confirmed by gross and histopathological examination, both of which significantly worsen prognosis and highlight the importance of early detection and management. The outcome of dogs with hypercalcemia is largely dependent on the underlying cause. Dogs with neoplasia-associated hypercalcemia, particularly lymphoma, apocrine gland anal sac adenocarcinoma (AGASACA) (Repasy et al., 2022), and mammary gland tumors (Russo et al., 2021), generally carry a poor prognosis due to the aggressive biological behavior of these tumors and the limited long-term treatment success (Zandvliet 2016; Kohart et al., 2017). Conversely, dogs with PHPT generally have a favorable prognosis after surgical treatment with long-term normocalcemia achieved in most cases (Jores and Kessler 2011; Young and Degner 2023). Human population data indicate that PHPT patients with a maximum serum calcium concentration ≥ 10.8 mg/dL have increased mortality and that parathyroidectomy significantly improves survival in this high-calcium subgroup (Wermers et al., 2021); however, comparable data defining calcium thresholds or demonstrating a survival benefit of parathyroidectomy at specific serum calcium concentrations are lacking in dogs. Nevertheless, untreated or poorly managed hypercalcemia in PHPT can still result in irreversible renal damage, reducing overall survival (Cortadellas et al., 2010; Assadipour et al., 2019).

Although rare, PHPT is a potentially curable disease, and dogs that undergo successful surgical removal of the affected parathyroid gland (s) generally have an excellent prognosis (Young and Degner 2023). Nonetheless, severe postoperative hypocalcemia is a recognized and common complication (Erickson et al., 2021; Travail et al., 2025).

Hypocalcemia is a clinically significant electrolyte disturbance in dogs and can arise from a variety of underlying conditions. It is most commonly associated with critical illness, acute kidney injury, eclampsia, certain toxicities, protein-losing enteropathy, and hypoparathyroidism (either primary or secondary to parathyroidectomy) (Holowaychuk et al., 2012; Coady et al., 2019; Gagliardo et al., 2020). Importantly, hypocalcemia can also occur following parathyroidectomy for primary hyperparathyroidism. In such cases, chronic suppression of normal parathyroid tissue due to persistently elevated circulating PTH concentrations may impair the ability of the remaining glands to restore normocalcemia postoperatively (Mu et al., 2023; Xu et al., 2024).

This report describes the clinical presentation, diagnostic workup, surgical management, and postoperative complications, especially hypocalcemia, of a dog with persistent hypercalcemia caused by parathyroid carcinoma. This case highlights the importance of accurate diagnosis, appropriate postoperative management, and the recognition of delayed hypocalcemia following parathyroidectomy in achieving a favorable outcome in a dog with parathyroid carcinoma.

Case description

An 11-year-old spayed female Chow Chow was presented to a private veterinary clinic with a history of decreased appetite and mild depression. A complete blood count (CBC) and serum biochemistry revealed mild azotemia with a creatinine concentration of 1.6 mg/dL (reference interval [RI]: 0.3–1.4 mg/dL) and markedly elevated total serum calcium at 16 mg/dL (RI: 8.6–11.8 mg/dL), a low serum phosphorous concentration of 2.3 mg/dL (RI: 2.9–6.66 mg/dL), and a PTH level of 2.4 pg/mL (RI: 10–65 pg/mL), measured by a human laboratory assay that is not validated for animals. Thoracic radiography demonstrated a normal cardiac silhouette and unremarkable pulmonary parenchyma. Abdominal ultrasonography revealed multiple small hyperechoic mineralizations within the renal diverticula without evidence of renal pelvis dilation. Bilateral adrenal gland enlargement was also identified. The left adrenal gland measured 2.34 cm at the cranial pole and 1.74 cm at the caudal pole, with three hyperechoic nodules in the cranial pole parenchyma. The right adrenal gland measured 0.7 cm at the cranial pole and 1.6 cm at the caudal pole, with a hyperechoic nodular lesion (0.4 × 0.5 cm) at the caudal pole. Fluid therapy was initiated to correct dehydration, and reassessment was performed one week later. Azotemia had improved; however, hypercalcemia persisted (16 mg/dL). Ionized calcium (iCa) was also elevated at 2.08 mmol/L (RI: 1.1–1.4 mmol/L).

The dog was referred to Prasu Arthon Animal Hospital (Faculty of Veterinary Science, Mahidol University, Thailand) for further investigation of persistent hypercalcemia. On examination, the dog was alert, responsive, 5% dehydrated, and exhibited mild hindlimb lameness. Total calcium remained elevated at 18 mg/dL (Biosystem BA400), and iCa was 2.0 mmol/L (Siemens, RAPIDLab®348EX). Based on the differential diagnoses of primary hyperparathyroidism versus paraneoplastic hypercalcemia, a computed tomography (CT) scan (helical contrast-enhanced CT scan, 1.25 mm and 0.625 mm slice thickness) was performed. CT findings included enlargement of the left external cranial parathyroid gland (0.6 × 0.5 × 0.5 cm) and the right internal caudal parathyroid gland (0.5 × 0.3 × 0.3 cm) (Figure 1). These findings supported differential diagnoses of functional parathyroid adenoma, adenocarcinoma, or hyperplasia. Bilateral adrenomegaly with a left adrenal mass (2.7 × 1.7 cm) was also observed.

Surgical intervention was undertaken via a ventral cervical approach. A left cranial parathyroidectomy and partial right thyroidectomy with caudal parathyroidectomy were performed, and tissue samples were collected for histopathological evaluation (Figure 2). Histopathology revealed a proliferation of neoplastic neuroendocrine cells arranged in short columnar patterns, with moderate amounts of eosinophilic cytoplasm. Nuclei were round, finely stippled to vesiculated, and contained 1–2 nucleoli, with one mitotic figure observed, finding highly suggestive of parathyroid carcinoma (Figure 3).

The dog was hospitalized for postoperative monitoring. During the immediate postoperative period (postoperatively days 1–5), iCa concentrations normalized (RI: 1.12–1.40 mmol/L), vital parameters remained stable, and no surgical complications were observed. The patient was discharged in good condition.

On postoperative day 9, iCa had decreased to 0.8 mmol/L, but the dog remained asymptomatic. The dog was closely monitored, with iCa measurements performed every other day, and the owner was instructed to observe for signs of hypocalcemia. By day 12, iCa had dropped to 0.6 mmol/L, accompanied by clinical signs of hypocalcemia, including muscle twitching and tetany. Emergency management consisted of intravenous administration of 10% calcium gluconate at 100 mg/kg administered over 30 minutes, followed by a continuous rate infusion at 100 mg/kg/h over 6 hours once daily for 3 consecutive days. Thereafter, oral calcitriol was administered at 0.01 µg/kg twice daily for 7 days. Serum iCa normalized to 1.45

mmol/L within 4 days of initiation of oral calcitriol treatment. The calcitriol dosage was gradually tapered to 0.01 µg/kg once daily for 7 days, then every other day for 14 days, and ultimately discontinued after a total treatment duration of 4 weeks. Two weeks after cessation of calcitriol, iCa remained stable at 1.26 mmol/L (Table 1), and the dog remained clinically normal.

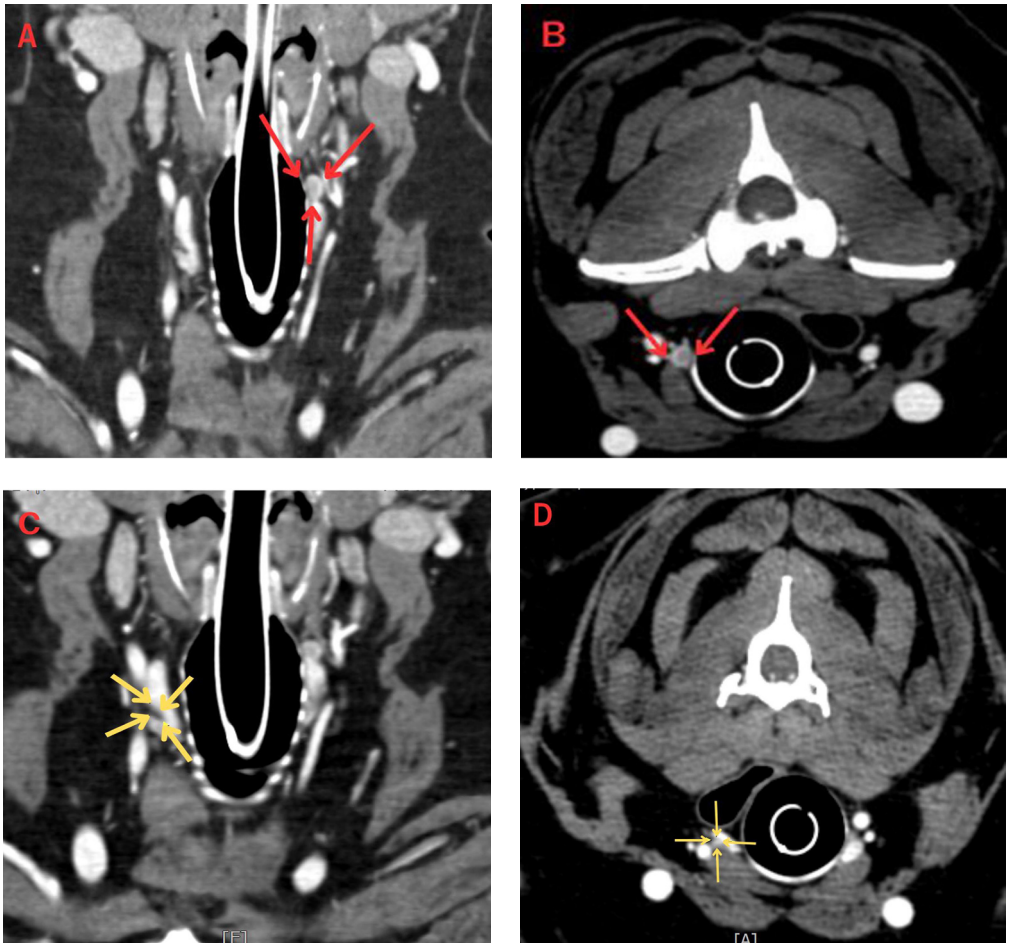

Figure 1. Computed tomography (CT) images of the parathyroid glands. (A) Dorsal and (B) transverse planes of the left external cranial pole parathyroid gland (red arrows). (C) Dorsal and (D) transverse planes of the right internal caudal pole parathyroid gland (yellow arrows).

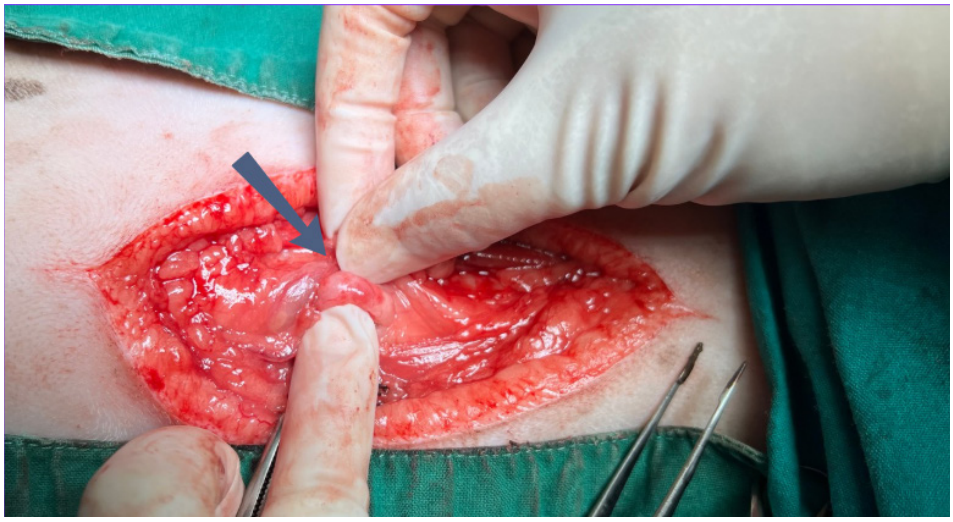

Figure 2. Parathyroidectomy, the grey arrow indicates the left external cranial parathyroid gland to be excised.

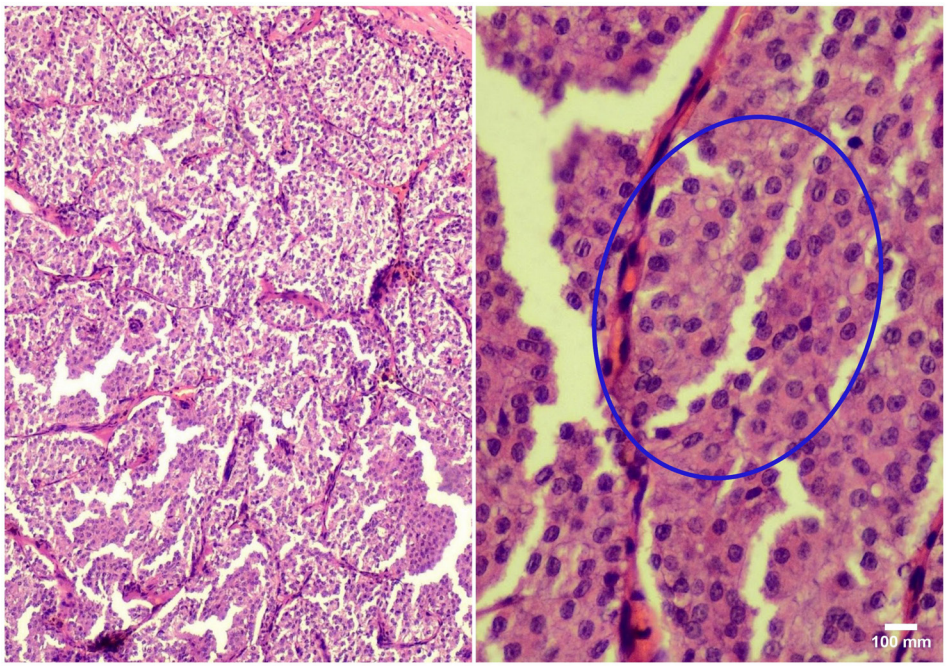

Figure 3. Histopathologic section of an ovoid parathyroid mass with soft to firm consistency, stained with hematoxylin and eosin (H&E). The blue circle indicates infiltrative proliferation of neoplastic neuroendocrine cells. The neoplastic cells are predominantly arranged in dense sheets separated by delicate collagenous stroma. The cells are short columnar with moderate eosinophilic cytoplasm. The nuclei are round with mild anisokaryosis.

Table 1. Summary of ionized and total calcium concentrations in the pre- and postoperative periods.

Day	Ionized calcium (1.12-1.4 mmol/L)	Total calcium (8-11.8 mg/dL)
Initial diagnosis	2.08	16
Parathyroidectomy	-	-
Postoperative day 1 (12 hours)	1.4	13.7
Postoperative day 1 (24 hours)	1.3	-
Postoperative day 2	1.1	-
Postoperative day 3	1.23	9.3
Postoperative day 4	1.4	-
Postoperative day 5	1.1	8.9
Postoperative day 9	0.8	-
Postoperative day 12	0.6	-
3 days after initial 10% calcium gluconate 2 ml/kg	0.8	7.6
4 days after initial oral calcitriol 0.01 μg/kg (BID)	1.45	-
7 days after oral calcitriol 0.01 μg/kg (BID)	1.4	-
7 days after oral calcitriol 0.01 μg/kg (SID)	1.5	11.6
14 days after oral calcitriol 0.01 μg/kg (QOD)	1.3	-
14 days after cessation of oral calcitriol	1.26	-

Discussion

Primary hyperparathyroidism (PHPT) is an uncommon endocrine disorder in dogs, most often caused by a functional parathyroid adenoma, and less frequently by hyperplasia or adenocarcinoma (Jores and Kessler 2011; Cordella et al., 2022). The concurrent adrenal masses observed in this dog, in the absence of clinical signs of hyperadrenocorticism, were considered likely incidental findings and most consistent with non-functional adrenal enlargement. Similar cases of adrenal gland enlargement without clinical manifestations have been reported in dogs (Baum et al., 2016). In this case, PTH measurement was performed at a private veterinary clinic; however, the results could not be interpreted because the assay used was designed for humans and is not validated for canine samples. Nevertheless, the combination of persistent hypercalcemia and CT evidence of bilateral parathyroid enlargement strongly supported a tentative diagnosis of primary hyperparathyroidism and informed surgical planning.

The microscopic description of this dog revealed an infiltrative proliferation of neoplastic neuroendocrine cells arranged in dense sheets, nests, and acini/tubules, characterized by short columnar cells with moderate eosinophilic cytoplasm. The nuclei were round with mild anisokaryosis. These features are consistent with the general appearance of neuroendocrine carcinoma, including that of the parathyroid gland (Kuwata et al., 2010; Champion et al., 2025), reflecting its neuroendocrine origin and infiltrative behavior, with a pathophysiology linked to PTH overproduction leading to bone resorption and hypercalcemia.

Parathyroidectomy remains the treatment of choice for parathyroid carcinoma (PTC) in dogs and is generally associated with favorable long-term outcomes. A large multicenter retrospective study of 100 dogs with PTC demonstrated that normocalcemia was restored in 89 of 96 cases within 7 days of surgery, and survival rates were also encouraging, with 1-, 2-, and 3-year survival rates of 84%, 65%, and 51%, respectively, and a median survival time of approximately 2 years (Erickson et al., 2021). In the present case, the dog remained alive and clinically stable for 16 months (1.4 years) following parathyroidectomy. Intensive monitoring of overall health, ionized calcium concentration, and adrenal mass size was performed monthly for the first 3 months postoperatively, during which all parameters remained within normal limits. Once stability was confirmed, the monitoring interval was extended to every 3 months. Throughout follow-up, ionized calcium levels remained within the reference range, the adrenal mass did not increase in size, and no clinical abnormalities were noted.

Although parathyroid carcinoma in dogs has the potential for local invasion, as well as regional nodal or distant metastasis, such findings have not been consistently documented (Sawyer et al., 2012). Recurrence of functional parathyroid carcinoma has been reported more than 6 months after surgical excision in some dogs (Capen 2002), highlighting the importance of long-term monitoring. In the current case, no evidence of recurrence or metastatic disease was detected during the 16-month follow-up period.

These findings highlight that PTC is a curable disease with a good prognosis when accurately diagnosed and appropriately managed. Despite these positive outcomes, postoperative hypocalcemia is one of the most common complications after parathyroidectomy. Approximately 36% of dogs undergoing surgery for PTC or PHPT experience hypocalcemia within the first 2–4 days postoperatively (Dear et al., 2017; Burkhardt et al., 2021; Travail et al., 2025). However, the prediction of postoperative hypocalcemia in dogs with primary hyperparathyroidism that underwent parathyroidectomy is difficult and depends on multiple factors. Dogs with higher preoperative calcium concentrations are more likely to develop hypocalcemia postoperatively, possibly due to negative feedback from high circulating levels of PTH, which suppresses function in the remaining parathyroid glands (Mu et al., 2023; Xu et al., 2024).

Importantly, the onset of hypocalcemia can be delayed, with reports documenting such cases. Twelve dogs developed hypocalcemia within a median of 34 hours after surgery, and some dogs developed clinical signs of hypocalcemia as late as 7 days after surgery (Arbaugh et al., 2012; Milovancev and Schmiedt 2013; Erickson et al., 2021).

In the present case, the dog remained normocalcemic for the first 5 postoperative days but developed severe hypocalcemia on days 9 and 12, with iCa levels dropping to 0.8 and 0.6 mmol/L, respectively. Clinical signs of hypocalcemia were evident, including muscle twitching and tetany. This delayed presentation emphasizes the importance of vigilant postoperative monitoring extending beyond the immediate recovery period. Intravenous calcium gluconate (at dosages of 0.5–1.5 mL/kg) remains the first-line therapy for acute symptomatic hypocalcemia, while calcitriol therapy (at a dose of 0.02–0.03 μg/kg for 3–4 days for induction, then 0.005–0.015 μg/kg for maintenance) is titrated to the desired serum calcium concentration. Intravenous calcium gluconate provided rapid correction of extracellular hypocalcemia, whereas calcitriol supported recovery by enhancing intestinal calcium absorption and maintaining serum calcium as parenteral supplementation was tapered (Groman 2012). In this case, this combination addressed both the acute calcium deficit and facilitated restoration of calcium homeostasis during postoperative recovery.

Importantly, dogs that underwent surgery before the development of severe renal disease had significantly better

outcomes. One retrospective study of 29 dogs with PHPT found that those without renal failure at the time of parathyroidectomy had better long-term survival times, ranging from 6 months to over 3.5 years after surgical treatment, with most patients maintaining normocalcemia following resolution of postoperative complications, while dogs with pre-existing or developing renal failure had poorer outcomes (Gear et al., 2005). These findings, combined with the present case, demonstrate the value of early, accurate diagnosis, timely surgery, and aggressive management of hypocalcemia following parathyroidectomy, not only resolving life-threatening hypercalcemia but also providing an excellent long-term prognosis.

In primary hyperparathyroidism, chronically elevated PTH concentrations result in increased bone resorption and high bone turnover. Following parathyroidectomy, circulating PTH concentrations decrease abruptly, and the previously high-turnover bone shifts from net mineral release to rapid mineral uptake. This process leads to a decline in blood ionized calcium (iCa) concentrations over hours to days and is commonly referred to as the "hungry bone" phenomenon (Graham et al., 2012; Travail et al., 2025).

In dogs, hypocalcemia after parathyroidectomy typically develops between 12 hours and 7 days postoperatively, most commonly between postoperative days 2 and 6 (Burkhardt et al., 2021; Travail et al., 2025). This delayed onset supports a bone-mediated mechanism rather than an immediate perioperative loss of calcium (Graham et al., 2012). Older dogs and those with prolonged or severe hypercalcemia are likely to have more extensive skeletal remodeling and, consequently, may experience more pronounced postoperative calcium uptake and hypocalcemia (Milovancev and Schmiedt 2013).

Although direct canine comparative data are limited, human and veterinary evidence suggest that parathyroid carcinoma is more genetically aggressive and functionally autonomous than adenoma (Li et al., 2024). This likely causes more profound chronic suppression and atrophy of the remaining parathyroid tissue, helping explain prolonged postoperative hypoparathyroidism and unusually severe hypocalcemia in affected dogs (Erickson et al., 2021).

Pre-existing nephrocalcinosis in this dog may substantially impair PTH-mediated tubular calcium reabsorption and calcitriol synthesis, limiting both renal conservation and intestinal uptake of calcium. This provides a plausible mechanistic link to unusually severe, prolonged postoperative hypocalcemia after parathyroid surgery.

In dogs with naturally occurring hypercortisolism with bilateral adrenomegaly, total and ionized calcium concentrations are typically within the reference range, although increased phosphate concentrations, increased urinary calcium excretion, and secondary elevations in parathyroid hormone have been reported (Mooney et al., 2020; Corsini et al., 2021). Overt hypocalcemia is uncommon in this setting. Therefore, while adrenal enlargement may indirectly influence calcium balance and endocrine regulation, it is unlikely to have been a primary contributor to hypocalcemia in this case.

A limitation of this case was the absence of a species-validated PTH assay. Only a human-based laboratory test was available, which is not validated for canine use and therefore uninterpretable. Consequently, the diagnosis could not be fully supported by PTH measurement. In this case, parathyroid carcinoma was diagnosed; however, the histologic evidence supporting malignancy was limited to mild anisokaryosis, and immunohistochemical confirmation would have strengthened the diagnosis. Unfortunately, immunohistochemistry could not be performed because of the owner's financial constraints.

In conclusion, this case highlights several important points for the clinical diagnosis of persistent hypercalcemia with parathyroid enlargement and management of canine PTC. First, although rare, PTC should be considered in dogs with persistent hypercalcemia, and diagnosis requires integration of iCa measurement, PTH assays, and advanced imaging. Second, parathyroidectomy is the gold standard treatment, offering

a high likelihood of cure and favorable survival outcomes. Finally, clinicians should extend postoperative monitoring to detect delayed hypocalcemia, the most common postoperative complication, which may persist for at least several days, as rare cases may present later and should be prepared to manage it with calcium gluconate and vitamin D supplementation. With accurate diagnosis, appropriate surgical intervention, and diligent postoperative care, parathyroid carcinoma in dogs is a rare but highly treatable disease with a good long-term prognosis.

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References

Arbaugh M, Smeak D, Monnet E. Evaluation of preoperative serum concentrations of ionized calcium and parathyroid hormone as predictors of hypocalcemia following parathyroidectomy in dogs with primary hyperparathyroidism: 17 cases (2001–2009). J Am Vet Med Assoc. 2012;241(2):233-6.

Assadipour Y, Zhou H, Kuo E, Haigh P, Adams A, Yeh M. End-organ effects of primary hyperparathyroidism: A population-based study. Surgery. 2019;165:99-105.

Baum J, Boston S, Case J. Prevalence of adrenal gland masses as incidental findings during abdominal computed tomography in dogs: 270 cases (2013–2014). J Am Vet Med Assoc. 2016;249(10):1165-9.

Berger B, Feldman EC. Primary hyperparathyroidism in dogs: 21 cases (1976–1986). J Am Vet Med Assoc. 1987;191(3):350-6.

Burkhardt S, Sumner J, Mann S. Ambidirectional cohort study on the agreement of ultrasonography and surgery in the identification of parathyroid pathology, and predictors of postoperative hypocalcemia in 47 dogs undergoing parathyroidectomy due to primary hyperparathyroidism. Vet Surg. 2021;50:1379-88.

Capen C. Tumors and nonneoplastic cysts of the parathyroid gland. In: Meuten DJ, editor. Tumors in domestic animals. 4th ed. Ames, IA: Iowa State Press; 2002. p. 665-9.

Champion C, Miller A, Parry S, Demeter E. Immunohistochemical and histomorphologic characterization of canine neoplasms of the disseminated neuroendocrine system. Vet Pathol. 2025;62:490-501.

Coady M, Fletcher D, Goggs R. Severity of ionized hypercalcemia and hypocalcemia is associated with etiology in dogs and cats. Front Vet Sci. 2019;6:276.

Cordella A, Bertaccini J, Rondena M, Zoia A, Bertolini G. Multidetector-row CT findings in dogs with different primary parathyroid gland diseases. Vet Sci. 2022;9(6):273.

Corsini A, Dondi F, Serio D, Zamagni S, Golinelli S, Fernandez M, et al. Calcium and phosphate homeostasis in dogs with newly diagnosed naturally occurring hypercortisolism. J Vet Intern Med. 2021;35:1265-73.

Cortadellas O, Del Palacio M, Talavera J, Bayón A. Calcium and phosphorus homeostasis in dogs with spontaneous chronic kidney disease at different stages of severity. J Vet Intern Med. 2010;24(1):73-9.

Dear J, Kass P, Della Maggiore A, Feldman E. Association of hypercalcemia before treatment with hypocalcemia after treatment in dogs with primary hyperparathyroidism. J Vet Intern Med. 2017;31:349-54.

Erickson AK, Regier PJ, Watt MM, Ham KM, Marvel SJ, Wallace ML, et al. Incidence, survival time, and surgical treatment of parathyroid carcinomas in dogs: 100 cases (2010–2019). J Am Vet Med Assoc. 2021;259(11):1309-17.

Gagliardo T, Ruggeri R, Di Paola A, Baroni M, Cherubini G, Gallucci A, et al. Clinical features of muscle cramp in 14 dogs. J Vet Intern Med. 2020;35(2):372-7.

Gear R, Neiger R, Skelly BJ, Herrtage M. Primary hyperparathyroidism in 29 dogs: diagnosis, treatment, outcome and associated renal failure. J Small Anim Pract. 2005;46(1):10-6.

Graham K, Wilkinson M, Ja C, Dhand N, Churcher R. Intraoperative parathyroid hormone concentration to confirm removal of hypersecretory parathyroid tissue and time to postoperative normocalcaemia in nine dogs with primary hyperparathyroidism. Aust Vet J. 2012;90(6):203-9.

Groman RP. Acute management of calcium disorders. Top Companion Anim Med. 2012;27(4):167-71.

Hilbe M, Sydler T, Fischer L, Naegeli H. Metastatic calcification in a dog attributable to ingestion of a tacalcitol ointment. Vet Pathol. 2000;37:490-2.

Holowaychuk M, Birkenheuer A, Li J, Marr H, Boll A, Nordone S. Hypocalcemia and hypovitaminosis D in dogs with induced endotoxemia. J Vet Intern Med. 2012;26(2):244-51.

Jores K, Kessler M. Primary hyperparathyroidism in the dog: diagnosis, therapy and postoperative management in 19 dogs. Tierarztl Prax K. 2011;39(6):389-96.

Kohart NA, Elshafae SM, Breitbach JT, Rosol TJ. Animal models of cancer-associated hypercalcemia. Vet Sci. 2017;4(2):21.

Kuwata K, Shibutani M, Kemmochi Y, Taniai E, Morita R, Ogawa BI, et al. A neuroendocrine carcinoma of undetermined origin in a dog. J Toxicol Pathol. 2010;23:151-5.

Li Y, Simonds WF, Chen H. A Comparative Genomic Analysis of Parathyroid Adenomas and Carcinomas Harboring Heterozygous Germline CDC73 Mutations. J Clin Endocrinol Metab. 2025;110(2):429-40.

Milovancev M, Schmiedt CW. Preoperative factors associated with postoperative hypocalcemia in dogs with primary hyperparathyroidism that underwent parathyroidectomy: 62 cases (2004–2009). J Am Vet Med Assoc. 2013;242(4):507-15.

Mooney CT, Shiel RE, Sekiya M, Dunning M, Gunn E. A Preliminary Study of the Effect of Hyperadrenocorticism on Calcium and Phosphate Concentrations, Parathyroid Hormone and Markers of Bone Turnover in Dogs. Front Vet Sci. 2020;7:311.

Mu Y, Zhao Y, Zhao J, Zhao Q, Zhang Y, Li Y, et al. Factors influencing serum calcium levels and the incidence of hypocalcemia after parathyroidectomy in primary hyperparathyroidism patients. Front Endocrinol (Lausanne). 2023;14:1276992.

Repasy A, Selmic L, Kisseberth W. Canine apocrine gland anal sac adenocarcinoma: A review. Top Companion Anim Med. 2022;47:100682.

Russo C, Simonelli S, Luz M, Moreno I, Carrera A, Bracarense A. Hypercalcemia in bitches with malignant mammary neoplasms. Pubvet. 2021;15(8):1-8.

Sawyer E, Northrup N, Schmiedt C, Culp W, Rassnick K, Garrett L, et al. Outcome of 19 dogs with parathyroid carcinoma after surgical excision. Vet Comp Oncol. 2012;10(1):57-64.

Strumpf A, Selmic L, Husbands B. Evaluation of the clinical outcome of hypercalcemia of malignancy and concurrent azotemia in dogs with lymphoma. J Vet Intern Med. 2023;38:308-15.

Travail V, Motta C, Lea C, Salas-Garcia A, Clarke K, Lombardo S, et al. Plasma parathyroid hormone concentration as a predictor of postoperative hypocalcemia in dogs diagnosed with primary hyperparathyroidism and treated with parathyroidectomy. J Vet Intern Med. 2025;39(2):e70016.

Wermers RA, Griebeler ML, Thapa P, Hathcock MA, Kearns AE. Survival in primary hyperparathyroidism over five decades (1965–2010): a population-based retrospective study. Bone. 2021;152:116099.

Wilson JW, Harris SG, Moore WD, Leipold HW. Primary hyperparathyroidism in a dog. J Am Vet Med Assoc. 1974;164(9):942-6.

Xu J, Kong N, Bai N, Zhang Z, Cui A, Tan S, et al. Identification of novel risk factors for postoperative severe hypocalcemia in patients with primary hyperparathyroidism undergoing parathyroidectomy. BMC Endocr Disord. 2024;24(1):88.

Young KM, Degner DA. Surgical description and outcome of ultrasound-guided minimally invasive parathyroidectomy in 50 dogs with primary hyperparathyroidism. Vet Surg. 2023;52(1):18-25.

Zandvliet M. Canine lymphoma: a review. Vet Q. 2016;36:104-76.