**Primary Hypoparathyroidism**

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Primary hypoparathyroidism is a rare condition in dogs and cats characterized by decreased synthesis and/or secretion of parathyroid hormone (PTH). Together with 1,25-dihydroxycholecalciferol (also known as [1,25-(OH)₂D₃], calcitriol, and vitamin D₃), PTH is responsible for normal calcium homeostasis. A lack of PTH results in hypocalcemia and potentially hyperphosphatemia.

Calcium levels are maintained via hormonal influences on the intestine, kidney, and bone. PTH has direct actions on both the bone and kidney and acts indirectly on the intestinal tract. Calcium and phosphorus are released from bone through the influence of PTH on osteocytes and osteoclasts; at the renal distal tubules, PTH promotes calcium and inhibits phosphate reabsorption from the glomerular filtrate. PTH exerts an indirect effect on the intestine by stimulating renal proximal tubules to synthesize calcitriol, which then promotes calcium and phosphate absorption. Serum calcium levels are the major regulator of PTH release: Hypocalcemia is stimulatory, and hypercalcemia is inhibitory.

Failure of the parathyroid gland to respond to hypocalcemia may be related to destruction of the gland (immune mediated, surgical complication, neck trauma) or idiopathic parathyroid atrophy. The result is often neurologic or neuromuscular signs related to hypocalcemia that can vary in severity from simple weakness or anorexia to life-threatening tetany or seizures. The prognosis is excellent because most cases can be managed with vitamin D and calcium supplementation; however, complications of initial management of hypocalcemia, such as severe calcinosis cutis, have been reported.

**Diagnosis Criteria**

**Historical Information**

**Gender Predisposition**

- **Dogs:** Females.
- **Cats:** None.

**Age Predisposition**

- **Dogs:** 6 weeks to 13 years (average, 4.8 years).
- **Cats:** 6 months to 6.7 years (mean age, 1.8 years).

**Breed Disposition**

- **Dogs:** Poodles, miniature schnauzers, retrievers, German shepherds, and terriers.
- **Cats:** Domestic shorthairs.

**Owner Observations**

Abrupt or gradual onset of intermittent neurologic or neuromuscular signs; excitement or exercise may initiate or perpetuate signs:

- Seizures or tetany.
- Muscle tremors, fasciculations, or twitching.
- Facial rubbing.
- Stilted gait or ataxia.
- Prolapsed nictitating membranes (cats).
- Anorexia.
- Weakness or lethargy.
- Panting.
- Disorientation, abnormal behavior, or aggression.
- Polyuria/polydipsia.

**Other Historical Considerations/Predispositions**

- Ocular calcification formation has been reported in dogs and cats.

**Physical Examination Findings**

No specific physical examination findings have been clearly associated with primary hypoparathyroidism, other than signs attributable to hypocalcemia:

- Tremors or tetany.
- Splinted abdomen with palpation.
- Stiff gait.
- Hyperthermia.
- Bradycardia, although paroxysmal tachyarrhythmias and muffled heart sounds with weak pulses have been reported in dogs.

**Laboratory Findings**

- Hypocalcemia (low total serum calcium and/or low ionized serum calcium).
- Hyperphosphatemia.
- Low or low-normal serum PTH levels with concurrent hypocalcemia.
Other Diagnostic Findings

- Electrocardiography may reveal deep, wide T waves; prolonged QT interval, and bradycardia.

Summary of Diagnostic Criteria

- Low or low-normal serum PTH level in the face of hypocalcemia is diagnostic for primary hypoparathyroidism.
- History and physical examination findings should exclude iatrogenic causes (e.g., surgery), trauma, and a cervical mass.

Diagnostic Differentials

- Any neurologic disorder resulting in seizures, tremors, or tetany (e.g., epilepsy, meningoencephalitis, head trauma, cerebral neoplasia, toxin ingestion, hypoglycemia, tetanus, idiopathic).
- Other causes of hypocalcemia:
  - Puerperal tetany (eclampsia): Common in small dogs; rare in cats and large dogs.
  - Chronic or acute renal failure.
  - Ethylene glycol toxicity.
  - Acute pancreatitis.
  - Phosphate-containing enemas, phosphate administration, or hyperphosphatemia (uncommon).
  - Sodium bicarbonate administration (uncommon).
  - Hypomagnesemia (uncommon).
  - Laboratory error (rare).
  - EDTA-anticoagulated blood samples (rare).
  - Vitamin D deficiency (rare).
  - Intestinal malabsorption (rare).
  - Nutritional (rare).
  - Acute tumor lysis syndrome (rare).
  - Transfusion with citrated blood (rare).
  - Massive soft tissue sarcoma (rare).
  - Rhabdomyolysis (rare).

TREATMENT RECOMMENDATIONS

Initial Treatment—Emergency Management of Seizures/Tetany

- Diazepam: 0.5 mg/kg IV bolus may alleviate signs until hypocalcemia is diagnosed.

- 10% Calcium gluconate: 0.5–1.5 ml/kg or 5–15 mg/kg slow IV over 10–30 min; bolus may be repeated every 30–60 minutes as necessary.
  - The final dose can be variable and should be based on resolution of tetany.
  - Electrocardiographic monitoring should be performed during calcium infusion. Therapy should be temporarily discontinued if bradycardia, premature ventricular complexes, or shortening of the QT interval is observed.
  - Once cessation of tetany has been achieved, the total IV dose (ml) of 10% calcium gluconate used initially to control tetany may be administered SC q6–8h. When administered SC, calcium gluconate should be diluted in an equal volume (1:1) of 0.9% NaCl. The frequency of parenteral calcium supplementation can be tapered depending on the vitamin D therapy selected and the patient’s ability to tolerate enteral calcium.
- Hyperthermia secondary to tetany often resolves with control of the tetany and rarely requires direct treatment.

Alternative/Optional Treatments/Therapy

- Refractory cases may need repeated IV bolus therapy. After initial treatment of tetany, calcium may be administered as a slow IV constant rate infusion in addition to SC. A dose of 2.5 ml/kg (23 mg/kg) of 10% calcium gluconate can be added to IV fluids as

<table>
<thead>
<tr>
<th>Preparation</th>
<th>Dose Form</th>
<th>Calcium Content</th>
<th>Dosage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Injectable Calcium</td>
<td>10% Solution</td>
<td>9.3 mg/ml</td>
<td>1–1.5 ml/kg (5–15 mg/kg) IV or SC slowly</td>
</tr>
<tr>
<td>Oral Calcium</td>
<td>Tablet</td>
<td>500 mg</td>
<td>200 mg</td>
</tr>
<tr>
<td></td>
<td>Tablet</td>
<td>650 mg</td>
<td>260 mg</td>
</tr>
<tr>
<td></td>
<td>Tablet</td>
<td>1,250 mg</td>
<td>500 mg</td>
</tr>
<tr>
<td>Vitamin D (Calcitriol)</td>
<td>Capsule (0.25 and 0.5 µg)</td>
<td>N/A</td>
<td>0.03–0.06 µg/kg/day</td>
</tr>
<tr>
<td></td>
<td>Solution (1 µg/ml)</td>
<td>N/A</td>
<td></td>
</tr>
</tbody>
</table>

TABLE 1
Preparations for Acute Therapy of Hypoparathyroidism

- Requires 1–4 days for maximal effect and 1–14 days for relief of toxicity.
- N/A = not applicable.
well as administered SC q6–8h. The total dose attained should be 60–90 mg/kg/day (6–10 ml/kg/day). It should be noted that these doses reflect the amount of elemental calcium needed and that 10% calcium gluconate contains only 9.3 mg elemental calcium/ml (see Table 1). $

Supportive Treatment

- Maintenance therapy with vitamin D should be instituted as soon as possible (Table 2). Unlike vitamin D$_2$, calcitriol and dihydrotachysterol do not require renal 1-$\alpha$-hydroxylase activation; the activity of this enzyme is often low in patients with hypoparathyroidism because of the lack of PTH influence. A lack of this activation is one of the explanations regarding the high doses of vitamin D$_2$ required to correct hypocalcemia.
  
  — Calcitriol has the most rapid onset of action and shortest half-life of the vitamin D therapies. It is also the most expensive, and the minute dosages necessary for small dogs and cats may be prohibitive. $\$
  
  — Dihydrotachysterol has an intermediate onset of action and half-life. It is more expensive than vitamin D$_2$. $
  
  — Vitamin D$_3$ can be used if the above, preferred medications are too cost prohibitive. It has a long onset of action (up to 3 weeks) and, because of its long half-life, hypercalcemia may persist for weeks despite discontinuation of the drug. $
  
  — Oral calcium supplementation should be provided while initiating vitamin D therapy and tapering parenteral calcium. Once a patient is stabilized, the calcium content of commercial pet food is usually enough to maintain patients. Oral calcium supplementation should be gradually tapered over 2 to 4 months once a vitamin D dose is established. $\$

Patient Monitoring

- Calcium levels should be checked once or twice daily in patients receiving parenteral calcium.
- Patients should be hospitalized for the initiation of vitamin D therapy. A stable serum total and/or ionized calcium concentration should be observed for several days before the patient is discharged.
- Ideal serum total and/or ionized calcium levels should remain in the low-normal range with chronic therapy.
- Oral phosphate binders may be necessary because vitamin D promotes hyperphosphatemia.

Home Management

- Owners should continue to monitor their pets for signs of hypocalcemia.
- Owners should also be made aware of signs of hypercalcemia (e.g., polyuria/polydipsia, anorexia, lethargy, constipation).
- Patients' serum calcium levels should be monitored weekly initially and every 1 to 3 months after levels stabilize.

Milestones/Recovery Time Frames

- Severe calcinosis cutis has been reported with SC administration of 10% calcium gluconate, even at the recommended dilution rate (1:1 in sterile saline).
- Recovery time depends largely on the vitamin D preparation selected and its onset of action.

Treatment Contraindications

- Calcium chloride should be avoided in the management of hypocalcemia because of the potential for tissue sloughing with extravasation and development of calcinosis cutis.
- Caution should be exercised when using supplemental calcium in hyperphosphatemic patients because a calcium–phosphorus product (Ca × P) above 70 may result in soft tissue mineralization.

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<table>
<thead>
<tr>
<th>Preparation</th>
<th>Dose Form</th>
<th>Dosage</th>
<th>Time to Maximal Effect</th>
<th>Time to Toxicity Relief</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Dihydrotachysterol</strong></td>
<td>Tablet (0.125, 0.2, and 0.4 mg)</td>
<td>Initial: 0.02–0.03 mg/kg/day</td>
<td>1–7 days</td>
<td>1–3 wk</td>
</tr>
<tr>
<td></td>
<td>Capsule (0.125 mg)</td>
<td>Maintenance: 0.01–0.02 mg/kg</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Solution (0.25 mg/ml)</td>
<td>q24–48h</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Vitamin D$_3$</strong></td>
<td>Capsule (25,000 and 50,000 U)</td>
<td>Initial: 4,000–6,000 U/kg/day</td>
<td>5–21 days</td>
<td>1–18 wk</td>
</tr>
<tr>
<td></td>
<td>Syrup (8,000 U/ml)</td>
<td>Maintenance: 1,000–2,000 U/kg</td>
<td></td>
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<tr>
<td></td>
<td></td>
<td>once daily–once weekly</td>
<td></td>
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</tbody>
</table>


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TABLE 2
Preparations for Maintenance Therapy of Hypoparathyroidism$^a$

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A U G U S T  2 0 0 6  V O L U M E  8 . 7
• IV fluids containing lactate, bicarbonate, phosphorus, or acetates should be avoided during calcium therapy.

PROGNOSIS

Favorable Criteria
• Prognosis is generally excellent when owners comply with administration of medications and careful follow-up monitoring.

Unfavorable Criteria
• Development of severe calcinosis cutis.
• Dystrophic mineralization caused by calcium–phosphorus product (Ca × P) above 70.

RECOMMENDED READING