Calcium Oxalate Urolithiasis and Hyperlipidemia – Canine

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Definition

Hyperlipidemia is defined as an increase in serum concentrations of triglyceride (TG), cholesterol, or both. In ***calcium oxalate urolithiasis***, uroliths composed of calcium oxalate monohydrate, dihydrate, or both form in the dog's urine. Calcium oxalate uroliths are often found in dogs with concurrent hyperlipidemia, typically ***hypertriglyceridemia***. For more on hyperlipidemia in dogs, see pages 80–81; for more on calcium oxalate urolithiasis, see pages 88–89.

Key Diagnostic Tools and Measures

Serum or plasma calcium concentration is checked to evaluate for hypercalcemia. Serum or plasma triglyceride and cholesterol concentrations may require a minimal 12-hour fast prior to collection. In the refrigeration test, collect a serum sample after a 12-hour fast and refrigerate the sample for 12 hours. If a creamy layer collects on top of clear serum, then hyperchylomicronemia is present. If the sample is turbid, then other lipoproteins are present. Endocrine testing for hyperlipidemia includes thyroid testing for hypothyroidism, adrenal gland testing for hyperadrenocorticism, and blood and urine glucose and ketones for diabetes mellitus. Chronic pancreatitis may result in hyperlipidemia; therefore, testing includes determination of serum or plasma amylase and lipase activities, serum canine pancreatic lipase immunoreactivity, and abdominal ultrasonography. A serum lipoprotein profile can be performed to further differentiate the concentration of lipoproteins present.

Pathophysiology

Hyperlipidemia does not cause calcium oxalate urolith formation; however, certain associations occur. Miniature schnauzers have a breed predisposition to hyperlipidemia and calcium oxalate urolithiasis. The etiology is unknown; however, lipoprotein lipase deficiency or decreased activity is suspected.

Hyperadrenocorticism can cause both conditions to occur. Hyperlipidemia occurs because of insulin resistance induced by hypercortisolemia resulting in alteration in lipid metabolism. Hypercortisolemia promotes hypercalciuria, which results in urinary oversaturation for calcium oxalate and possibly urolith formation.

Signalment

Miniature schnauzers have a breed predisposition to primary hyperlipidemia and calcium oxalate urolithiasis. Breeds at risk for hyperadrenocorticism include, but are not limited to miniature schnauzers, miniature and toy poodles, dachshunds, and Boston terriers.

Key Nutrient Modifications

A low-fat diet may help decrease the degree of hyperlipidemia. A high-fiber diet may decrease absorption of fat. Fiber may also reduce the absorption of calcium from intestinal tract, but currently there is no evidence that this beneficially influences urinary calcium. Low-calcium diets are a risk factor for calcium oxalate urolithiasis so should be avoided. Increased sodium intake may decrease urinary saturation for calcium oxalate. Increased urine volume may reduce the risk for urolithiasis so water intake should be encouraged. Increased water intake may be promoted by feeding wet food, or adding water to a canned or dry food.

Recommended Ranges of Key Nutrients

Nutrient	% DM	g/100 kcal	% DM	g/100 kcal
	Recommended dietary level		Minimum dietary requirement*	
Fat	7–12	1.5–3.5	5	1.4
Fiber	7–14	2–8	n/a	n/a
Calcium	0.6–1.5	0.2–0.35	0.6	0.17
Sodium	0.2–1.5	0.06–0.35	0.06	0.02

Modified intake of these nutrients may help address metabolic alterations induced by disease states. The recommended dietary composition is shown as percent of dietary dry matter (DM) and as g or mg per 100 kcal metabolizable energy. All other essential nutrients should meet normal requirements adjusted for life stage, lifestyle, and energy intake. Increased water intake should be encouraged. Use of canned, high moisture diets or sodium-supplemented diets may help increase water intake.

*Nutrient requirement for adult animals as determined by the Association of American Feed Control Officials

Therapeutic Feeding Principles

With primary hyperlipidemia, dietary fat restriction is recommended with or without higher fiber. Increased fiber is sometimes recommended for the management of calcium oxalate uroliths although no studies have been published to support this. If hyperlipidemia is secondary to endocrine disease, management of disease is the main treatment. Dietary management to minimize risk factors that promote recurrent calcium oxalate disease also is indicated.

- **Treats** – Avoid high-fat treats.
- **Tips for Increasing Palatability** – Water can be added to food to increase palatability.
- **Diet Recommendations** – A high-fiber, low-fat diet or a very low-fat diet is recommended for dogs with hyperlipidemia. Dogs with calcium oxalate urolithiasis should be fed a non-acidifying diet that reduces calcium oxalate urinary supersaturation. A high-moisture, low-fat diet would be recommended for the patient with both of these problems.

Client Education Points

- Hyperlipidemia (high levels of circulating fat in blood) can occur as a primary disease, as in miniature schnauzers, or secondary to other diseases, such as hyperadrenocorticism.
- Calcium oxalate uroliths occur when urine contains high levels of calcium and/or oxalate. This can occur in association with diseases that cause hyperlipidemia.
- Clients should avoid giving supplements containing calcium or vitamin C, as well as human-food treats high in oxalate.

Common Comorbidities

Hyperadrenocorticism, hypothyroidism, chronic pancreatitis, seizures with hypertriglyceridemia, and hyperparathyroidism, if hypercalcemic, are common comorbidities in dogs with hyperlipidemia and calcium oxalate urolithiasis.

Interacting Medical Management Strategies

For the hyperlipidemia, the following may be required if dietary management does not appropriately decrease triglyceride concentrations: omega-3 fatty acids (may decrease synthesis of certain lipoproteins; 10–30 mg/kg orally (PO) every 24 hours); gemfibrozil (decreases certain

lipoprotein and triglyceride synthesis; 100–300 mg PO every 12 hours); chitin or chitosan (may bind certain dietary lipids in the intestinal tract thereby decreasing their absorption; no good studies in dogs; 150–300 mg PO 30 minutes prior to feeding); and niacin (reduces triglyceride synthesis; vasodilatory resulting in erythema and pruritus; 25–150 mg PO every 12 hours).

For calcium oxalate urolithiasis, potassium citrate, a urinary alkalinizing agent, can be given to increase calcium oxalate solubility with alkaluria. Citrate may inhibit calcium oxalate crystal formation and aggregation at a dose of 50–100 mg/kg PO every 12 hours; adjust to urine pH of approximately 7.5. Thiazide diuretics increase distal renal tubular reabsorption of calcium resulting in lowered urinary calcium excretion, but can result in hypercalcemia. There are no long-term studies in dogs on safety or efficacy. Hydrochlorothiazide is given at 2 mg/kg PO every 12 hours. Vitamin B6 is involved with oxalate metabolism, although there is no evidence that vitamin B6 supplementation provides any benefit in dogs.

Monitoring

For hyperlipidemia, check for resolution of clinical signs (if present). Fasting triglyceride and cholesterol concentrations should be monitored; if secondary to endocrine disease, treatment for that disease should be monitored.

For calcium oxalate urolithiasis, urinalysis should be done monthly for 3 to 6 months to monitor response to treatment; pH should be neutral to alkaline, specific gravity should be dilute, and crystalluria should be absent. Survey abdominal radiography or ultrasonography should be performed at 6 and 12 months and then every 6 to 12 months depending on response. Serum calcium should be monitored 1 month after starting hydrochlorothiazide and then every 3 to 6 months. If secondary to endocrine disease, appropriate monitoring of management is indicated.

Algorithm – Nutritional Management of Concurrent Canine Calcium Oxalate Uroliths and Hyperlipidemia