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LONG ISLAND VETERINARY SPECIALISTS

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Long Island Veterinary Specialists
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Feline Idiopathic Hypercalcemia

Joshua W. Tumulty, DVM, DACVIM
Dept of Internal Medicine

Over the last 2 decades, idiopathic hypercalcemia has emerged to become the most common type of hypercalcemia in cats. Cats with idiopathic hypercalcemia range in age from very young to geriatric, and longhaired cats appear to be over-represented. The diagnosis is established by the laboratory findings of elevated serum total and ionized calcium concentrations with low to low-normal PTH values, after excluding other less common causes of hypercalcemia (primarily cancer).

Because the pathogenesis for idiopathic hypercalcemia remains unknown, treatment for this condition remains difficult. Response to dietary changes have produced mixed results, and most cats eventually require medical management to control the hypercalcemia. Recently, the utilization of an oral bisphosphonate (i.e., alendronate; Fosamax) has become a more consistent form of therapy.

Total serum calcium cannot be reliably used to predict the metabolically active ionized calcium fraction in cats. An overall diagnostic discordance of 40% during evaluation of hypercalcemic cats was demonstrated in one study. The extent of hypercalcemia known to exist when ionized calcium is measured is underdiagnosed when serum total calcium is used as the screening test for calcium disturbances. Thus, screening cats for calcium metabolic disorders is better served by the measurement of ionized calcium. Once ionized hypercalcemia has been identified, the next step is to determine if the process is PTH-dependent (high PTH from failure to suppress abnormal parathyroid glands) or PTH-independent (PTH is appropriately suppressed as the response of normal parathyroid glands) **Figure 1**. Compared to dogs, cats have a higher frequency of PTH-independent hypercalcemia. PTH must always be interpreted along with ionized calcium in the same sample in order to determine appropriateness of the PTH concentration. In those with parathyroid-independent hypercalcemia, malignancy-associated hypercalcemia (MAH) needs to be excluded. MAH most often results from humoral mechanisms as the tumor secretes calcemic substances such as PTHrP into the

"Cats with idiopathic hypercalcemia range in age from very young to geriatric, and longhaired cats appear to be over-represented."

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Michel Selmer, DVM, CTCVMP (Integrative Medicine)

Dr. Michel Selmer is an Integrative Veterinarian and one of only a handful of Traditional Chinese Veterinary Medicine Practitioners in the United States.

Dr. Michel Selmer attended Long Island University and graduated Cum Laude with a Bachelor of Arts Degree in Psychology. Following his undergraduate studies, he was admitted to Michigan State University School of Veterinary Medicine and earned his Doctorate of Veterinary Medicine in 1995. Following his Traditional Veterinary studies, he was admitted to the Chi Institute where he graduated with the top honor of being a Certified Traditional Chinese Veterinary Medicine Practitioner (CTCVMP).

Dr. Selmer is a published author and consults with other veterinarians as well as pet parents around the globe. In 2018, he made the exciting decision to join the Long Island Veterinary Specialists team as the Lead Veterinarian in their Integrative Medicine Department.

The passion Dr. Selmer has for his profession - and his love for all animals - has contributed to the high quality medicine that he practices.

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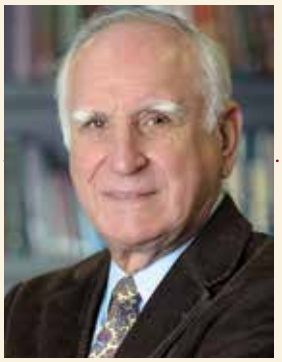
A Note from the Editor



We are elated to share the "glad winter's over" feeling with many Long Islanders; happy to put our snow blowers away and look forward to warm days and spring colors. The last one was the 4th "Nor'easter" this year and everyone was glad the storm was followed by above freezing temperatures allowing the white stuff to melt quickly.

We welcomed to the LIVS family another IT team member recently, Chris Kuscsik and he and Spiro are keeping communications of all sorts humming. The March lunch was held on the 22nd and featured Greek specialties and colorful decorations, a real treat for our staff.

On the "Ides of March", Dr. Dominic Marino, our chief of staff, presented a talk to the NY FOP (Fraternal Order of Police) at Westbury Manor, covering LIVS' work with the training and care of police and military dogs. It included an exhibition of the original presence of LIVS' personnel at the WTC events on 9-11. A week later, a group of 30 veterinarians from China visited LIVS and were treated to a presentation on the pioneering advances started at LIVS. They were impressed with the retinal surgery and hip replacement programs.

Members of the staff participated in the annual "St. Baldrick's" event, a non-profit foundation which stands in solidarity with kids fighting cancer, raising money to find cures. This volunteer-driven charity funds more in childhood cancer research grants than any organization except the U.S. government. At the shearing portion, so much hair was removed from Steve Sadowski's chin and head that it transformed him in one brief session, all for the kids. A joyous party was held following the event and good times shared.

LIVS continues to uniquely offer Medical Infrared Imaging, 3 Tesla MRI, Brachytherapy and Nano/Micro Total Hip replacements among its many services to the veterinary community.

Our extended hours for consultation at LIVS, including our newest, the Behavior Department, led by Dr. Sabrina Poggiagliolmi —"Dr. Sabrina"— is designed to serve our clients more efficiently. Appointments can be made through our telephone receptionists at 516 501-1700.

On a regular basis, Dr. Curtis Dewey, associate professor and section head of Neurology/ Neurosurgery at the College of Veterinary Medicine at Cornell is here at LIVS for consultation. Appointments can be made also at 516-501-1700.

We continue to welcome input and opinions which can be directed to the editor at lmarino@livs.org.

Leonard J. Marino, MD, FAAP, LVT

Feline Idiopathic Hypercalcemia

➤ *Continued from Front Cover*

Figure 1

Biochemical Parameters* Expected with the Common Causes of Feline Hypercalcemia

Disorder	tCa	iCa	Creatinine	Phosphorus	PTH	PTH-rP	Vitamin D**
Renal failuire	N, ↑, ↓	N, ↓, occ ↑	↑	↑, N	occ ↑	N	N, ↓
Primary hyperparathyrodism	↑	↑	N, occ ↑	↓, occ ↑	↑, occ N	N	N
Neoplasia (nonosteolytic)	↑	↑	N, occ ↑	↓, occ ↑	↓	↑, N	N
Idopathic	↑	↑	N	N	N	N	N
Vitamin D toxicosis	↑	↑	N, ↑	N, ↑	↓	N	↑

* These parameters are generalizations. Individual cases may not fit these patterns.
++ 25-hydroxycholecalciferol

↑ = increased; ↓ = decreased; N = normal; occ = occasional;
PTH = parathyroid hormone; PTH-rP = parathyroid hormone-related protein

circulation; local osteolytic hypercalcemia is far less common. A low or undetectable PTHrP does not exclude malignancy as the cause for hypercalcemia since other cytokines that cause calcemia can be elaborated by the tumor instead of PTHrP on occasion. Idiopathic hypercalcemia (IHC), CKD, and neoplasia are the most common and important differential diagnoses to exclude as the cause for parathyroid-independent hypercalcemia. Overt hypervitaminosis D, granulomatous disease, and hypoadrenocorticism are other far less common causes of hypercalcemia in cats. IHC is currently the most common cause of hypercalcemia in cats. While MAH is the number one cause of pathological hypercalcemia in the dog, it occurs far less frequently in the cat. Patients with MAH are usually "sick," as it takes a reasonably large tumor burden to synthesize the messengers that result in hypercalcemia. MAH is less likely to be the diagnosis if the hypercalcemia persists for a long period of time without the cat showing more clinical signs. The less sick the cat is in the face of persistent ionized hypercalcemia, the more the likelihood for the diagnosis to be that of IHC or primary hyperparathyroidism.

In many cases the diagnosis will be obvious upon analysis of history and physical examination. In others, the cause may not be obvious, and further workup including hematology, serum biochemistry, body cavity imaging, cytology, and histopathology will be necessary. The magnitude of elevation of serum total calcium concentration cannot be used to make a diagnosis, as there is considerable overlap in the degree of hypercalcemia in cats with idiopathic hypercalcemia or other conditions. Nearly ½ of patients diagnosed with IHC are asymptomatic. Other clinical signs have included gastrointestinal signs, including mild weight loss, chronic constipation, vomiting, and decreased appetite. Lower urinary tract signs may be observed, especially if urolithiasis is present. A diagnosis of idiopathic hypercalcemia is made when all other causes of parathyroid-independent hypercalcemia are excluded **Figure 2**.

Management of IHC usually begins with a dietary recommendation to attempt to restore normocalcemia. Treatment with bisphosphonates and glucocorticosteroids usually are reserved for cats with IHC that fail dietary treatment. It has not been determined how much of the hypercalcemia in IHC cats results from too much dietary calcium intestinal absorption, increased bone resorption, reduced renal excretion of calcium, or combinations of these processes. In order to rationally choose a diet that is lower in calcium intake, a complete diet history must be obtained; this includes the cat's primary diet, treats, and any supplements. Once this starting dietary calcium concentration has been determined, the veterinarian can determine which diets would provide

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Figure 2

- Always verify hypercalcemia with a repeat total calcium (tCa) or ionized calcium (iCa) concentration measurement.
- An elevated iCa concentration defines true hypercalcemia.
- Measure the iCa concentration in any cat with a tCa concentration at or above the upper end of the reference range.
- Measure the iCa concentration in any cat with renal failure or calcium oxalate urolithiasis, even if its tCa concentration is normal.
- Severe hypercalcemia mandates immediate attention, and therapy to reduce the serum calcium concentration may be necessary while further diagnostic tests are performed.
- Most causes of feline hypercalcemia can be diagnoised with a straightforward work-up.
- Idiopathic hypercalcemia can only be diagnoised after all other causes have been excluded.

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➤ *Continued from Page 6*

less calcium, considering potential comorbid conditions. A veterinary therapeutic renal diet low in calcium (and phosphorus) content may be appropriate for a cat with hypercalcemia and CKD, but it may not be appropriate to feed a young, otherwise healthy cat a reduced phosphorus, reduced protein diet. Feeding of a high protein and low carbohydrate food similar to what cats would eat in the wild has been recommended to effectively lower serum calcium concentration in some cats with IHC, especially those with low magnitude hypercalcemia. No matter what type of diet is chosen, it is best to feed a wet-only diet to promote urinary dilution and lessen the chance for calcium oxalate stones to form. Unfortunately, in most cats with idiopathic hypercalcemia, dietary therapy will be of minimal long-term benefit, but this management is very unlikely to be harmful. In addition, because many cats with idiopathic hypercalcemia do not show clinical signs for months to years, it is appropriate to try dietary therapy as the first line of therapy at least in cats with mild ionized hypercalcemia. Ionized calcium should be measured following 4 to 6 weeks of feeding the new diet. In some cats, there will be a dramatic decrease in ionized calcium to within the reference range or closer to it. Continued feeding of the newly recommended diet should be continued for another 2 to 3 months to see if any observed benefits are sustained. It is possible that feeding of a new diet will require 2 to 3 months to see maximal benefits, if any. Consideration for treatment with bisphosphonates or prednisolone should be considered for those with high levels of ionized hypercalcemia and/or clinical signs associated with hypercalcemia not responding to dietary change.

The oral bisphosphonate alendronate has become the preferred option to treat IHC cats after dietary modification has failed to restore normocalcemia. Treatment with bisphosphonates may be useful to decrease the magnitude of hypercalcemia in cats with IHC by altering osteoclastic bone resorption. Bisphosphonates reduce the activity and number of osteoclasts following binding to hydroxyapatite. Any food in the stomach can drastically reduce the absorption of alendronate. To maximize intestinal absorption of alendronate, it is recommended that cats be fasted overnight for 12 hours prior to the administration of medication, giving the pills in nothing other than tap water, and then feeding the cat two hours later. A risk of esophagitis and stricture associated with oral bisphosphonate treatment has been reported in humans. The starting dose is usually 10 mg/cat per week initially. It is recommended that a whole tablet be administered, as cut tablets may increase exposure of the esophagus and stomach to adverse effects. Serum ionized calcium levels should be checked at 2 weeks, 1 month, 2–3 months, and then every 4–6 months as long as the ionized calcium concentration remains within normal limits. Most cats seem to have at least a partial calcium-lowering response to alendronate. The drug dose can be raised to up to 30–40 mg per week, as needed. In resistant cats, prednisolone and alendronate can be used together. Administration of glucocorticoids can decrease serum calcium concentration by decreasing intestinal absorption of calcium, by decreased renal tubular calcium reabsorption, and decreased skeletal mobilization of calcium. Prednisolone is given orally at 5 mg/cat/day for one month before re-evaluation. One disadvantage of prednisolone is that approximately a third of cats will develop secondary diabetes, especially with long-term, high-dose steroid therapy. The long-term safety and efficacy of oral alendronate therapy has not been reported in cats. Mild hypocalcemia is sometimes encountered during oral alendronate treatment of IHC in cats, but overt clinical signs are not usually encountered. Drug-induced esophageal damage (erosive esophagitis and esophageal stricture) and gastritis are of concern in humans taking oral bisphosphonates. An increased risk for bone fracture and osteonecrosis of the jaw (ONJ) has been reported in humans on long-term bisphosphonate treatment, presumably because of the increased brittleness of bone due to bisphosphonate therapy.

Alendronate treatment should be stopped in IHC cats that fail to regain normocalcemia despite 30- to 40-mg weekly doses. It is not known how long oral alendronate treatment should be continued in those IHC cats that have regained normocalcemia for long periods of time. It may not be enough to just monitor calcium and renal function status in IHC cats during treatment interventions. Including baseline long-bone radiographs for all IHC cats being treated with oral bisphosphonates for more than one year, and then yearly thereafter to more readily detect early bone injury that may be developing may need to be considered. ❑

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Laryngeal Paralysis

Fernando Leyva, DVM, Staff Surgeon

LARYNGEAL PARALYSIS : What is Geriatric Onset Laryngeal Paralysis Polyneuropathy (GOLPP)?

Laryngeal paralysis is the failure of the arytenoid cartilages to properly abduct secondary to muscle or nerve damage. Neurogenic atrophy of intrinsic laryngeal muscles, particularly the cricoarytenoideus dorsalis muscle, causes failure of the arytenoid cartilages and vocal folds to abduct, resulting in upper airway obstruction. Laryngeal paralysis is characterized by clinical signs of inspiratory stridor, exercise intolerance and respiratory distress. Clients often report voice change, coughing, and/or gagging; however, severely affected patients may present in respiratory distress, cyanosis and collapse. Clinical signs are often not evident until the disease is bilateral in dogs. Although this condition can occur it cats, its is far more common in dogs and should be considered as a possible differential diagnosis for animals with the above mentioned clinical signs. Unlike in dogs, cats with unilateral laryngeal paralysis can present with significant clinical signs.

Laryngeal paralysis is characterized by clinical signs of inspiratory stridor, exercise intolerance and respiratory distress.

Etiology

Congenital laryngeal paralysis

Congenital laryngeal paralysis is uncommon. It has been reported in Bouviers des Flanders, Bull terriers, Dalmatians, Rottweilers, Pyrenean Mountain Dogs, Huskies, Leonbergers, Italian Spinoni, Russian Black Terrier, Alaskan Malamute, Miniature Schnauzers and Tibetan Mastiff. Dalmatians and Huskies are most commonly reported in the United States. The onset of clinical signs in dogs with congenital laryngeal paralysis is usually before 1 year of age. An association between laryngeal paralysis and generalized polyneuropathy has been reported in young Dalmatians.

Acquired laryngeal paralysis

Acquired laryngeal paralysis can occur secondary to trauma, cervical/thoracic masses, neuromuscular disease (immune-mediated, endocrinopathy), or idiopathic. Acquired laryngeal paralysis is a common condition of older large and giant breed dogs. It is most commonly reported in Labrador and Golden Retrievers, Saint Bernards, and Irish Setters; the median age of affected dogs is 9 years. There is an association between acquired laryngeal paralysis and hypothyroidism, but a direct link has not been determined. For dogs diagnosed with concurrent hypothyroidism, thyroid supplementation should be instituted, but this typically does not improve clinical signs of laryngeal paralysis, however, thyroid supplementation could help prevent or slow the progression of possible hypothyroid-induced generalized neuromuscular disease that may cause peripheral weakness. If an underlying cause of laryngeal paralysis cannot be determined, then these cases are deemed idiopathic. Acquired idiopathic laryngeal paralysis is the most commonly encountered form of the disease in practice. Recent studies by Stanley et al. and Thieman et al. seem to confirm that acquired idiopathic laryngeal paralysis is part of a polyneuropathy affecting other muscle groups other than the laryngeal musculature. From these studies, it appears that idiopathic laryngeal paralysis cases are in fact presenting with chronic, progressive polyneuropathy, and that until the disease is better understood, it may be better referred to as "geriatric onset laryngeal paralysis polyneuropathy" (GOLPP) syndrome.

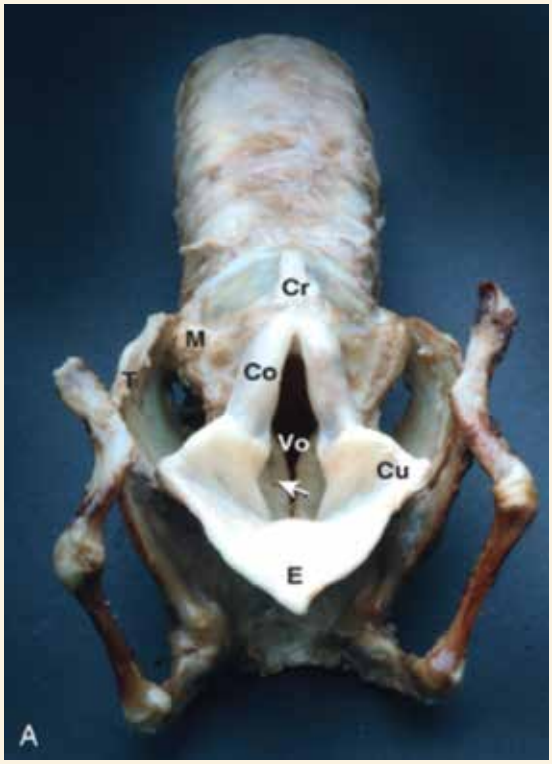

Figure 1: Biochemical parameters expected with common causes of feline hypercalcemia

Diagnosis

Suspicion of laryngeal paralysis arises from the history of the dogs as well as consistent clinical signs. Definitive diagnosis of laryngeal paralysis requires visual examination of the larynx under a light plane of anesthesia. By definition, laryngeal paralysis is the absence of arytenoid abduction on inspiration. In particular, the corniculate process of the arytenoid cartilages that frame the rima glottidis dorsally are observed for purposeful motion. In **Figure 1** the corniculate process of the arytenoid cartilage is labeled "Co". The clinician should not confuse the presence of paradoxical movement of the arytenoids, which can lead to false negative results. In paradoxical movements, the arytenoid cartilages move inward during inspiration owning to negative intraglottic pressure that is created by breathing against the obstruction. The cartilages then return to their original position during the expiratory phase, giving the impression of abduction. What cannot be overemphasized is the importance of correlating any laryngeal movement with the phase of respiration. The author recommends that an assistant aid in stating the stage of respiration (i.e. saying "in" for inspiration) so that the clinician doing the laryngeal examination can differentiate normal from abnormal motion. If laryngeal paralysis is strongly suspected, it is advantageous to schedule laryngoscopy so that surgery can be performed immediately after if the diagnosis is confirmed.

Treatment

Surgery is the treatment of choice for

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animals with moderate to severe clinical signs or a decreased quality of life. Dogs that are asymptomatic at rest or mildly affected can occasionally be managed with weight loss, stress reduction, exercise restriction, and avoidance of high ambient temperatures. Several surgical techniques have been developed for treatment of laryngeal paralysis. Variations of unilateral arytenoid lateralization (cricoarytenoid laryngoplasty) are the most commonly performed procedures and are the procedures of choice of many surgeons. **Figure 2** depicts suture placement for cricoarytenoid laryngoplasty. Other surgical techniques include partial laryngectomy (partial arytenoidectomy with vocal fold resection) and modified castellated laryngofissure with vocal fold resection.

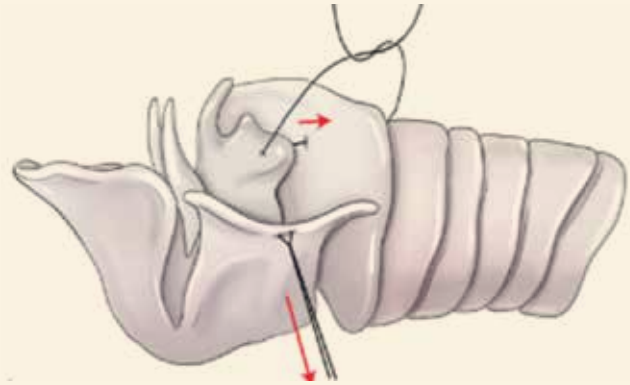

Figure 2: Guidelines for evaluating hypercalcemic cats

Prognosis

The long-term prognosis for patients with laryngeal paralysis is based on etiology and if surgery is pursued. The prognosis for dogs with congenital laryngeal paralysis is poor, especially in those with concurrent polyneuropathy at an early age. The prognosis for dogs with acquired laryngeal paralysis is generally good if cancer is ruled out, there is no evidence of megaesophagus (or any cause of regurgitation), and proper surgical treatment is performed. Postoperative aspiration pneumonia has been reported to occur in 8 to 19% of dogs following unilateral arytenoid lateralization. Although aspiration pneumonia is most likely to occur in the first few weeks following surgery, these dogs have a potential life-long risk. Factors that have been significantly associated with a higher risk of complications include preoperative aspiration pneumonia, postoperative megaesophagus, temporary tracheostomy tube placement, and concurrent neoplastic disease.

Clinicians should be aware that while laryngeal paralysis may be the only presenting clinical sign in acquired idiopathic laryngeal paralysis, it could be the only clinical sign of a generalized neuromuscular disorder. Although dogs diagnosed with laryngeal paralysis may eventually develop clinical signs of a generalized polyneuropathy, the author believes that this possibility should not discourage veterinarians from performing corrective surgery for the laryngeal paralysis, because the polyneuropathy is usually slowly progressive. Owners however, should be informed of the possibility of progressive polyneuropathy and the increased risk of developing aspiration pneumonia after the procedure. ❑

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