Veterinary professionals attended the 2013 American College of Veterinary Internal Medicine (ACVIM) Forum to discuss recent advancements in practice and research. The forum also explored One Medicine topics as part of ACVIM’s efforts to foster research for both animal and human healthcare.

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Mycoplasmas & Novel Viral Pathogens in Canine Infectious Respiratory Disease

The detailed pathogenesis of canine infectious respiratory disease (CIRD) is complex and poorly understood, partially because of its multiple causes. Some microbes can overcome the innate and adaptive immune system barriers, enabling bystander access to other microbes. Respiratory coronaviruses and mycoplasmas are notable invaders. The authors discovered a novel coronavirus and clinical association with canine mycoplasmas; they suggest there is a mechanism by which respiratory coronaviruses can potentiate bacterial superinfection with mycoplasmas. Several novel viruses (eg, canine pneumovirus, canine hepatitis, and canine bocavirus) have recently been discovered in dogs with respiratory disease; many Mycoplasma spp infections of dogs with respiratory disease are underdiagnosed. Whether M. cynos is a primary or secondary pathogen in the CIRD complex is unknown, but ongoing investigation suggests it is capable of both.—Brownlie J, Mitchell JA, Walker CA, Erles K

Top Ten Treatment Tips for Feline Heart Disease: Feeding & Pharmacology

There are several tips for addressing feline heart disease: 1) Body and muscle condition scores should be determined at every visit. 2) The optimal dose of furosemide should clear significant edema while avoiding adverse effects. 3) Major dietary changes should be avoided during hospitalization, as forced changes can induce food aversions. 4) Diet should be reevaluated at each visit. 5) The larger the left atrium, the higher indication for an ACE inhibitor. 6) The larger the left atrium, the more an antithrombotic should be considered. Unfractionated heparin can be used at high doses (275–350 IU/kg q6–8h) for active arterial thromboembolism (ATE), but long-term use is usually not advised. Low molecular weight heparins (eg, dalteparin, enoxaparin) and clopidogrel (Plavix) have been used to prevent thrombus formation in cats at risk for ATE. 7) Owners should be made aware that the cat’s dietary preferences may vary and that options should be provided. 8) Treats and supplements should be addressed. 9) Pimobendan can be used in most cats for management of chronic congestive heart failure, but not before onset of signs or in cats with asymptomatic hypertrophic cardiomyopathy. 10) Some factors may reduce quality of life (eg, respiratory distress, weakness after arterial embolism, drug adverse effects, veterinary visits, altered interactions with humans or other animals).—Freeman LM, Rush JE

Treatment of Cushing’s Disease in the Dog: Arguments For & Against

Potential adverse effects, cost of treatment, degree of illness, quality of life, and expected survival time are often considered when deciding whether to treat hyperadrenocorticism (HAC). In some cases, owner quality of life can be a factor (eg, a frustrated owner may request euthanasia of a severely polyuric dog unless the problem can be resolved).
Studies indicate the lifespan of a dog with pituitary-dependent hyperadrenocorticism (PDH) can be influenced by type of treatment (eg, selective or nonselective adrenocorticolysis, dopamine agonist therapy with cabergoline, transphenoidal hypophysectomy) and early treatment. Recent studies have shown that trilostane at low dosages (0.5–1 mg/kg q12h) can reduce the prevalence of adverse effects to 4%. Complications associated with HAC include chronic urinary tract infections, thromboembolic disease, neurologic impairment from an enlarging pituitary tumor, hypertension, proteinuria, renal tubular disease, chronic pyoderma and calcinosis cutis, calcium uroliths, acute blindness, diabetes mellitus, and biliary mucoceles. Abdominal ultrasonography is recommended in all dogs with suspected Cushing’s disease to screen for biliary mucoceles; any animal diagnosed with a biliary mucocele should be evaluated for underlying HAC. Hypertension and proteinuria are common complications with HAC; however, treatment generally does not resolve hypertension and sometimes does not resolve proteinuria. —Nichols R

Diagnosis of Canine & Feline Myopathies
Myopathies can be acquired (eg, immune-mediated, inflammatory) or inherited (eg, muscular dystrophies, congenital myopathies). Regardless of underlying cause and type, prompt and accurate diagnosis is essential to preventing irreversible damage and determining treatability, prognosis, and appropriate therapy. Myopathy should be a differential diagnosis for dogs with weakness; muscle atrophy or hypertrophy; or stiff, short-strided gait. Minimum database for suspected myopathies should include CBC; serum biochemistry profile; urinalysis; serum creatine kinase (CK), cardiac troponin I, and plasma lactate measurements; and determination of thyroid status. CK is specific for myofibril damage. Higher levels indicate necrotizing myopathies or muscular dystrophies; moderate elevations are seen with inflammatory myopathies, mild elevations with focal, endocrine, and other congenital myopathies. Causes independent of disease can elevate CK levels (eg, intramuscular injections, external muscle damage, trauma, surgery, prolonged recumbency, anorexia [cats]). Myopathy should be considered with persistent CK elevations and when external causes are ruled out. Cardiomyopathies may be present with certain myopathies; measurement of cardiac troponin I, a protein marker of myocardial cellular injury, may aid diagnosis. Muscle biopsies should be collected before irreversible fibrosis and muscle contracture. Studies to broaden the list of DNA-based tests for determining breed-specific neuromuscular diseases are underway.—Shelton GD

Maximal Reduction of Proteinuria: Beyond ACE Inhibitors
Increased protein excretion (and other causes of end-stage renal disease) is the main clinicopathologic abnormality in dogs with protein-losing nephropathies. Activation of the renin-angiotensin-aldosterone system (RAAS) is responsible for many changes associated with end-stage disease. Inhibition of angiotensin-converting enzyme (ACE) activity decreases serum concentrations of angiotensin II and aldosterone, which also reduces intraglomerular hydrostatic pressure and systemic blood pressure. ACE inhibitors are meant to reduce protein loss in PLN disease but can worsen azotemia by reducing glomerular filtration rate (GFR). Enalapril may be the most commonly used ACE inhibitor in dogs. Instituting therapy and carefully monitoring azotemia in dogs is important, adjusting doses accordingly. Alternative ACE inhibitors (eg, benazepril) may be tried in cases that develop refractory adverse effects associated with enalapril, but these drugs have not been compared in dogs with naturally-occurring disease. RAAS activity can also be blocked by administering angiotensin II-receptor blockers (ARBs) (eg, losartan [Cozaar, merck.com]). These drugs may help patients that are refractory to ACE inhibition. In humans, aldosterone receptor antagonists (eg, spironolactone) reduce proteinuria and stabilize kidney function if used with ACE inhibitors and ARBs; anecdotal experience suggests spironolactone is not very effective in reducing proteinuria in dogs with glomerular disease. Low-protein diets may be useful in conjunction with ACE inhibitors.—Pressler B cb

Save the Date
The next ACVIM Forum will be held June 4–7, 2014, in Nashville, Tennessee.

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