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RESPIRATORY DISEASE IN DOGS & CATS - 4 PAGES

Respiratory therapy is most effective if based upon cytology and culture/sensitivity results obtained from a bronchoalveolar lavage (BAL) or a transtracheal wash. The results from this procedure also define the level of therapy needed to control or resolve the pathology. Respiratory symptoms can have infectious, allergic, parasitic, foreign body, fungal and neoplastic origins. Efficacious therapy maximizes precise targeting of the causative agent and minimizes suppression of the body's own defense mechanisms (muco-ciliary activity, BAL, reflexes).

Acute Respiratory Distress Syndrome must be kept in mind in cases of acute respiratory distress that can mimic congestive heart failure in dogs and cause non-cardiogenic pulmonary edema. The following risk factors were presented by Dr. DeClue at ACVIM 2008:

Risk factors associated with ARDS in veterinary medicine

Direct pulmonary injury Indirect pulmonary injury

Microbial pneumonia

Parasitic pneumonitis

Aspiration pneumonia

Smoke inhalation

Strangulation

Pulmonary contusions

Hyperoxia

Lung lobe torsion

Sepsis

Babesiosis

Parvovirus

SIRS

Paraquat poisoning

Pancreatitis

Shock

Gastric and splenic torsion

Trauma

Multiple transfusions

Bee envenomation

Disseminated intravascular coagulation

Interstitial Lung Disease in dogs

Clinical Signs of Interstitial Lung Disease—can mimic signs of cardiac disease as well as signs of chronic bronchitis; clinical signs include progressive exercise intolerance, tachypnea, coughing and collapse.

Interstitial lung disease often occurs in terriers that are barrel chested. Subsequently, these patients will have plump/larger looking hearts given their confirmation. This can be exaggerated by the phase of respiration that the radiograph is taken in.

Diagnostics:

BAL (looking for lipids and proteins; can also perform aerobic and anaerobic culture and sensitivity to rule out bacterial, fungal, parasitic, neoplastic disease processes)

Evaluating drug history

Echocardiogram (to rule out presence of heart disease and determine if pulmonary hypertension is present)

Treatment includes: prednisone (will not help eradicate changes that are already present), azothioprine, N-acetylcysteine (mycolytic agent), bosentan (used in human medicine to treat pulmonary hypertension; is an endothelial receptor antagonist and therefore decreases pulmonary vascular resistance. However it is very expensive and can only be obtained from certain pharmacies), and pirfenidone (an antifibrotic used in human medicine; slows tumor cell proliferation by inhibiting fibroblast growth factor, epidermal growth factor, platelet-derived growth factor and transforming growth factor beta 1.)

Pulmonary thromboembolic disease should also be considered in older patients with predisposing underlying disease such as Cushing's disease, diabetes, protein losing disease, neoplasia, pulmonary hypertension, congestive cardiac disease, chronic infectious disease and so forth. Clinical signs may initiate with minor unexplained tachypnea and restlessness, persistent "allergic"-type cough especially at night, to full blown respiratory distress and rapid right heart enlargement due to ensuing pulmonary hypertension.

Therapeutic rationale controls secretions with antibiotics and steroids if justified by C&S and cytology results respectively. Nasal swabs are basically useless (identifying normal contaminant flora) while tissue scrapings providing more fruitful and just results. Respiratory diseases are often complicated by secondary bacterial infection. G+ organisms inhabit the upper airways while G- organisms reside in lower airway tissues in minimally oxygenated environments. C&S results derived from BAL samples will usually reveal heavy growth in cases of primary bacterial infection, while moderate/light growth usually means these organisms are normal flora that may be complicating the recovery. The presence of salmoniella in C&S results usually means aspiration from the nasal cavity has occurred which carries a poorer prognosis. Location of infiltrates on radiographs and level of growth should be considered regarding antibiotic selection. Fungal organisms are often missed on TT washes and more often found on BAL. Fungal serology is helpful (i.e. cryptococcus chronic nasal discharge in cats) but can provide F- results especially in cases of Histoplasmosis and Blastomycosis (regional diseases.)

Given the presence of the blood-bronchus barrier, many systemic antibiotics do not reach therapeutic concentrations necessary to be effective against the etiologic or complicating organism. For

example, only 4% of amoxicillin given parenterally reaches bronchial secretions; while gentamycin provides 25% penetration. Therefore, aerosol or **intra-tracheal** injection (diluted in 2 cc saline) therapy for 3-5 days initially followed by systemic treatment is the best approach for moderate to severe diseases of the bronchial tree. Respiragard II updraft **nebulizer** (approx \$6) is a suggested instrument for in hospital use to be attached to the O2 tank.

Cytology results may justify a tapering prednisone protocol (0.5 mg/kg PO BID tapering after abatement of symptoms). Typically 70-80% monocytes are the usual cell finding. Excessive mast cells, lymphocytes and plasma cells justify cortisone therapy. Cyclosporin can be considered in difficult cases to inhibit these infiltrates. Elevated lymphocyte presence is a concern for lymphoma. PCR testing on cytology for lymphoma can be performed at Colorado State University veterinary laboratory for approximately \$60 if lymphoma is suspected. Hypoallergenic dietary trials are justified if allergic etiology is a concern.

Nonproductive coughs can be treated with **Hydrocodone** at 0.22mg/kg q 4-6 hrs as needed. **Productive coughs** require percussion therapy and expectorants such as **Albuterol** (20ug/kg PO BID x 5d) for dogs or **Terbutaline** (Brethine) for cats (0.625 mg PO BID or 0.01 mg/kg SQ in a crisis).

Bronchodilators are indicated if crackles, elongated expiratory time and effort, tracheal collapse, flattened diaphragm, or cough are present. Non-generic **extended release theophylline** (Inwood labs 10 mg/kg PO BID) is the most effective given that doseages and release pharmacodynamics are product specific. **Ciproheptadine** can also be used in cats as a bronchodilator. Bronchodilator and steroid nebulizers can be utilized for best penetration (Ferraris Medical Infant Panda Mask 640-111 \$6; 800 205-7187).

Feline asthma: Acute crisis: 1) **Epinephrine** (1:1000) 0.1-0.2 ml iv or sc. 2) Shock dose of **steroid** (pred 2-4 mg/kg sid weaned to 1.0 – 0.5 mg/kg q 48 hrs). Management: 3) **Theophylline** extended release (above) or **Terbutalene** (Brethine) (0.625 mg PO BID or 0.15 mg/kg PO BID; 0.01 mg/kg SQ in emergency). 5) **Periactin** (serotonin inhibitor) 2 mg po bid or **Accolate** 5-10 mg po sid/bid as maintenance (needs 2-3 wks to be effective).

Chronic rhinitis cases in cats typically respond best to doxycycline (10 mg/kg SID) as a first choice against primary pathogens. Quinolones (enrofloxacin 5-20mg/kg/day), chloramphenicol (10 mg/kg BID), or azithromycin (10 mg/kg BID x 5d then q 72 hrs for 3 weeks) can be used for resistant cases. All of these medications need to be followed by a water bolus to avoid esophagitis and stricture!!!! Nasal herpes (rhinitis,conjunctivitis)or calicivirus (URI + stomatitis+/- polyarthritis) are often primary culprits with secondary pathogens. Pasteurella and mycoplasma live in low oxygen tissue under the mucous layers in the nose and mouth and often grow secondarily on viral induced lesions. Clindamycin (10 mg/kg PO SID), Clavamox (62.5mg/cat SID), and Metronidazole (15 mg/kg PO BID; also an anti-inflammatory) are prudent choices. Resistant viral infections can be treated with Lysine (250 mg PO BID standard cat; 125 mg/small cat), alpha interferon (30 IU PO SID part in nostril diluted in saline, remainder oral), or acyclovir 25 mg/kg PO BID) can be attempted as off label modulators. Last-ditch efforts with Piroxicam (0.3 mg/kg PO SID liquid formulation) can be attempted as an immune modulator with kidney

The preceding was a summary of information obtained from ACVIM 2002-2011, Western States Veterinary Conference, VIN, as well as other sources.

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