When and How to Image Dyspneic Cats

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The adage "cats are not small dogs" is especially pertinent when dealing with the dyspneic cat. This is a creature who is teetering on the edge of Darwin's "survival of the fittest" cliff. Decisions regarding treatment and handling need to be made judiciously and quickly. The car ride (in a cat carrier) alone is enough to stress even a healthy cat.

Clinical considerations

The most common reasons for cats to present in respiratory distress are congestive heart failure, asthmatic crisis, pleural effusion/pneumothorax and neoplasia. Effusions can include transudates and modified transudates, pus and blood. Chyle is the most common modified transudate. In the cat, chylous effusion is due to heart failure, neoplasia or idiopathic. Cats in left sided heart failure can present with pulmonary edema, pleural effusion or a combination.

History is very helpful when it comes to determining the cause of the respiratory distress. Cats do not cough with heart disease; they cough with asthmatic or inflammatory airway disease. Owners will often describe seeing the cat "trying to cough up a fur ball" more frequently before presentation. Asthmatic cats can have flare ups with the changes in season (furnace being turned on) or construction in the house (dry wall dust). Cats in heart failure often have no warning signs and may present in failure with an aortic thromboembolism or saddle thrombus. Dyssynchronous or paradoxical breathing can be seen with any form of respiratory distress in cats whereas it usually points to pleural space disease in dogs.

Cats in respiratory distress may not have the same clinical signs as the dog will. Many present in a sphinx position with their sternum held off the cage bottom to allow for maximal chest excursion. Abdominal effort is common and flaring of the nostrils is a subtle sign of trying to "open up their airway". Non-cardiac causes of pleural effusion are usually due to a chronic condition and the cat will adapt and show a minimum of signs until very late in the disease process.

Empirical therapy

Most feline respiratory distress patients need treatment before imaging can be performed. Do not try to take radiographs on a dyspneic cat; they may die on you if you do. In the case of pleural space disease, evacuation of fluid or air is vital to stabilization. Do not rely on pharmacotherapy (i.e. furosemide) to remove the fluid; it must be removed manually. Thoracocentesis is relatively simple and does not require a tremendous amount of supplies. If sedation is needed to perform this procedure, butorphanol (0.2-0.4mg/kg) works well.

Trying to distinguish heart failure from asthma can be difficult in the cat. Some clues may help to differentiate the two. Pulmonary crackles can be heard with both conditions but more commonly with heart failure. With asthma there is typically an end-inspiratory wheeze and expiratory push to the respiratory pattern. Body temperature is usually lower (<99°F) in heart failure whereas it is normal to elevated with asthma. Coughing up pink, frothy fluid is heart failure until proven otherwise.

Because we wish to do no harm and sometimes the answer may not that easy to determine initially, we often need to treat empirically. Try and use medications that will cause the least amount of harm if your suspected diagnosis is wrong. One reasonable dose of furosemide (~2mg/kg) is not likely to harm the asthmatic. Parenteral furosemide administration can initially cause bronchodilation and pulmonary vasodilation giving some relief before the diuretic effect kicks in. Recall that a patient in heart failure has poor perfusion so intravenous administration is preferred but may not be possible in the most fragile patients. The next best option would be intramuscular over subcutaneous injection. If the patient is suspected to be asthmatic, bronchodilation will lead to immediate relief. This can usually be accomplished with the use of an albuterol inhaler with a specially designed face mask to deliver the "puffs". Albuterol, a β adrenergic agonist, has mostly β2 effects at the typical lower dose. There is the danger of β1 effects that could be deleterious for patients with heart disease but again, one dose will not likely be that harmful if the diagnosis is incorrect. Corticosteroids are the long term treatment for asthma and should not be used until the diagnosis has been confirmed radiographically. Although poorly supported in the literature, there are anecdotal reports of corticosteroids being harmful in cats with heart disease. Theories claim that it has an aldosterone, fluid-retaining effect or that they lead to hyperglycemia and increases in intravascular volume.

Although daunting, sometimes a decision must be made to take a cat's airway, ideally before the patient arrests on its own. Having a crash cart with a variety of endotracheal tubes and laryngoscopes nearby is important. Quick induction drugs are ideal in this situation; although propofol can have serious cardiovascular depressive effects it is fast acting but short lived. Etomidate is ideal but not very practical for the general practice setting. Ketamine/valium is also another alternative although ketamine may increase myocardial oxygen demands and may not be ideal in the cardiac patient. For cats in fulminant heart failure who are coughing up the pink, frothy fluid rapid intubation and emptying the larger airways is very helpful. I find that holding the cat up by its rear end and "tea potting" it empties the airways much faster than a suction unit can. Be very careful in protecting the airway when doing this

procedure and make sure the endotracheal tube does not twist around in the cat's trachea. Know that by choosing this option you will need to be committed to ventilating for this animal until they can do so on their own.

Once a patient is stable enough, radiographs or imaging can be performed. If the patient is not stabilizing with empirical therapy, I start to worry about the "untreatable" conditions such as neoplasia or fungal disease.

Summary

Often our most delicate patients, dyspneic cats demand the utmost efficiency with the minimal stress during imaging. While most radiologists would appreciate 2 or 3 view imaging, the practical clinician will attempt to minimize the stress inherent in radiography by obtaining a single view. This session will discuss radiographic views, differential diagnoses and "clinical pearls" of radiology, and advanced thoracic imaging of dyspneic cats.

Principles of localization

Reading radiographs accurately requires a method. One method is to quickly review the entire image looking for a recognizable lesion. This "Aunt Minnie" technique serves us so well in so many cases, we are apt to err in using this method to the exclusion of a more complete method. Any method that provides a complete evaluation of all structures is OK with me. I may start with an Aunt Minnie approach but invariably complete the interpretation with a systematic approach.

Emergency radiography

Don't kill the cat! Make it quick and as stress free as absolutely possible. Along these lines make sure to measure the cat in its cage. Place the cassette on the tabletop (if you are still analog!), set the technique on you machine and put on your lead all before removing the stressed patient from their little temporary home. Limit your views to those that the cat will temporarily allow, starting with a lateral, up to a lateral of the neck region as clinically indicated. A four-view series is often necessary to sort through all potential underlying regions.

Where is the problem?

The most important interpretation may be to isolate the primary lesion site. Is the disease pleural? Pneumothorax and pleural free fluid must be diagnosed early and confidently to speed definitive therapy. The next most important decision tree branch is; heart or lung? This can often be quite difficult. Concurrent lesions may prevent complete assessment of the cardiac silhouette and therefore our ability to assess cardiomegaly. Often I would perform an quick echocardiogram rather than perform an elaborate radiographic series or consider post-therapy radiographs just for speed of reaching the final diagnosis. Similarly, primary mediastinal diseases warrant an ultrasound examination early in the process for concurrent fine needle aspiration of the causative mass lesion.

Lung lesions

Let's say that the cat has no pleural or mediastinal disease and obvious increased lung opacity. This is where we use the pattern approach for lung lesion characterization. The twist is that the rules differ from basic dog rules. Most important is the rule of cardiogenic pulmonary edema being predominantly perihilar. In cats it may be perihilar or just as likely ventral, multifocal or solitary. In other words, it is a difficult diagnosis to confidently rule-out until echocardiography is supportive. However concurrent clinical signs, such as a murmur, hypothermia, thrombosis, and radiographic cardiomegaly (next section) are very supportive.

Pneumonia looks like pneumonia, except when we consider atypical locally prevalent causes, such as mycoplasmosis, toxoplasmosis, histoplasmosis, and blastomycosis amongst others. Primary lung neoplasia may be a large solitary mass, a la dog, but more commonly appears as a nonconsolidating alveolar pattern. These lesions can be focal or multifocal, unilateral or bilateral, and can overlap quite readily for the patterns seen with cardiogenic edema an atypical pneumonia.

Finally, how can a discussion of the dyspneic cat be complete without a review of asthma? The two classic manifestations are the hyperlucent, hyperinflated appearance associated with the acute phase and the bronchial pattern seen in the more chronic phase. However, a normal appearing thorax is still a viable manifestation of a severely asthmatic cat. In fact asthma (or the latest "in vogue" term/acronym) is the primary rule-out for a severely dyspneic cat with a normal-appearing thorax. As a consideration, remember that infections, such as mycoplasmosis, can have a substantial immune component to the chronic bronchitis. Mineralization is seen with primary lung neoplasia and atypical pneumonia, but not with edema.

Cardiomegaly

Cat hearts are much more difficult to interpret than dogs. The rules of "% of the width of the chest" or "# of intercostal spaces" is extremely dependent on body condition, phase of respiration, ability to deeply inspire and concurrent medical conditions. With obesity so very common in our feline patients, a heart filling the chest may be more a manifestation of obesity than cardiomegaly. Two tools seem relevant after we discount other more variable criteria; 1) shape of the caudal heart base on the lateral projection and, 2) vertebral heart scale on the VD view. On both projections the normal cat heart is "almond" shaped. With left atrial enlargement, the

caudal heart base becomes concave, instead of convex. This appearance is likened to the normal curved contour of the kidney (reniform!?). On the VD view the normal heart is less than 4 vertebrae wide. While this is not very sensitive, it is highly specific. Cats deposit fat adjacent to the heart, which widens the heart on the VD view and, while of a disparate physical density, often causes border effacement with the heart.

Conclusions

Be gentle, quick and efficient. Where is the lesion? How big is the heart? Primary lung or secondary to heart? Don't forget asthma.

References

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