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Current State of Knowledge

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Treatment of heart disease in cats is very confusing, particularly as so few feline clinical trials have been reported. Most cats hate being medicated (and most owners hate medicating them), so shrewd drug choice is essential. Importantly, some cats may do well without treatment. An empirical approach is often adopted. Although there are a number of different forms of feline myocardial disease, therapeutic strategies depend more on clinical signs and stage of disease than by aetiology. A classification proposed by the American Heart Association can be adapted for use with feline patients.

A. AT RISK CATS

Although cats without heart disease do not require treatment, subclinical heart disease is often not detected without active screening. Maine coons and Ragdolls are at increased genetic risk of hypertrophic cardiomyopathy (HCM), and myosin binding protein C (MBPC) mutation testing is available. Other breeds may also be at risk, such as Persians, Cornish rexes, Sphinxes, Norwegian Forest Cats etc. Breeders may wish to screen for HCM in these breeds, and echocardiography remains the gold standard for providing a definitive diagnosis. Echocardiography is highly operator-dependent, and ruling out mild HCM can be difficult even for experienced sonographers. Biomarker testing (such as with NT-proBNP) has not been evaluated as a screening test in cats, but may identify cats with subclinical disease.

B. ASYMPTOMATIC CATS (HCM)

There are no studies reporting the effects of drug therapy in cats with preclinical heart disease, and treatment in this population is controversial. As HCM is believed to be very common, and prognosis is good in many cats, treatment is not mandatory for all cases. Three categories of asymptomatic cat may require treatment:

1. Outflow tract obstruction
   It is not known if cats with dynamic left ventricular outflow tract obstruction (DLVOTO) are at increased risk of adverse outcome, as is the case in human HCM. A retrospective study suggested that such cats may even have a better outcome, although some owners report ‘improvement’ following therapy to suppress DLVOTO. More recent studies suggest that DLVOTO could be a risk factor for sudden death in cats. Beta-blockers are the most effective choice for relieving obstruction, and atenolol (2-12.5mg q12h PER CAT) is most commonly used. Diltiazem is unlikely to be effective at relieving DLVOTO.

2. Left atrial enlargement
   Prognosis is worse in cats with left atrial (LA) enlargement, so treatment may be justified in these cats (if we assume that drug intervention may alter outcome). No studies have been reported in this patient population. ACE inhibitors have been proposed, and appear to be well-tolerated. There is no good evidence of benefit with diltiazem in asymptomatic cats with LA enlargement. Some cats may be asymptomatic despite severe changes, and may be at risk of aortic thromboembolism as well as congestive heart failure (CHF).

3. Congenital heart disease
   As with dogs, PDAs should be closed. In contrast with dogs, ventricular septal defects (VSDs) are the most common defect. The prognosis for VSDs is good, and intervention of any kind is rarely needed.

C. PAST OR PRESENT HEART FAILURE

Management in cats depends more on the stage of heart failure than the underlying condition.

The acutely-decompensated cat

Such cats are fragile, and do not tolerate handling very well. Ideally, one will suspect CHF from the physical exam, but ultrasound is less stressful than radiographs for identifying pleural effusion. Tachypnea and crackles with a murmur is strongly suggestive of pulmonary oedema, particularly if accompanied by a gallop. Identification of left atrial enlargement can support aggressive management of congestive failure.

Improve oxygenation

Administer O2: cats are small enough for oxygen cages to be practical.
Sedation: It may be even more important to sedate cats than dogs, as dyspnoeic cats often become very distressed (butorphanol (0.25mg/kg IM) works well)

IV furosemide to effect: initial dose should be lower than with dogs (2 mg/kg) with subsequent doses of 1-2mg/kg every 60 mins until respiratory rate decreases.

Thoracocentesis: Significant pleural effusions are more common than in dogs with congestive heart failure, and these should be drained while causing minimal stress (generally with a butterfly cannula).

Increase cardiac output?

This is difficult; generally in normotensive cats the emphasis is on treating congestive signs. Intravenous fluid therapy should be avoided, as it is unlikely to increase cardiac output, and will worsen CHF.

MILD-MODERATE CHF

Eliminate abnormal fluid retention

Furosemide: (1-4 mg/kg q12-24h PO) A sufficient dose should be given to eliminate pulmonary oedema, or until unacceptable azotaemia develops. The dose should be decreased once congestive signs have cleared. A respiratory rate of <40 breaths/min often indicates acceptable control.

ACE inhibitor: ACE inhibitors are well-tolerated in normotensive cats, with a target dose for benazepril of 0.5 mg/kg q24h

Optimize haemodynamic function

Despite conventional wisdom, it is very difficult to improve diastolic function.

Decrease heart rate?

In the past, diastolic heart failure has been treated with drugs to slow heart rate. Since the preliminary results of the multicentre study by Fox et al., the wisdom of this approach has been called into question for cats that have a history of congestive heart failure.7

The choice has generally been between a beta-blocker such as atenolol, and a calcium channel antagonist such as diltiazem. Diltiazem had fewer adverse effects on outcome than atenolol in the study by Fox et al., and there are reports of diltiazem improving relaxation in acute studies in human HCM patients. However, it is likely that the effects on relaxation are indirect.

D. REFRACTORY HEART FAILURE

In cats where CHF persists despite high oral doses of furosemide plus an ACE inhibitor, a few additional options are available.

Spironolactone: not licensed in cats - dosing is similar to dogs (1mg/kg q24h) Facial lesions have been reported.

Thiazides: hydrochlorothiazide can be cautiously added to furosemide & spironolactone, but renal function and electrolytes must be monitored very closely.

Pimobendan: not licensed in cats, but may be used in refractory CHF with systolic dysfunction. It should NOT be used if DLVOTO is present.

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SCIVAC International Congress 2012 - Rimini, Italy
230