Feline Cardiomyopathies: Treatment modalities

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INTRODUCTION

Cardiomyopathy, or heart muscle disease, describes a heterogeneous group of conditions that affects the heart muscle functionally and/or structurally. The classification, aetiology and diagnostic approach to the conditions in cats have been described in two previous articles (UK Vet Vol 9 Nos 4 and 5).

TREATMENT

A good understanding of the underlying pathophysiology is required to enable a sensible approach to the treatment and management of heart disease and/or failure. It is crucial to exclude the presence of an underlying pathology (e.g. thyrotoxicosis, hypertension, taurine deficiency) because treatment of the underlying cause can often reverse the cardiac changes.

Knowing that every feline patient might react differently to different drugs, the results of repeated clinical examinations, blood pressure measurement and thoracic radiography (Figs. 1 and 2) should be used as a guideline for drug dosage adaptation.

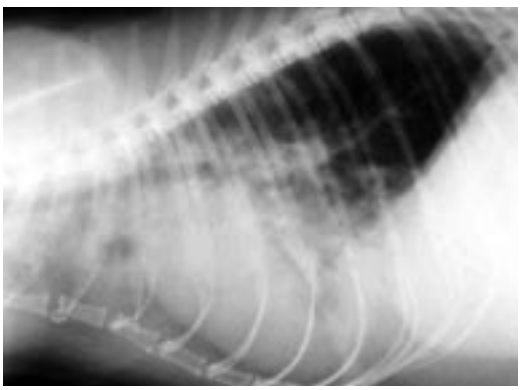

Fig. 1: *Thoracic radiograph of a cat with decompensated HCM pre treatment.*

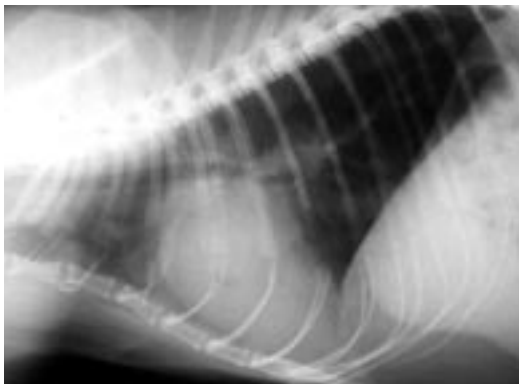

Fig. 2: *Thoracic radiograph of a cat with decompensated HCM post treatment (disappearance of pulmonary oedema).*

ASYMPTOMATIC CATS

It is a commonly asked question whether completely asymptomatic cats with **hypertrophic/restrictive cardiomyopathy** should be treated. This question remains, because of the lack of controlled clinical studies, unanswered. Very little is known about survival in these animals with many following individual courses. Unfortunately the long-term outcome is nearly always fatal.

Most cardiologists will make a 'to treat or not' decision on an individual basis: the degree and type of diastolic dysfunction and the heart rate are the determining factors. Beta-blockers are indicated in the presence of a **dynamic outflow tract obstruction** because they are proven to reduce the pressure gradient across the obstruction most effectively. Diltiazem, a calcium-channel blocker, is often first choice in the presence of a **restrictive pathophysiology** but it is theoretically contra-indicated in the case of an obstructive form because its peripheral vasodilatory properties might increase the pressure gradient across the obstruction.

If a **malignant familial history of sudden death** is present beta-blockers will often be advocated because of their protective anti-arrhythmic properties.

In pedigrees with a **high-risk genotype** (e.g. Maine Coon cats, British shorthair (Fig. 3) beta-blockade or calcium-channel blockade is often considered because of the experimental evidence that the pathway of high left ventricular pressure and workload leading to the phenotypic expression of left ventricular hypertrophy might be interrupted by these drugs.

Fig. 3: *British shorthair with HCM.*

Angiotensin converting enzyme inhibitors (ACEI) may blunt **neuro-endocrine activation** and prevent **deleterious cardiovascular remodelling**.

SYMPTOMATIC CATS

Acute heart failure

In cats, the most common reason for acute congestive heart failure is diastolic dysfunction (secondary to hypertrophic or restrictive cardiomyopathy). These cats do not benefit from positive inotropes or arteriodilators and therapy should be designed to relieve pulmonary congestion and improve ventricular filling. Intravenous **frusemide** (Lasix®1 mg/kg IV q 1-2 hrs until improvement) combined with topical **nitroglycerine** (Percutol® 2%; 0.5-0.75 cm q 8 hrs) are advocated in cases of pulmonary oedema. Renal function should be closely monitored because cats are very sensitive to diuretics. **Thoracocentesis** (Fig. 4) should be performed if the respiration is compromised by pleural effusion. An **oxygen** cage (Fig. 5) is preferred over an intranasal catheter because excessive handling and stress can tilt the balance in the already severely compromised feline.

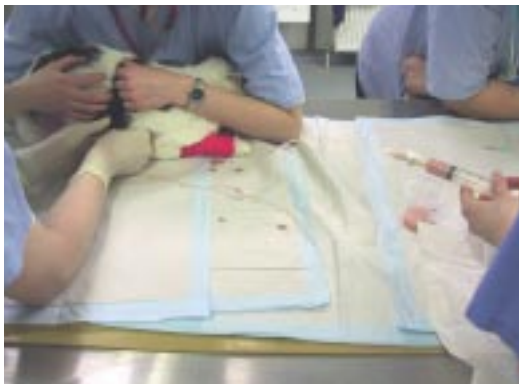

Fig. 4: *Thoracocentesis to alleviate pleural effusion in a cat.*

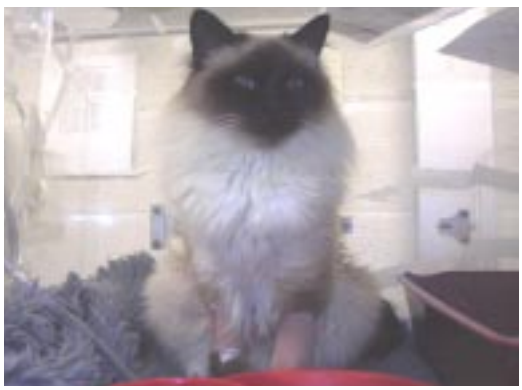

Fig. 5: *Oxygen supplementation in a cat in congestive heart failure.*

TABLE 1.

Drug	Preparation	Dose
Benazepril	5, 20 mg Fortekor® (Novartis)	0.25-0.5 mg/kg PO q 24 hrs
Enalapril	1, 2.5, 5, 10, 20 mg Enacard® (Merial)	0.5 mg/kg PO q 12-24 hrs
Ramipril	1.25, 2.5, 5 mg Vasotop® (Intervet)	0.125 mg/ kg PO q 24 hrs
Imidapril	150, 300 mg Prilium® (Vetoquinol)	0.5 mg/kg PO q 24 hrs
Propranolol β_1, β_2	10, 40 mg Inderal®	2.5-5.0 mg/cat PO q 8 hrs
Atenolol β_1	25,50, 100 mg Tenormin®	6.25-12.5 mg/kg/cat PO q 24 hrs
Diltiazem Ca-channel blocker	10 mg Hypercard® (Arnolds)	10 mg/ cat PO q 8 hrs

Cats in acute congestive heart failure because of systolic dysfunction should be treated in the same way except for the addition of positive inotropic support (dobutamine 2-10 μg/kg/min in continuous rate infusion) and taurine (250-500 mg/cat PO q 8-12 hrs) in dilated cardiomyopathy.

Maintenance therapy for diastolic dysfunction

In the absence of prospective randomised clinical trials in cats, decisions related to drug efficacy have been extrapolated from comparable human data and clinically derived observations associated with the theoretical knowledge of the pathophysiology.

Diuretics are essential in the control of congestive heart failure. **Frusemide** (1-2 mg/kg q 12 hrs) is the most commonly used diuretic in small animal medicine. It is potent and has few side effects. Care should be taken in cats because of their sensitivity to diuretics. Once the clinical signs of congestive heart failure are controlled, attempts should be made to reduce the diuretic dose. If very high dosages of frusemide (more than cumulative dose of 5 mg/kg/day) are required to control the heart failure, another diuretic should be added to the existing treatment. The author prefers to use the potassium sparing diuretic **spironolactone** (2-4 mg/kg PO q 24 hrs). Hyperkalaemia may result if ACE inhibitors or potassium supplements are concurrently administered and therefore serum potassium levels should be repetitively monitored.

Beta-blocking agents (**propranolol, atenolol**) and calcium channel blockers (**diltiazem**) have been used, because of their negative chronotrope effects, advocated to improve left ventricular filling and cardiac performance in cats with HCM. Beta-blockers are generally more effective than calcium-channel blockers in reducing heart rate. Calcium-channel blockers also have arteriodilatory (including coronary arteries) action. They reduce afterload and increase the pressure gradient in the left ventricular outflow tract when hypertrophic obstructive cardiomyopathy is present and are therefore theoretically contra-indicated. Because calcium-channel blockers have positive lusitrope properties they might be beneficial in non-obstructive hypertrophic cardiomyopathy and in early restrictive cardiomyopathy. Beta-blockers are contra-indicated when concurrent asthma is present because they cause bronchoconstriction.

Angiotensin converting enzyme inhibitors (ACEI) have a number of theoretical beneficial effects (vasodilatory, anti-aldosterone effect, attenuation of sympathetic drive, renoprotective, prevention of cardiac remodelling and fibrosis). The activation of the renin-angiotensin-aldosterone system in cats with HCM is a well-established fact. Currently only **benazepril** is licensed for use in cats (for chronic renal failure) but most other ACEI have been used safely in this species.

However, only very recently (ACVIM congress 2003) the preliminary results of a double-blinded, multi-centre evaluation of chronic therapies (frusemide plus placebo, frusemide plus atelonol, frusemide plus diltiazem, frusemide plus enalapril) for feline diastolic failure was presented by Dr Fox. Although only preliminary (130 cats divided into 4 groups), some very interesting facts have come forward. There was no significant difference between **frusemide** (1.1-2.5 mg/kg/24 hrs) alone and any group except the **frusemide and atenolol** group, in which there was a significantly higher rate of recurrence of congestive heart failure. These cats were decompensating and dying earlier. There was no statistical difference between the frusemide and the **frusemide plus diltiazem** group. The same was found for the **frusemide and enalapril** group but visualising the data suggested that there might be a small benefit in this group. This is the best veterinary clinical trial done on cats in congestive heart failure to date and in this era of evidence-based medicine we are awaiting the final results of this study to make more appropriate recommendations.

Anticoagulation

If there is a predisposition for a thrombo-embolic event (enlarged left atrium with blood stasis) anti-coagulation is indicated. The most commonly used form is aspirin. Aspirin is used for its anti-platelet aggregating properties. The classical advocated aspirin dosage of 75 mg/cat/72 hrs has recently been replaced by a similarly effective, but less harmful (gastro-intestinal side-effects) 5 mg/cat/72 hrs dosage. This low posology apparently spares the beneficial vascular endothelial prostacyclin function which has a vasodilatory role. Unfortunately the value of aspirin in preventing the formation of cardiogenic emboli is unknown as is its value in preventing thrombus recurrence (Fig. 6).

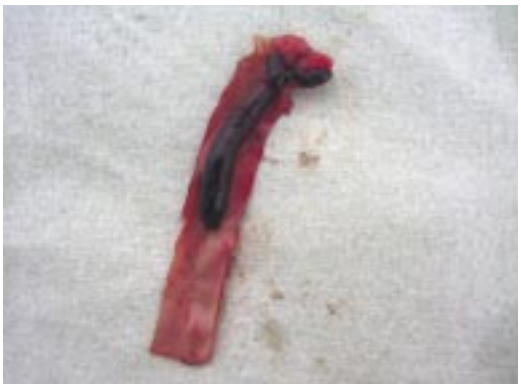

Fig. 6: *An aortic thrombo-embolism in a cat with restrictive cardiomyopathy.*

The more aggressive and effective preparations like **warfarin** have an increased risk of fatal internal haemorrhage and should only be used in indoor cats under close monitoring.

Maintenance therapy for systolic dysfunction

This extremely rare form of cardiomyopathy can be treated in a similar way to systolic dysfunction in the dog (ACEI, diuretics as above). **Taurine** supplementation (250-500 mg/cat PO q 8-12 hrs) is strongly advocated whilst waiting for the results of serum taurine analysis. In the case of taurine deficiency the cardiomyopathic changes are nearly always completely reversible and over a six-week period the cat can be weaned off cardiac drugs. Digoxin can be useful as adjunctive inotropic support but the half-life and toxicity of digoxin is much higher in cats than in dogs. It should therefore only be used by experienced individuals. There are currently no clinical data about the use of pimobendan in DCM in cats.

Anti-arrhythmic treatment

Beta-blockers appear to be the most commonly used drugs for control of supraventricular and ventricular tachyarrhythmias in cats. Digoxin (0.03 mg/4.5 kg cat q 48 hrs PO) or diltiazem are mainly administered for refractory atrial tachyarrhythmias and heart rate control in atrial fibrillation.

CONCLUSION

Despite some of the dilemmas about the most appropriate treatment for cats with cardiomyopathies coming closer to resolution many questions remain unanswered. It is important to monitor the feline patient regularly knowing that each patient may react differently to different treatments. Where necessary, the treatment should be adapted appropriately depending on the clinical findings. There is no doubt that repeated monitoring by experienced individuals improves quality and duration of life.

FURTHER READING AND REFERENCES

KITTLESON and KIENLE (1998) Small Animal Cardiovascular Medicine (Mosby).

FOX, SISSON, MOISE (2000) Textbook of canine and feline cardiomyopathy (W. B. Saunders).

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These multiple choice questions are based on the above text. Readers are invited to answer the questions as part of the RCVS CPD remote learning program. Answers appear on the inside back cover. In the editorial panel's view, the percentage scored, should reflect the appropriate proportion of the total time spent reading the article, which can then be recorded on the RCVS CPD recording form.

1. **Which is the best treatment of an asymptomatic cat with hypertrophic cardiomyopathy:**
   a. Beta-blockers
   b. Diltiazem, a calcium-channel blocker
   c. This question remains unanswered, because of the lack of controlled clinical studies,
   d. ACEI
   e. Frusemide

2. **Which is the best treatment for an asymptomatic cat with restrictive cardiomyopathy:**
   a. Diltiazem, a calcium-channel blocker
   b. ACEI
   c. This question remains unanswered, because of the lack of controlled clinical studies
   d. Beta-blockers
   e. Frusemide

3. **Which statement is false regarding anticoagulation in HCM/RCM:**
   a. If there is a predisposition for a thrombo-embolic event (enlarged left atrium with blood stasis) anti-coagulation is indicated.
   b. Aspirin has been proven to be very valuable in preventing thrombus formation.
   c. The value of aspirin in preventing thrombus recurrence remains unknown.
   d. The more aggressive and effective preparations like warfarin have an increased risk of fatal internal haemorrhage.
   e. Warfarin should only be used in indoor cats under close monitoring.

4. **Which of the following treatment modalities is contra-indicated in acute congestive heart failure in cats:**
   a. Frusemide intravenously
   b. Nitroglycerine percutaneously
   c. Beta-blockers, unless warranted for rhythm control
   d. Oxygen cage
   e. Thoracocentesis

5. **Which of the following treatment modalities has been shown to be detrimental in the only clinical trial in cats with chronic congestive heart failure secondary to diastolic dysfunction in cats:**
   a. Frusemide.
   b. Spironolactone.
   c. Atenolol.
   d. Diltiazem.
   e. ACEI.