Feline cardiomyopathy

Luca Ferasin

Cardiomyopathy is the most common form of heart disease observed in cats and patients present with a wide spectrum of structural and functional cardiac abnormalities. Although several attempts have been made to standardise the classification of the various forms of cardiomyopathy, substantial disagreement still exists among cardiologists since classification criteria are often subjective and are continuously evolving as the aetiology of myocardial disease becomes better understood. This article describes the current classification of cardiomyopathies, as well as the pathophysiology, clinical findings and treatment of the disease in feline patients.

Traditional classification

According to the World Health Organization, cardiomyopathy is defined as a 'disease of myocardium associated with cardiac dysfunction' (Richardson and others 1996). Currently, its classification is primarily based on echocardiographic examination (Table 1), although there is a substantial phenotypic variability within the same form of cardiomyopathy (Fig 1) and this often causes subjective interpretations of echocardiographic diagnosis, especially by inexperienced ultrasonographers. Pathological investigation is an alternative approach, but this is less relevant to practical clinical considerations.

Hypertrophic cardiomyopathy

Hypertrophic cardiomyopathy (HCM) represents the most common myocardial disease in cats. It is characterised by increased cardiac mass associated with left ventricular hypertrophy (LVH), which can affect different portions of the interventricular septum (IVS) and/or left ventricular free wall (LVFW). These lesions can be accompanied by left atrial dilation, aneurismal thinning of the left ventricle (LV) apex, right ventricular hypertrophy and right atrial enlargement.

Hypertrophic obstructive cardiomyopathy is a form of HCM that is associated with dynamic outflow obstruction (see systolic anterior motion, p 209).

Restrictive cardiomyopathy

Restrictive cardiomyopathy (RCM) is characterised by myocardial stiffness and diastolic dysfunction (restrictive pathophysiology), and is the second most common form of cardiomyopathy in cats (approximately 20 per cent of referred feline cardiomyopathy cases [Ferasin and others 2003]). The spectrum of phenotypes in RCM is even wider than that observed in HCM. In the human literature, RCM has both myocardial and endomyocardial forms (Hare 2008), and this classification has also been used to describe RCM in cats (Fox 2004). The myocardial form of feline RCM is characterised by restrictive filling, a normal or mildly thickened LVFW or IVS, apparently preserved systolic function and severe atrial (often bi-atrial) enlarge-

Table 1: Criteria used for the traditional classification of feline cardiomyopathy

<table>
<thead>
<tr>
<th>Hypertrophic cardiomyopathy</th>
<th>Restrictive cardiomyopathy</th>
<th>Dilated cardiomyopathy</th>
<th>Arrhythmogenic right ventricular cardiomyopathy</th>
<th>Unclassified cardiomyopathy</th>
</tr>
</thead>
<tbody>
<tr>
<td>LVH ± RVH. It can present in different forms: concentric, segmental or asymmetric. The left atrium is often enlarged. Dynamic outflow obstruction (ie, systolic anterior motion) may be present</td>
<td>Normal or near-normal LV thickness. Restrictive physiology with apparently normal systolic function. Severe left atrial (or bi-atrial) enlargement is common</td>
<td>Dilated and hypococontractile LV ± RV chambers. Atrial (or bi-atrial) enlargement</td>
<td>Dilated and hypococontractile RV with little involvement of the LV. Right atrial enlargement</td>
<td>Myocardial diseases that do not readily fit into any other group</td>
</tr>
</tbody>
</table>

Modified from Ferasin (2009a)

LVH Left ventricular hypertrophy, RVH Right ventricular hypertrophy, LV Left ventricle, RV Right ventricle

Luca Ferasin graduated from the University of Bologna, Italy, in 1992, and gained a PhD for research in the field of endocrinology from the BBSRC in Cambridge in 1996. Following three years as assistant professor at the University of Padova, Italy, he moved to the University of Bristol and taught feline and canine cardiorespiratory medicine. In 2006 he was appointed associate professor in veterinary cardiology at the University of Minnesota, USA. He returned to the UK in 2008 and is currently a visiting cardiology consultant in southern England and northern Italy. He holds the RCVS certificate in cardiology, the certificate in teaching and learning in higher education, and the European College of Veterinary Internal Medicine diploma in cardiology. He is an RCVS recognised specialist in veterinary cardiology.
ment. The endomyocardial form of feline RCM differs from the myocardial form due to the presence of extensive reparative fibrotic lesions, which affect primarily the LV and can present as large scars bridging the ventricular lumen.

Dilated cardiomyopathy
A severely dilated LV chamber and hypococontractile myocardium represent the main features of dilated cardiomyopathy (DCM). This used to be one of the most common forms of feline cardiac disease until Pion and others (1987) reported the association between taurine deficiency and DCM. This cardiomyopathy is reversible if oral taurine supplementation is promptly instituted. Based on this discovery, food companies increased taurine concentration in commercial feline diets with a dramatic reduction of the risk of taurine deficiency in the following years. Nevertheless, some rare cases of DCM that occur secondarily to taurine deficiency are still observed, although they are normally related to a non-conventional diet (ie, vegetarian/vegan diets or canine diets). Conversely, some cases of DCM diagnosed in non-taurine-deficient cats may be a manifestation of an end-stage form of another cardiac disease such as HCM, atrioventricular (AV) valvular dysplasia, ischaemic myocardial disease,
sustained rapid tachycardia (tachycardiomyopathy) or unrecognised episodes of toxicity or viral infection.

**Arrhythmogenic right ventricular cardiomyopathy**
Arrhythmogenic right ventricular cardiomyopathy (ARVC) is characterised by markedly enlarged right chambers with a thin and hypokinetic myocardial free wall, while the left chambers are minimally involved. ARVC is one of the major causes of sudden cardiac death in young people due to the presence of malignant arrhythmias (Hare 2008). Similarly, cats with ARVC may present with a variety of arrhythmias and conduction disturbances (Harvey and others 2005).

**Unclassified cardiomyopathy**
A significant number of feline myocardial diseases show features that are not typical of any other commonly recognised cardiomyopathy and are therefore described as ‘unclassified’, although they are likely to be an evolutionary phase of another recognised form of cardiomyopathy, (eg, an early or end-stage HCM or myocardial infarction) rather than an individual pathological entity (Cesta and others 2005, Ferasin 2009a) (Box 1).

**Box 1: Phenotypic classification: does it really matter?**
The classic differentiation of cardiomyopathy as hypertrophic, restrictive or dilated mixes structural changes (eg, hypertrophy and dilation) with functional abnormalities (eg, a restrictive pattern of ventricular filling). Consequently, confusion may arise because the same disease could appear in two categories, forcing many clinicians to consider an additional category called ‘unclassified cardiomyopathy’. Although some lesions may appear almost identical on echocardiographic examination, they may originate from different aetiologies (eg, valvular, ischaemic or inflammatory diseases). This is because the structural remodelling and compensatory mechanisms may be similar, even if they are initiated by different pathologies. Moreover, rhythmic disturbances and enhanced arrhythmogenicity also participate in heart remodelling (tachycardiomyopathy) and should be considered in the current classification of cardiomyopathy. During the natural course of the disease, myocardial lesions may initially affect the left ventricle and subsequently, as a consequence of increased pulmonary arterial pressure (pulmonary hypertension), the right heart chambers can appear as the only affected regions, mimicking an arrhythmogenic right ventricular cardiomyopathy.

Echocardiographic appearance may vary during the progression of myocardial disease. These echocardiographic images are from a 13-year old female neutered domestic shorthair cat presented initially for sudden-onset dyspnoea. All images are obtained from the right parasternal short-axis view. (a) Diffuse and substantial asymmetric hypertrophy affecting the left ventricular free wall (LVFW) and part of the interventricular septum (IVS). The right ventricle (RV) appears normal. (b) Significant dilation of the left atrium (LA) and left auricle (LAu). The echocardiographic features in (a) and (b) would be consistent with a diagnosis of hypertrophic cardiomyopathy. (c,d) The same patient when presented nine months later for follow-up. The left side of the heart seems ‘normalised’ while the RV and right atrium (RA) appear dilated. There is ‘flattening’ of the IVS secondary to RV pressure/volume overload. These findings could mimic a form of arrhythmogenic right ventricular cardiomyopathy. This case demonstrates how the same myocardial abnormality can present with different phenotypes during the natural course of the disease. RVFW Right ventricular free wall, Ao Aorta, RVOT Right ventricular outflow tract.
Myocardial hypertrophy

There is no consensus on criteria to define LVH. Wagner and others (2010) compared different definitions of LVH (Table 2) and concluded that different criteria for LVH yield different diagnoses and that there is limited agreement between M-mode and B-mode measurements. Furthermore, false positive results may be generated by M-mode measurements due to the presence of false tendons (Fig 2), which are often difficult to identify with this technique and can be easily and erroneously included in the measurement of the IVS thickness.

Aetiology and epidemiology

Different studies report different median ages of cats diagnosed with cardiomyopathy, which may be due to varying feline populations at different veterinary institutions. However, with some exceptions, cats affected by cardiomyopathy are generally mature individuals, although cardiomyopathy can be recognised in younger animals. Male cats seem to be more affected than females. Some Maine coon, ragdoll, Norwegian forest, Persian and Burmese cats may be genetically predisposed, but the majority of cardiomyopathy cases in practice are in domestic shorthair cats.

The primary aetiology of feline cardiomyopathy is not fully understood, although familial HCM has been described in different breeds (eg, Maine coon, ragdoll and British shorthair cats). A causative mutation for HCM has been identified in the sarcomeric gene for the cardiac myosin binding protein C (MYBPC3) both in Maine coons (Meurs and others 2005) and ragdolls (Meurs and others 2007), although this mutation is located in different regions of the same gene in each breed. Not all cats with this mutation go on to develop HCM and, similarly, HCM can be diagnosed in cats that appear negative on genetic testing. It is therefore possible that other mutations play a role in the phenotypical expression of HCM, and not just at a sarcomeric level.

Primary HCM needs to be differentiated from secondary LVH caused by LV pressure overload (ie, LV outflow obstruction, systemic hypertension), hypertomatropism and hyperthyroidism. However, it has been observed that hyperthyroid cats present with only a modest septal hypertrophy (Connolly and others 2005).

Other causes of myocardial hypertrophy are given in Table 3.

Pathophysiology

Several cardiac functional abnormalities are observed in cats that are affected by cardiomyopathy.

Diastolic dysfunction

Feline cardiomyopathy is inevitably accompanied by reduced ventricular relaxation, which can be secondary to myocardial hypertrophy, interstitial fibrosis,

Table 2: Different classification criteria to determine left ventricular hypertrophy on echocardiographic examination in cats

<table>
<thead>
<tr>
<th>Method</th>
<th>Definition</th>
<th>Type of echocardiographic assessment</th>
</tr>
</thead>
<tbody>
<tr>
<td>LVH5.5-MM</td>
<td>IVSd or LVFWd 5.5 mm M-mode</td>
<td>M-mode, LV in right parasternal short-axis view</td>
</tr>
<tr>
<td>LVH6.0-MM</td>
<td>IVSd or LVFWd 6.0 mm M-mode</td>
<td>M-mode, LV in right parasternal short-axis view</td>
</tr>
<tr>
<td>LVH5.5-BM</td>
<td>IVSd or LVFWd 5.5 mm B-mode</td>
<td>B-mode, LV in right parasternal short-axis view</td>
</tr>
<tr>
<td>LVH6.0-BM</td>
<td>IVSd or LVFWd 6.0 mm B-mode</td>
<td>B-mode, LV in right parasternal short-axis view</td>
</tr>
<tr>
<td>LVH5.5%</td>
<td>&gt;50 per cent of one wall segment or 25 per cent of two neighbouring wall segments 6-0 mm B-mode</td>
<td>B-mode, LV in right parasternal short- or long-axis view</td>
</tr>
</tbody>
</table>

Modified from Wagner and others (2010)

LVH Left ventricular hypertrophy, IVSd Interventricular septum in diastole, LVFWd Left ventricular free wall in diastole, LV Left ventricle

Table 3: Potential causes of myocardial lesions to consider before making a diagnosis of primary cardiomyopathy

<table>
<thead>
<tr>
<th>Potential cause of myocardial hypertrophy</th>
<th>Potential myocardial lesion observed</th>
</tr>
</thead>
<tbody>
<tr>
<td>Systemic hypertension</td>
<td>Concentric LVH</td>
</tr>
<tr>
<td>Left or right outflow obstruction</td>
<td>Concentric LVH or RVH</td>
</tr>
<tr>
<td>Acromegaly</td>
<td>Concentric LVH</td>
</tr>
<tr>
<td>Myocardial tumours (eg, lymphomas, mesotheliomas)</td>
<td>Concentric symmetric or asymmetric LVH and hypokinesis</td>
</tr>
<tr>
<td>Hyperthyroidism</td>
<td>Modest septal hypertrophy</td>
</tr>
<tr>
<td>Dystrophin-deficient hypertrophic feline muscular dystrophy</td>
<td>Concentric LVH and hypokinesic endocardium</td>
</tr>
<tr>
<td>Myocardial ischaemia</td>
<td>Depressed and hypokinetic myocardial areas, ventricular chamber dilation</td>
</tr>
<tr>
<td>Mitral/tricuspid dysplasia</td>
<td>LV/RV dilation and LA/RA enlargement</td>
</tr>
<tr>
<td>Myocarditis</td>
<td>Concentric or asymmetric LVH or RVH</td>
</tr>
</tbody>
</table>

Modified From Ferasin (2009a)

LVH Left ventricular hypertrophy, RVH Right ventricular hypertrophy, LV Left ventricle, RV Right ventricle, LA Left atrium, RA Right atrium
loss of cellular architecture or a combination of these mechanisms. An increased heart rate may also exacerbate the diastolic dysfunction by reducing the time available for ventricular filling and for coronary blood flow, which may lead to myocardial ischaemia. The diastolic dysfunction causes an increase in the LV and left atrium (LA) filling pressure, resulting in pulmonary venous hypertension and eventually pulmonary oedema and/or pleural effusion (congestive heart failure [CHF]).

**Systolic dysfunction**

Although reduced myocardial contractility is a predominant feature of DCM and ARVC, systolic dysfunction has also been demonstrated in cats with HCM by pulsed tissue Doppler imaging (TDI) techniques (Koffas and others 2006). Systolic impairment can appear in all forms of cardiomyopathy accompanied by ischaemia and replacement fibrosis, and systolic dysfunction will result in increased ventricular filling pressure and CHF.

**Arterial thromboembolism**

An altered blood flow or blood stasis within the cardiac chambers can increase the risk of red blood cell aggregation and intracavitary thrombus formation, especially at the level of an abnormally enlarged left auricle. When a thrombus or a fragment of it dislodges and moves into the systemic circulation via the aorta, it can block one of the major arteries causing limb paresis/paralysis, renal infarction or sudden death. Although not all patients with echocardiographic evidence of intracavitary thrombi develop an arterial thromboembolism (ATE), this finding usually represents a severe risk of haemodynamic complication (Fig 3).

**Arrhythmias**

Unpublished studies by the author based on 24-hour ECG Holter recordings (Fig 4) show that almost all cats affected by cardiomyopathy suffer from significant
ventricular or supraventricular arrhythmias. Furthermore, many patients with cardiomyopathy have conduction abnormalities with complete AV block being the most common finding. The haemodynamic disturbances secondary to arrhythmias contribute to the complex pathophysiology of feline cardiomyopathy.

**Myocardial ischaemia**

Regional myocardial ischaemia is commonly recognised in feline patients with all forms of cardiomyopathy and is often associated with replacement fibrosis (Fox 2003a, 2004, Cesta and others 2005). Ischaemia can be secondary to intramural coronary arterial disease caused by myocardial hypertrophy, and is frequently followed by malignant arrhythmias, as well as systolic and diastolic impairment. The significant elevation of cardiac troponin-I (cTn-I) in cats with HCM might indicate ongoing myocardial damage, possibly secondary to a concurrent myocardial infarction (Connolly and others 2003).

**Systolic anterior motion**

Systolic anterior motion (SAM) of the mitral valve is a form of dynamic LV outflow obstruction that is present in approximately half of feline HCM cases. It is characterised by an abrupt movement of the anterior leaflet of the mitral valve towards the IVS, interfering with the LV outflow in mid-systole. The abnormal position of the mitral leaflet in systole is also responsible for a simultaneous mitral regurgitation, resulting in a typical ‘double jet’ on colour Doppler echocardiography. The abnormal movement of the septal leaflet can also be observed on M-mode imaging of the mitral valve and on spectral Doppler evaluation of the LV outflow tract, with an increased aortic peak flow velocity and an abrupt acceleration in mid-systole, which produces a characteristic asymmetric waveform (Fig 5).

The dynamic obstruction caused by SAM has many consequences:
- It reduces the stroke volume, hence the cardiac output;
- It causes LV pressure overload, which may stimulate further cardiac hypertrophy;
- Mitral regurgitation may lead to atrial remodelling (LA dilation);
- The continuous mechanical contact between the septal mitral leaflet and the proximal IVS induces fibrotic lesions (‘contact lesions’) that can affect the normal function of both affected anatomical parts.

The blood flow turbulence originating during SAM is also responsible for the presence of systolic murmurs on auscultation in many cats with cardiomyopathy.

![Fig 5: Systolic anterior motion of the mitral valve (MV) in a six-year-old male neutered domestic shorthair cat.](image-url)

(a) Right parasternal long-axis B-mode echocardiography with colour Doppler interrogation of the MV area and left ventricular outflow tract (LVOT) showing the typical ‘double jet’ during the dynamic outflow obstruction. (b) Spectral Doppler interrogation of the LVOT showing increased aortic outflow velocity and the characteristic asymmetry with a mid-systolic ‘step’ (arrows). (c) Right parasternal short-axis M-mode echocardiography of the left ventricle (LV) at the level of the MV showing a mid-systolic movement of the septal mitral leaflet towards the interventricular septum (IVS) (arrows). (d) Right parasternal long-axis colour M-mode Doppler echocardiography of the LV at the level of the MV showing a mid-systolic jet during the abnormal movement of the mitral septal leaflet (arrows). LVFW Left ventricular free wall, LA Left atrium, Ao Aorta
Heart murmurs
Heart murmurs are very common in cats with cardiomyopathy (approximately 60 per cent of cases [Ferasin 2009a]) and usually originate from dynamic left ventricular outflow tract obstruction and/or mitral regurgitation (Fig 6). The murmurs are systolic, often with variable intensity, and can easily be heard over both parasternal regions. They may be present at rest or may become audible when the heart rate and cardiac contractility increase (eg, during stress or excitement). Several cases of cardiomyopathy are diagnosed in asymptomatic cats following the detection of a heart murmur. Murmurs originating from SAM can also be present in the absence of echocardiographically detectable LVH and, for this reason, may allow early identification of animals that may develop myocardial changes later in life.

Gallop sounds
Gallop sounds present another common clinical finding in cats with cardiomyopathy. They are caused by audible diastolic sounds ($3$ and/or $4$) in the presence of reduced myocardial compliance (eg, myocardial hypertrophy, infiltration, fibrosis, tachycardia or a combination of these factors).

**Muffled heart sounds**
Muffled heart sounds can be heard when a pleural and/or pericardial effusion is present.

**Tachypnoea/dyspnoea**
Tachypnoea/dyspnoea is a common clinical sign in cats with cardiomyopathy complicated by CHF (pulmonary oedema and/or pleural effusion). Laboured breathing sometimes occurs acutely after a stressful event (eg, a car journey, hospitalisation or restraint), so cats suspected of having cardiomyopathy should always be examined gently and cautiously.

**Limb paresis/paralysis**
Limb paresis/paralysis associated with ATE is often seen in cats with cardiomyopathy. Although bilateral hindlimb paresis represents the most common presentation (71 per cent of all ATE cases [Smith and others 2003]), unilateral hindlimb or forelimb involvement is also possible. Thromboembolism can also potentially cause sudden death.

**Cardiac arrhythmias**
Ventricular or supraventricular cardiac arrhythmias are frequently detected in patients with cardiomyopathy. However, some forms of paroxysmal arrhythmias may not be detected during physical examination or standard electrocardiographic recording. Arrhythmias contribute to a reduction in myocardial performance and may cause syncope and sudden death.

**Arterial hypotension**
Arterial hypotension (ie, a systolic blood pressure below 120 mmHg) has been recorded in approximately 15 per cent of cats with cardiomyopathy as a result of reduced cardiac output (Ferasin and others 2003).

**Ascites**
Ascites can occur in patients with right-sided CHF and are likely to be associated with ARVC, DCM or other forms of cardiomyopathy complicated by pulmonary hypertension.

**Diagnosis**
**Echocardiography**
Diagnosis of feline cardiomyopathy is based primarily on echocardiographic examination. Echocardiographic recognition of basic patterns is very intuitive but clinicians should be aware that incorrect conclusions may result from inexperience or intra- and interoperator variability. For example, LVH can be misdiagnosed if the M-mode cursor is incorrectly aligned or if a papillary muscle or a false tendon is included in the LV measurements. Therefore, a high frame rate B-mode examination with good image quality is often indicated for an accurate diagnosis of LVH. Clinicians should also remember that changes in preload and afterload can affect LV measurements. A patient’s dehydration status can induce a ‘pseudo- hypertrophy’, and overzealous
fluid therapy can dilate the cardiac chambers simulating a dilated form of cardiomyopathy.

The recognition and assessment of SAM is based primarily on colour flow and spectral Doppler studies, which require correct positioning, optimal settings and experienced operator skill and interpretation.

Advanced echocardiographic techniques, such as TDI and myocardial strain analysis, allow further quantification of regional myocardial function and can potentially be useful in the early detection of myocardial dysfunction in cats.

Radiography
Thoracic radiographs are indispensable for recognising CHF, which is characterised by cardiomegaly, engorged pulmonary veins and pulmonary oedema or pleural effusion with, in some cases, ascites. Pleural effusion and ascites can easily be detected by percussion and ultrasonography. The distribution of alveolar infiltrate can be diffuse or patchy, in contrast to the more consistent caudalos dorsal localisation of cardiogenic pulmonary oedema seen in dogs. Cardiomegaly is not always obvious in feline cardiomyopathy, especially when there is no significant chamber enlargement (Fig 7).

Genetic tests
Laboratory tests are available for Maine coon and ragdoll cats to identify individuals with a mutation in the MYBPC3 gene. The test can be performed on EDTA blood samples or buccal swabs. However, the mutation in the two breeds is located in a different gene locus and therefore the test is not interchangeable. A positive heterozygous or homozygous result indicates that an individual may be at risk of developing HCM but it does not signify a diagnosis of HCM – this ultimately requires echocardiographic examination. Furthermore, HCM can also occur in animals that are negative on genetic testing, suggesting that there are likely to be other mutations that have not yet been identified.

Biomarkers
cTn-I is a sensitive and specific marker of cardiac myocyte injury and an increase in its plasma concentration, which may occur in all forms of the disease, indicates ongoing myocardial damage. This assay may provide useful information on the severity of myocardial damage and prognosis.

The N-terminal pro B-type natriuretic peptide (NT-proBNP) assay is a new laboratory test that provides evidence of ongoing myocardial stress. It is highly sensitive and can detect early myocardial changes associated with cardiomyopathy. A full cardiac evaluation, including echocardiographic examination, should always be performed to identify the cause of NT-proBNP elevation and determine the proper clinical management.

Therapy
At present, with the exception of dietary taurine supplementation in cats with cardiomyopathy secondary to taurine deficiency, there are no available treatments that have convincingly demonstrated increased survival times and/or quality of life in cats with myocardial disease.

Asymptomatic cats
Anecdotal reports claim that asymptomatic cats with HCM that are treated with diltiazem or beta-blockers have improved physical activity. However, randomised placebo-controlled studies are lacking to confirm this and the clinical use of these drugs in such animals has still to be proven, especially in cases that are accompanied by SAM, which could potentially benefit from a reduction of dynamic outflow obstruction. Similarly, angiotensin-converting enzyme inhibitors and spironolactone have failed to demonstrate significant improvements in cats with subclinical forms of HCM (Macdonald and others 2004, 2006, Taillefer and Di Fruscia 2006). Cats with asymptomatic forms of cardiomyopathy and echocardiographic evidence of intracavitary thrombi may benefit from antithrombotic prophylaxis to reduce the risk of ATE but clinical evidence has not been demonstrated clearly.

Symptomatic cats
Patients presenting with acute CHF require sedation, cage rest and oxygen supplementation in addition to parenteral diuresis (ie, furosemide). Significant pleural effusion should be treated promptly by thoracocentesis. Once pulmonary oedema is clinically controlled, furosemide can be given orally at the lowest effective dose, to reduce the risk of prerenal azotaemia and hypokalaemia. The risk of hypokalaemia could be reduced by concomitant administration of potassium sparing agents, such as spironolactone, although sufficient data of its clinical efficacy in cats are not available.

Diltiazem is licensed in the UK for the management of HCM due to its bradycardic, lusitropic and coronary vasodilating properties. However, its beneficial effects have not been demonstrated clearly in controlled randomised clinical studies. Moreover, the licensed formulation should be administered three times daily and many clients struggle to comply with this schedule.

Beta-blockers have also been suggested for the treatment of feline cardiomyopathy, but the only available, and still unpublished, controlled study shows
that cats receiving enalapril survived as long as or longer than the placebo group, although the difference was not statistically significant (Fox 2003b).

Pimobendan is not licensed for use in cats, although small, uncontrolled studies seem to demonstrate an improvement in appetite and demeanour in patients with feline cardiomyopathy accompanied by systolic dysfunction. However, the pharmacokinetics of pimobendan in cats are different from those described in dogs, with a more rapid absorption and longer plasma half-life; the correct dosage and frequency of administration is unknown.

A prudent approach should therefore be taken when recommending any of these drugs in addition to furosemide, for the chronic treatment of feline cardiomyopathy.

References and further reading


Prognosis

The life expectancy of a patient depends on the form of myocardial damage and its severity, with a shorter survival time in cats with severe cardiac remodelling and clinical signs of CHF. A retrospective study conducted on a population of 127 cats with HCM shows a median survival of 194 days in symptomatic cats versus 3617 in the asymptomatic group (Payne and others 2010). In the same study, left atrial enlargement represents another negative prognostic variable. Other negative prognostic indicators include the presence of arrhythmias, gallop sounds, left atrial ‘smoke’ (spontaneous echocontrast), and reduced fractional shortening. Interestingly, the presence of SAM is associated with a longer survival time and this is possibly because it produces an audible heart murmur and allows earlier identification of the disease. Prospective longitudinal studies in progress at the time of writing and will hopefully add valuable prognostic information for cats with HCM in the near future.

Other forms of cardiomyopathy carry a less favourable prognosis, with a median survival time of 132 days for RCM and 11 days for DCM (Ferasin and others 2003). Such poor prognosis is probably associated with the fact that a dilated and hypocontractile myocardium could represent the end-stage of another form of cardiomyopathy (Ferasin and others 2003).

Summary

Feline myocardial disease remains a controversial topic in veterinary cardiology, despite recent discoveries on aetiology and pathophysiology that have helped the understanding of this important clinical condition. Although echocardiographic diagnosis remains extremely challenging, it is often oversimplified by non-experienced ultrasonographers, especially in cases that are complicated by dynamic outflow obstruction, which has important implications for the clinical management of patients. Hopefully, the completion and publication of some important longitudinal studies currently in progress will provide answers to the questions still arising in this clinical area.
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