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Next WSAVA Congress:

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Cardiomyopathy describes a functional impairment or structural abnormality of the heart muscle. It may be primary (idiopathic) where there is no identified cause or may be secondary or specific where there is an identifiable systemic, metabolic or nutritional disease. Several types of idiopathic cardiomyopathy are recognised; hypertrophic, dilated restrictive and arrhythmogenic right ventricular cardiomyopathy. A further category of unclassified cardiomyopathy is now recognised for cases that do not fit neatly into any of the above categories. Secondary cardiomyopathies are of particular importance in cats and an attempt should always be made to rule these out before categorising a case of cardiomyopathy as ‘idiopathic’. Common secondary cardiomyopathies include hypertrophy secondary to hypertension or hyperthyroidism.

Types of cardiomyopathy

Hypertrophic cardiomyopathy (HCM)

In this form of cardiomyopathy there is inappropriate hypertrophy of the left ventricle and on occasions the right ventricle may also be involved. This results in abnormal diastolic function. Contractile function is usually normal. Hypertrophy may be symmetrical, asymmetrical or localized. Most cases are classed as primary/idiopathic. The most notable secondary/specific causes in cats include hyperthyroidism and systemic hypertension. Other specific causes that have been implicated include acromegaly, obesity, muscular dystrophy and glycogen storage disorders. Familial forms of HCM have been described in several breeds of cats and a mutation of myosin binding protein C has recently been implicated in HCM in Maine Coon and Ragdoll cats. Several different genetic mutations have been identified in HCM in humans and no doubt further mutations probably also exist in cats.

Hypertrophic obstructive cardiomyopathy (HOCM)

Hypertrophy of the interventricular septum may result in narrowing of the left and/or right ventricular outflow tract. The mitral valve may also be sucked into the outflow tract (systolic anterior motion of the mitral valve - SAM) resulting in further obstruction of the outflow tract. Obstruction of the outflow tract may be dynamic or fixed.

Dilated cardiomyopathy (DCM)

The main feature of this form of cardiomyopathy is systolic failure. The cardiac chambers are dilated and there is reduced contractility. In contrast to hypertrophic cardiomyopathy the majority of cases are secondary or specific. Dietary taurine deficiency is well documented as a cause of DCM. Adriamycin has also been shown to cause myocardial failure. DCM is reported in cats with normal serum taurine levels and no history of chemotherapy. The aetiology of the DCM in these cases is unknown. The myocardial failure may represent the end stage of various possible insults to the myocardium, including viral or immune-mediated inflammation, toxins or myocardial failure secondary to infarction.

Restrictive cardiomyopathy (RCM)

This form of cardiomyopathy is characterised by varying degrees of focal or diffuse fibrosis of the myocardium, subendocardium and endocardium. These changes reduce ventricular compliance and restrict filling, thus leading to diastolic failure similar to that caused by HCM. The clinical diagnosis of RCM is made by observing clinically significant diastolic dysfunction without evidence of significant hypertrophy. There is typically severe atrial enlargement. The aetiology of RCM in cats is unknown, thus the majority of cases are classified as idiopathic/primary. Myocarditis has been implicated as a possible cause. Myocarditis is difficult to diagnose clinically and the prevalence in cats may be underestimated. Other types of infiltrative myocardial disease may also lead to diastolic dysfunction and may be referred to as secondary restrictive cardiomyopathies.

Arrhythmogenic right ventricular cardiomyopathy (ARVC)

This form of cardiomyopathy has recently been described in cats. The aetiology is unknown, however a familial form has been reported in humans. It is characterised by severe right atrial and ventricular enlargement and marked tricuspid regurgitation due to distortion of the tricuspid valve; arrhythmias are common. It is possible that cases of ARVC have previously been misdiagnosed as tricuspid valve dysplasia.

Unclassified cardiomyopathy (UCM)

In recent years an increasing number of cats have been identified that do not fit into any recognised disease classification using echocardiographic and pathological criteria. Typically these cats have severe biatrial enlargement, normal left ventricles or mild hypertrophy and normal or slightly decreased systolic function, but they do not have the typical post-mortem findings of fibrosis seen in restrictive cardiomyopathy. Many cats have enlargement of the right ventricle. It is not known if these cats represent a progressive or regressive form of...
other known cardiomyopathic states.

**Clinical presentation**
Cats with cardiomyopathy (HCM, DCM, RCM, ARVC, UCM) may present with incidental murmurs or arrhythmias noted at routine visits. Alternatively they may present with signs of weakness, collapse, respiratory distress, inappetance, ascites, exercise intolerance, intermittent forelimb lameness or hind quarter paralysis due to aortic thrombo-embolism. On clinical examination murmurs are often heard over the left or right mid-heart or over the sternum. The murmurs may be due to atrio-ventricular valve insufficiency or due to left or right outflow tract obstruction. Sinus tachycardia, arrhythmias and gallop sounds are common.

**Clinical differentiation of feline cardiomyopathies**
It can be very difficult to differentiate the various types of cardiomyopathies clinically. Often a combination of history, clinical examination, electrocardiography, thoracic radiology, echocardiography and blood tests may be needed to reach a definitive diagnosis. Echocardiography plays a key role in diagnosis and the recent increased use of Doppler tissue imaging has helped further in characterising and staging the disease. There are always going to be cases where only endomyocardial biopsy or post mortem will give us a definitive diagnosis and on occasions even gross pathological findings may still be equivocal.

**Treatment of feline cardiomyopathies**
Once a diagnosis of cardiomyopathy has been reached then it is very important to make every effort to rule out secondary causes of cardiomyopathies. Thyrotoxicosis, hypertension, taurine deficiency can all be treated resulting in reversal of cardiac changes. There is still very limited information on the natural history of cardiomyopathies in cats and few long term clinical trials assessing the affects of therapy. The treatment of asymptomatic cats with primary HCM remains controversial. In general treatment decisions are very much specific for each case and tend to be based on the presence of the following:
- **Diastolic failure**
- **Systolic failure**
- **Congestive heart failure**
- **Outflow tract obstruction**
- **Sinus tachycardia**
- **Brady or tachyarrhythmias**
- **Thromboembolic disease**

It is very important to characterise the disease as best one can. It is also important to monitor cases carefully as treatment needs may change as the cardiac disease progresses e.g. hypertrophic cases may have diastolic dysfunction early on in the disease, however poor systolic function may occur later in the course of the disease. Recent publications on Doppler tissue imaging findings would suggest that abnormal systolic function is present in many HCM cases. Brady- or tachyarrhythmias may develop during the course of the disease due to fibrosis or infarcts which may necessitate a change in therapy. Progressive left atrial enlargement needs to be monitored as it may predispose to thrombo-embolic disease.

**Efficacy of therapy**

**Diastolic failure**

Numerous clinical trials have been undertaken in the asymptomatic and symptomatic cat with idiopathic HCM to assess efficacy of medication. Clinical trials with diltiazem have been conducted which support a decrease in wall thickness and increased exercise tolerance in cats with HCM, however no trial to date has supported increased survival. No clinical trials have been published showing long term effects of beta blockers in the treatment of asymptomatic HCM in cats. One recent trial has shown adverse affects of betablockers in the presence of congestive heart failure compared to other medications such as calcium channel blockers or angiotensin converting enzyme (ACE) inhibitors. Independent trials using either enalapril or benazepril have shown a decrease in left ventricular wall thickness in cats with HCM again the significance of this finding is uncertain.

**Systolic failure**

Digoxin has been shown to improve systolic function in cats with systolic failure. Taurine has been recommended for the treatment of systolic failure in idiopathic dilated cardiomyopathy, however there is no evidence that this affects the course of the disease. Pimobendan is presently being used in the treatment of systolic failure, evidence of efficacy is anecdotal.

**Outflow tract obstruction**

Beta blockers have been shown to decrease the outflow tract gradient in cats and reduce or eliminate murmurs in cats with obstructive hypertrophic cardiomyopathy. It is unknown whether this results in increased survival. Interestingly in one study cats with the obstructive form of HCM were shown to have better survival than cats with no obstructive HCM.

**Sinus tachycardia**

Cats with heart rates of > 200 bpm at presentation have been shown to have a decreased survival time compared to cats with heart rates < 200 bpm. This finding probably reflects severity of disease. There are no studies showing that control of tachycardia in asymptomatic cats affects survival.

**Thromboembolic disease**

In the past aspirin has been the main drug used in
prevention of thromboembolism in cats at risk. In recent years an increased number of drugs have been used including low molecular weight heparin and novel anti-platelet aggregation drugs such as clopidogrel. It is hoped that these newer drugs used either alone or in combination may be superior to aspirin in preventing this devastating complication of feline cardiac disease.

**Conclusion**

There has been a great increase in our understanding of the feline cardiomyopathies over the past ten years. This has been partly due to the increased availability of ultrasound and partly due to a better understanding of the pathology. There are still many gaps in our knowledge especially regarding the natural history and the best therapeutic options. Hopefully our understanding and knowledge will continue to grow over the years to come.