A Review of Primary Cardiomyopathy in the Cat

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INTRODUCTION

Primary cardiomyopathy in cats has been recognized in the literature as a clinical entity only since 1970. Prior to this, the recognition of this disease had been in association with aortic thromboembolism, an important complication of cardiomyopathy.[1] Because the etiologies of the several forms of cardiomyopathy are unknown, classification has been based on the nature of the hemodynamic fault or the anatomical abnormality that is present.[2] The two most basic and common forms are hypertrophic cardiomyopathy and congestive cardiomyopathy.

Hypertrophic cardiomyopathy represents severe left ventricular hypertrophy which causes impaired ventricular distensibility and compliance. Left atrial dilatation follows and adds to the diastolic dysfunction which results in left-sided congestive heart failure. Congestive cardiomyopathy is characterized by poor myocardial contractility, with dilatation of the heart chambers and resultant failure of the heart as a pump.[3] A more recently described form of idiopathic cardiomyopathy is restrictive cardiomyopathy. This form is seen as a greatly diminished ventricular compliance due to a rigid, poorly distensible ventricular wall.[4]

In addition to being a very important disease in the cat, feline cardiomyopathy bears many similarities to human cardiomyopathies, and thereby provides a useful model for the study of this disease in humans.[5]

INCIDENCE

The increased incidence of idiopathic cardiomyopathy in recent years is thought to be due more to increased awareness than to increased frequency of the disease in cats. Of the two major forms of feline cardiomyopathy, the hypertrophic form is more common. The cause of hypertrophic cardiomyopathy in cats is unknown, but evidence suggests a hereditary component. The disease has been found in related cats, and in man a large number of cases have a familial incidence. Cats can be affected at any age, but hypertrophic cardiomyopathy is most common in middle-aged cats. Males are more commonly affected than females.

Cats with congestive cardiomyopathy are usually middle to older-aged. Like hypertrophic cardiomyopathy, it occurs more commonly in males than females. The third and least common form, restrictive cardiomyopathy, is also usually found in middle-aged cats.

There has been much speculation that the various forms of cardiomyopathy may represent different stages of a single disease, but this has yet to be proven.

CLINICAL DESCRIPTION

Hypertrophic Cardiomyopathy

As stated earlier, hypertrophic cardiomyopathy represents severe left ventricular hypertrophy which causes impaired ventricular distensibility and compliance. The hypertrophy of the myocardium can be either symmetric or asymmetric. In asymmetric hypertrophy the septum is thicker than the left ventricular free wall. In addtion to diminishing the volume of the left ventricle, the outflow tract may be obstructed by myocardium causing obstructive cardiomyopathy. The most common form of left ventricular hypertrophy in cats is symmetric. The septum, left ventricular free wall, and the papillary muscles are hypertrophied resulting in a diminished volume of the chamber.

As with the other forms of feline cardiomyopathy, the etiology is unknown but appears to

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involve a hereditary factor. The only breed predilection identified at this time is the Persian.[3] Some of the latest speculation raises the possibility that hypertrophic cardiomyopathy may be a disorder of developing embryonal endocrine tissue.[6] It is hypothesized that a genetic fault results in disturbance of interaction between immature myocardial adrenergic receptor sites and extra-cardiac catecholamines during cardiac embryogenesis. This results in cellular disarray and ventricular hypertrophy. Other research is being directed at possible abnormal calcium metabolism, which may contribute to the basic disease process.[4]

The most common clinical signs of hypertrophic cardiomyopathy in the cat are dyspnea and aortic thromboembolism. Other less common presenting signs include lethargy, anorexia, and sudden death. Auscultation is a valuable tool in diagnosing this disease. Many cats have a systolic murmur of mitral valvular insufficiency and a third or fourth heart sound.

Macroscopic pathology shows hypertrophy of the ventricular septum as well as the left ventricular free wall and papillary muscles in most cases. This results in a significant decrease in the size of the left ventricular cavity as well as obstructing the outflow tract in many cases.[7] Mitral valvular fibrosis/thickening and hypertrophied papillary muscles result in a mitral valvular insufficiency. Left atrial dilatation is present, often to extreme degrees. Stress and tachycardia appear to be important precipitating factors in producing the secondary effects, pulmonary edema and acute dyspnea. A tachycardia produced by the stress of exercise shortens diastolic filling time. Cardiac output falls, further limiting diastolic filling. The smaller lumen also contributes to decreased ventricular filling and the resulting change in end diastolic pressure. Cardiac output falls because the tachycardia is not sufficient to compensate for the drop in stroke volume caused by the poor filling. Pulmonary congestion and edema with dyspnea result from the left-sided heart faulure.[3] Another very important sequela, thromboembolism, occurs when blood stagnates and clots in the very dilated left atrium or in the apex of the left ventricle. After these clots break free and enter the aorta, they may lodge in several locations, the most common of which is the trifurcation of the descending aorta at the external iliac arteries. Other less common areas of thromboembolism include the front legs, the kidneys, and the brain. Aortic thromboembolism itself is a cause of stress and tachycardia because of the pain and shock produced, and therefore, further compromises cardiac function.

Microscopic pathology in hypertrophic cardiomyopathy is also very distinctive. The muscle cells are enlarged and have large hyperchromatic nuclei. Muscle bundles show bizarre disarrangement and are separated by an increased amount of interstitial connective tissue. There are often focal areas of the endocardium that are replaced by active fibroplasia. Also, the atrioventricular Purkinje fibers are frequently interrupted by or mixed with many dense collagen fibers.[7]

Electrocardiography is an important tool in diagnosing hypertrophic cardiomyopathy. However, a variety of abnormalities may be seen, and this must be taken into account when interpreting results. Prolongation and increased amplitude of both the P wave and the QRS interval in lead II are the most commonly observed abnormalities. Most cats will also show a left axis deviation, although normal axis and right axis deviations have been recorded in affected cats. Several types of atrioventricular conduction disturbances are commonly seen, as well as a left anterior fascicular branch block. Rhythm disturbances include premature ventricular contractions, atrial fibrillation, atrial tachycardia, and ventricular tachycardia.

Radiology can also be very helpful in diagnosing hypertrophic cardiomyopathy. Gross cardiomegaly is present in most cases. In the lateral view, the heart remains in its normal oblique position within the thorax. There is usually some tracheal elevation due to dilatation of the left atrium. There is often radiographic evidence of left ventricular enlargement, and there may be an increase in sternal-cardiac contact area. The dorsoventral projection is usually the most helpful in visualizing the typical signs of hypertrophic cardiomyopathy. The radiographic hallmark in this view is the increased width of the cranial portions of the cardiac silhouette with the apex remaining narrow or becoming pointed, giving the appearance of a characteristic "valentine-shaped" heart. A prominence at the two o'clock position is contributed by an enlarged left auricular appendage, and the right prominence at the nine to eleven o'clock position is due to the "displaced" right ventricle.[2] A mild amount of perihilar pulmonary edema is present in cases with left heart failure. In more severe cases in-

creased alveolar densities will be visualized most extensively in the caudal lung lobes.

Angiography is even more helpful than survey radiographs in diagnosing hypertrophic cardiomyopathy. Angiographic studies of the left ventricle reveal severe left ventricular hypertrophy with greatly reduced end diastolic volume. Muscular hypertrophy around the body of the ventricle usually appears to be greater than that at the apex. Many angiocardiograms also show evidence of radiolucent filling defects in the left ventricle caused by hypertrophied papillary muscles. Mitral insufficiency is present along with a dilated left atrium.[7]

Hemodynamic studies indicate two consistent readings that may help diagnose the cardiomyopathy. First, mean systolic pressures remain within normal limits. Second, the end diastolic pressure is consistently increased several times normal.

Laboratory findings with hypertrophic cardiomyopathy are inconsistent. A neutrophilic leukocytosis may occasionally be found. This probably represents a stress reaction but must be remembered when differentiating this disease from bacterial endocarditis. The muscle enzyme levels are also occasionally elevated, presumably from myocardial necrosis or skeletal muscle infarctions from arterial thromboembolism. Renal function tests may show renal pathology if the renal artery is thrombosed.[2]

Congestive Cardiomyopathy

Feline congestive cardiomyopathy is characterized by dilated ventricles and atria with poor myocardial contractility. The result of these changes is the failure of the heart as a pump. Congestive cardiomyopathy is considered a primary myocardial disease of unknown cause; however, several etiologies have been proposed. Present thought is that congestive cardiomyopathy is the result of myocardial damage caused by a variety of myocardial insults. Since the domestic cat is affected by many viral diseases, the possible progression of viral myocarditis to cardiomyopathy presents the most popular hypothesis at this time. Picornaviruses, herpesviruses, and reoviruses have been isolated from affected cats, and a latent or carrier state has been implicated for feline herpesvirus.[7] This still remains largely unsupported, and there have been few observations of a transition from myocarditis to congestive cardiomyopathy. Viral myocarditis is a well-established entity in humans and is also considered a possible cause of cardiomyopathy in man. There are a variety of alternative etiologies that have been postulated with little supporting evidence. One of these is an immunologic disorder causing circulating antimyocardial antibodies and defective suppressor cell function of lymphocytes. Others include endocrine abnormalities involving prostaglandin E_1, and effects of chemical toxins on the myocardium.

The most common presenting clinical signs seen with feline congestive cardiomyopathy include anorexia and lethargy. Other signs include dyspnea, shock, depression, dehydration, hypothermia, bradycardia, and aortic thromboembolism.[3] Pleural effusion is a common result of the right-sided heart faulure, yet ascities is rarely seen. Auscultation may reveal muffled heart sounds due to pleural effusion. Systolic murmurs of mitral and tricuspid insufficiency as well as third heart sounds are also frequently found if there is not excessive fluid in the thorax.

Macroscopic pathology of congestive cardiomyopathy shows generalized cardiomegaly with ventricular and atrial dilatation. This results in a globular-shaped heart with thin, flabby ventricular walls; papillary muscles and trabeculae are flattened and atrophic. Also, areas of endocardial fibrosis are seen as with hypertrophic cardiomyopathy. Hydrothorax is often seen, along with pulmonary congestion and edema, and hepatic congestion. Thromboembolism is also frequently seen, again most commonly in the caudal abdominal aorta.[5]

Microscopic examination reveals only minor cardiac histopathologic alterations. There is usually a mild hypertrophy of the muscle fibers in the ventricular walls, as well as a mild, diffuse myocardial interstitial fibrosis. Unlike the hypertrophic form, myofibril disarray is not present.[5]

The most frequent electrocardiographic abnormalities seen with congestive cardiomyopathy include premature ventricular contractions and a tall, wide QRS complex in lead II. Other abnormalities may be inconsistently observed, but there usually is no significant axis deviation.[7]

Radiology can be helpful in identifying cats with congestive cardiomyopathy; however, due to a high degree of variability, radiographic signs are not normally considered diagnostic. Pleural effusion, a complication often present

in these cats, must be resolved with thoracocentesis or diuretic therapy prior to radiography. The radiographic change most commonly seen is an enlarged, globular-shaped heart due to dilatation of all chambers. Hepatomegaly is another frequently seen sign of right heart failure in addition to pulmonary edema. As stated previously, ascites is rarely seen, unlike right-sided heart failure in the dog.

Angiography is one of the most reliable methods of diagnosing congestive cardiomyopathy if the patient is not debilitated to undergo the study. There is often extreme ventricular dilatation with increased end diastolic volume. Atrioventricular valvular insufficiency is also commonly seen together with dilated atria. Another very distinctive change often observed is a very prolonged circulation time.[3]

Hemodynamic studies have shown two changes that can be helpful in diagnosing congestive cardiomyopathy. First, the left ventricular systolic pressure is commonly low or at the low end of normal. Second, as in hypertrophic cardiomyopathy, the left ventricular end diastolic pressure is consistently several times higher than normal.[7]

Laboratory findings are inconsistent and similar to those found with the hypertrophic form of feline cardiomyopathy. Possible differences that may be observed with the congestive form are an increase in the levels of the liver enzymes due to hepatic congestion, and a pre-renal azotemia.

Restrictive Cardiomyopathy

Restrictive cardiomyopathy is the most recently described form of the feline cardiomyopathies. It is also the most uncommon. Two basic pathologic forms of restrictive cardiomyopathy have been described. The first is characterized by severe endocardial fibrosis. The second form is less common and is associated with increased numbers of abnormal moderator bands in the left ventricle. At the present time, the etiologies of both of these are unknown.

Restrictive cardiomyopathy due to endocardial fibrosis shows many of the same signs as the other cardiomyopathies. It occurs most frequently in middle-aged male cats. The most common presenting signs are dyspnea, anorexia, and thromboembolism. Electrocardiography often reveals conduction and rhythm disturbances including premature ventricular contractions and atrial fibrillation. Radiology frequently shows left atrial enlargement and sometimes shows the presence of pleural fluid. Angiography is again the best way to show the changes of endocardial fibrosis in the living cat. The most frequently observed signs are apical dilatation of the left ventricle and irregular filling defects. A-V valvular insufficiency and severe left atrial enlargement are also common. Gross pathology of the heart reveals severe endocardial thickening, especially of the left ventricular inflow and outflow tracts, papillary muscles, and chordae tendinae. Left atrial enlargement is usually evident. Histologically, severe endocardial fibrosis is seen.[4]

Restrictive cardiomyopathy due to abnormal moderator bands is also most often found in middle-aged male cats. Again, the most common clinical signs include dyspnea, lethargy, anorexia, and thromboembolism. The pathological change in this form of cardiomyopathy is an abnormal network of left ventricular moderator bands and an associated cardiac decompensation. The moderator bands are increased in number and size and are seen bridging the left ventricular free wall and septum and entangling the papillary muscles. Electrocardiographic abnormalities show intraventricular conduction defects, especially right bundle branch block, and atrioventricular blocks and bradyarrythmias. Radiology may reveal generalized cardiomegaly, hydrothorax, and pulmonary edema. Angiography shows irregularly shaped or indented left ventricles and a dilated left atrium.[9]

Aortic Thromboembolism

Systemic arterial embolism occurs in approximately 50% of all cats with symptomatic cardiomyopathy, making this the most common complication accompanying this disease.

Aortic thromboembolism was well-described many years before its relationship with cardiomyopathy was known. This recognition comes from the relatively dramatic clinical signs that occur when a major artery is occluded.

It is generally thought that most thrombi originate in the left atrium, aorta, and apex of the left ventricle. Most are considered to be due to mural or arterial lesions. The most common site of acute major arterial thromboembolism is the caudal aorta at the trifurcation of the external iliac arteries. Usually both rear legs are affected, but the lesion can be unilateral. Other common areas of thromboembolism include

the brachial arteries, the renal arteries, and the mesenteric arteries.

Clinical signs on presentation will be determined by the area of ischemia involved. Hind leg signs range from pain and ataxia soon after the embolism, progressing to complete flaccid paralysis. The affected limbs will be cyanotic, cool, and painful with no femoral pulse. The gastrocnemius muscle is often contracted and the local neural reflexes are lost. Also, a toenail may be clipped short and it will not bleed normally. Renal function tests may show severe azotemia if one of both renal arteries are occluded. Abdominal pain may also result from renal or mesenteric artery embolization.

The severity of signs will depend on how quickly adequate collateral circulation can be established. Simple mechanical occlusion will not prevent the development of adequate collaterail circulation. However, there is a release of vasoactive substances from the thrombus, most importantly serotonin, which causes vasoconstriction of collateral vessels. Therefore, effective collateral circulation only occasionally develops in clinical cases of aortic thromboembolism.[1] Selective arterial or nonselective venous angiography can be used to determine the location and extent of a thrombus as well as to visualize any developing collateral circulation.

TREATMENT

In general, therapy for a cat presented with the common signs of cardiomyopathy (dyspnea or thromboembolism) is directed toward stabilizing the patient until their complete status can be evaluated. Phenothiazine tranquilizers are recommended for two reasons. First, tranquilizers will sedate the animal and reduce stress, which is an important precipitating factor in the cardiac decompensation. Second, these drugs cause vasodilation which will help open collateral vessels in the case of thromboembolism. Also, phenothiazines are hypotensive drugs, which must be remembered if the animal is in circulatory failure.

Oxygen therapy should be administered if the animal is hypoxic, and 35% alcohol can be nebulized if pulmonary edema is severe. Furosemide should also be started in cases with dyspnea from pulmonary edema or pleural fluid. An initial dose of 1 to 2 mg/lb is given intravenously for fast effect. Thoracocentesis can also be done for cases with significant pleural effusion. Aminophylline may be indicated at a dosage of 5 mg/lb for its bronchodilator effects if dyspnea is severe. Rapid digitalization may rarely be indicated in cases with severe heart failure, but cats tolerate digitalis glycosides very poorly and toxicity reactions are common.

Arterial Thromboembolism

Once the patient is stabilized, the most important presenting problem is bilateral rear leg paresis/paralysis due to aortic thromboembolism. The neurologic and muscular status of the area should be evaluated to determine the severity of the insult and to evaluate the progress of treatment. A phenothiazine tranquilizer like acetylpromazine maleate should be given for its sedative effects. Its sympatholytic effect also helps establish collateral circulation by causing vasodilation.

Anticoagulants are also indicated to prevent further thrombosis. Arterial thrombi are initiated by platelet aggregation with secondary fibrin deposition, whereas venous thrombi consist primarily of fibrin clots. Therefore, platelet anti-aggregates are indicated, usually aspirin. A dosage of buffered aspirin of ¼ of a five grain tablet every day can be given to the average-sized cat and is an effective platelet anti-aggregate. Aspirin works by inhibiting the prostaglandin thrombroxane A_2, which is the factor that is thought to promote collateral inhibition and platelet aggregation after aortic thrombosis in the cat.[10] Heparin may also be used for its anti-clotting effects at a dosage of 50 IU intravenously and repeated every eight hours subcutaneously.

Surgical embolectomy can be attempted but is not widely recommended. These cats are often very poor anesthetic risks. Also, the success rate of the surgery is poor due to the associated vasoconstriction and recurrence of the clot. If embolectomy is to be attempted, the patient should be stable and the surgery must be performed as soon as possible to increase the chances of success. The aorta is opened over the area of the thrombus via a ventral midline incision. Indications for embolectomy include thrombosis proximal to the renal arteries, complete neurologic dysfunction of the hindlegs, a worsening condition with medical treatment, and thrombosis in a cat with stable cardiovascular function.

Thrombolytic agents like urokinase or streptokinase may be of benefit, but have not been clinically evaluated; also, prostacyclin has been advocated.

Hypertrophic Cardiomyopathy

Once the diagnosis has been made of the type of cardiomyopathy an individual patient has, treatment can be initiated to try to correct the underlying cardiac problems. In hypertrophic cardiomyopathy the pathogenesis is a stress-related release of endogenous catecholamines which causes stimulation of beta-adrenergic receptors in the heart. This causes an increase in heart rate and myocardial oxygen consumption and a decrease in diastolic filling time. Propranolol is a beta-adrenergic antagonist that is commonly used to counteract the increased level of catecholamines. Propranolol should be used only after pulmonary edema and other signs of heart failure have been resolved. By using this drug, it is hoped that ventricular filling will be improved, oxygen requirements will be reduced, and cardiac automaticity will be decreased. In addition, propranolol inhibits platelet aggregation which may help in the prevention of thrombus formation. Propranolol should not be used in patients with aortic thromboembolism due to its vasoconstrictive effects. It is used at a dosage of 2.5 to 5.0 mg, two or three times a day in an average size cat. The main benefit of propranolol is as a preventative of heart failure in hypertrophic cardiomyopathy, and it is very important that signs of heart failure not be present before or after propranolol is used. A $beta_1$ antagonist may also be used for its more selective effects.

Furosemide is also indicated for long-term treatment of hypertrophic cardiomyopathy. It is given to eliminate excess fluid from the patient and to cause a slightly negative sodium balance. The dosage should be adjusted to the individual's response and be kept to the smallest effective dose on an intermittent schedule.[4]

Congestive Cardiomyopathy

Cats that are diagnosed as having congestive cardiomyopathy usually are presented with pulmonary edema and pleural effusion. As stated earlier, diuretics and thoracocentesis are usually indicated. Shock and dehydration are also common findings, so fluids like 5% dextrose may be administered intravenously. The major treatment for the heart failure is a cardiac glycoside for its positive inotropic and negative chronotropic effects. Digoxin is tolerated very poorly in the cat, so dosages must be monitored very closely. Toxicity is characterized by vomiting, diarrhea, and anorexia. Cats in very serious condition may be rapidly digitalized intravenously with a dose of 0.01 mg/lb of body weight divided into four doses given over four hours. Slow digitalization is preferred and done at a dose of 0.003 to 0.005 mg/lb of body weight per day divided into two doses.[2] Vasodilators such as nitroglycerine ointment may be of benefit in the acute treatment of some cats with heart failure. The prognosis for cats with congestive cardiomyopathy is grave, and they commonly die within the first day or two. Long-term treatment for those that survive the initial episode includes digoxin and furosemide therapy with restriction of exercise and sodium.

Some recent work has also shown that a beta-blocker like propranolol may be of help after the patient has been digitalized and is out of heart failure. It will reduce a high heart rate to allow better diastolic filling and more efficient use of oxygen by the myocardium. Other recent work indicates that an oral vasodilator may be indicated for long-term treatment. It is postulated that the beneficial effects are from the decreased work of the heart due to peripheral vasodilatation.

Restrictive Cardiomyopathy

There has not been sufficient research on the treatment of the two forms of restrictive cardiomyopathy to suggest the best treatment regimen. In many cases progression of the disease is so advanced by the time of presentation that therapy may be of little benefit. Therapy is based on the presenting signs with digoxin and furosemide being two of the most commonly used drugs.

CONCLUSION

Substantial advances in our understanding of feline cardiomyopathy have been gained over the past 15 years. However, these beginnings have dealt largely with the clinical and pathologic aspects of this major problem in veterinary cardiology. Much has yet to be learned about the etiologies and relationships of the different forms of feline cardiomyopathy. There is also much work to be done in finding the best treatments for the wide range of pathological disturbances this disease presents. In addition, it appears that feline cardiomyopathy closely resembles and will be an important contribution to the understanding of human cardiomyopathy. One thing is certain, the understanding of the many aspects of feline cardiomyopathy will continue to be a challenge to researchers and clinicians alike.

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AVMA Sponsors Meeting on FDA's Revised Drug-Use Policy

A recently announced change in the Food and Drug Administration (FDA) policy on the use of drugs in food-producing animals resulted in a conference called by the AVMA at its Schaumburg, Illinois, headquarters on September 27, 1983.

The meeting was organized in response to an outcry of concern from veterinarians and producers that a new policy announced by the FDA's Bureau of Veterinary Medicine last July would impose excessive restrictions on extra-label use of food-animal drugs. Under the FDA's revised policy, a finding of illegal drug residues will no longer be the only reason for initiating punitive action. The revised policy states that use of a drug in food animals for a purpose or in a dosage not specified on the product's label directions will also be justification for punitive action.

The changes recommended at the September conference would assure that the FDA will *not* interfere when, in the veterinarian's professional judgment, there is a need to treat a food animal with a drug or a dosage not specifically approved by the FDA for an existing disease condition.

It is expected that the recommended changes will be reviewed for possible endorsement by the AVMA and other national groups, and hopefully acted upon by FDA authorities.

If the FDA fails to amend its revised policy, it is possible that veterinarians will be practicing under a regulatory cloud of impropriety when they decide to use a drug for purposes not exactly stated on the label.

However, if the recommended changes *are* adopted, FDA authorities would continue to take action against veterinarians in cases where drugs are illegally used and/or distributed for purposes not stated on the product label, and in the absence of a veterinarian-client-patient relationship. FDA authorities would respect the veterinarian's professional prerogatives when a veterinarian-client-patient relationship has been established so that the veterinarian is familiar with the owner, the animal, the premises, and knows the management conditions. FDA authorities, however, will still hold the veterinarian and the client responsible for any drug residues in food products that may occur following treatment regimens that do not follow label directions.