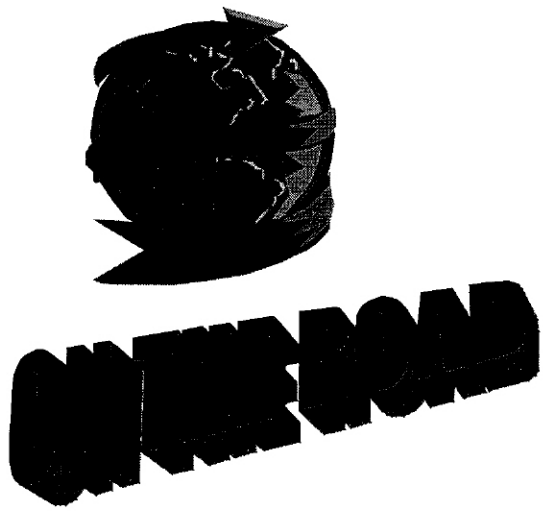

Sharon Young, Mary Woodruff, Tom Hooper and **Dr. Ching Ching Wu** attended the Association of Veterinary Microbiologists Heartland Chapter annual meeting in Madison, Wisconsin, April, 2003.

Dr. **Ching Ching Wu** spoke to swine and poultry groups on antimicrobial usage in London, England and Paris, France, May 2003.

Drs. Leon Thacker, Bill VanAlstine, Alok Sharma, Ingeborg Langohr, and **Phaedra Stiles** attended the North Central Conference for Veterinary Laboratory Diagnosticians in Minneapolis, MN, June, 2003.

Dr. Leon Thacker attended the American Veterinary Medical Association annual meeting in Denver, Colorado, July, 2003.

Dr. **Tsang Long Lin** attended the Ixth International Symposium on Nidoviruses in Egmondaar Zee, Netherlands. May, 2003.

Dr. **Ching Ching Wu** attended the 2003 Conference on Modern Vaccines, Adjuvants and Delivery Systems in Dublin, Ireland. June, 2003.

Idiopathic hypertrophic cardiomyopathy in the cat

One of the most common primary feline cardiac diseases is idiopathic hypertrophic cardiomyopathy (HCM). HCM is characterized by a massive left ventricular hypertrophy without dilation and is present without any other cardiac or systemic disease. The secondary form of HCM is usually associated with disease processes such as acromegaly, systemic hypertension, or hyperthyroidism.

HCM has been more frequently observed in males than females. It is most commonly described in middle-aged cats with a range distribution from 5 months to 17 years. In a study done by Atkins et al, the median survival rate of cats with HCM was about two years. Cats without clinical signs survived longer than those with heart failure or signs of embolism. In the same study, the survival rates of cats having heart rates $\leq$ 200 beats/minute survived longer than those with heart rates $\geq$ 200 beats/minute.

Etiology: The etiology of HCM is usually not known. Multiple etiologies have been proposed including hereditary predisposition, elevation in circulating catecholamines, abnormal myocardial calcium metabolism, abnormal compensatory myocardial hypertrophy due to ischemia and fibrosis, or abnormal primary collagen resulting in secondary ventricular hypertrophy. Researchers have found evidence that some cases of HCM are inherited in the Maine coon cat and the American shorthair as an autosomal dominant pattern. Kraus et al identified a litter of five 18-month-old mixed breed cats that all had HCM.

Pathophysiology: Clinical signs are generally associated with myocardial

hypertrophy due to decreased left ventricular diastolic filling and myocardial ischemia. In many cases there are also systolic abnormalities such as intraventricular pressure changes leading to asynchrony of the contraction and relaxation of the heart muscle.

Depending on the type of myocardial alterations found, there are several complications that can occur. Pulmonary edema can be present when there is an increase in left atrial and pulmonary venous pressure. Arrhythmias can also be present where then is myocardial ischemia. Also, due to localized endocardial injury, circulatory stasis and altered blood coagulability, there is a potential risk of developing a thrombus within the left atrium that could potentially lead to a thromboembolic episode.

Clinical signs: Cats with HCM can be asymptomatic and often experience sudden and unexpected death. Others can show symptoms of acute dyspnea after stressful episodes. On a routine physical exam, HCM can be suspected if there is a murmur or gallop rhythm. When clinical signs are present, they are associated with left sided heart failure or arterial embolization.

Clinical signs observed with HCM are variable and may include dyspnea, tachypnea, pulmonary crackles, lethargy, reluctance to move, syncope, gagging, anorexia, and sometimes abdominal distention or vomiting. When pericardial or pleural effusion is present, the heart and lung sounds can be muffled.

Thromboembolism has been recognized in approximately 50% of cats with HCM. When aortic thromboembolism is present, the clinical signs will vary according to its location. The most common sign of thromboembolism is unilateral or bilateral pelvic limb paresis or paralysis. When the embolization occurs at the brachial artery, pain and paresis of forelimbs has been observed. Blockage of the renal artery will produce acute renal failure. If the thromboembolism occurs at the cranial mesenteric artery, it is possible to observe clinical signs of colic. Central nervous system abnormalities can be seen if the embolus affects the cerebral artery. Respiratory distress can be observed after embolization of the pulmonary vasculature.

Diagnosis: It is possible to have a tentative diagnosis based on the history, physical examination, electrocardiographic findings, and thoracic radiographs.

Arrhythmias and conduction disturbances have been detected in 60-70% of cats with HCM. Several types of arrhythmias have been observed, including premature ventricular contraction, atrial fibrillation, atrial tachycardia, atrial premature contraction, paroxysmal ventricular tachycardia, and ventricular bigeminy. Varying degrees of atrioventricular block, right and left bundle branch blocks, and Wolff-Parkinson-White syndrome are some of the conduction disturbances that have been observed. Prolongation of P waves greater than 0.04 seconds and prolongation of the QRS complex greater than 0.04 seconds with R wave amplitude greater than 0.9 mV in lead II are parameters that have been used as electrocardiographic indications of an enlarged left ventricle.

Depending on the degree of compromise, the thoracic radiographs may show different degrees of cardiomegaly with pulmonary edema. On the ventrodorsal view, the classic radiographic sign of HCM is a "valentine" shaped cardiac silhouette with biatrial enlargement and normal looking apex. It is common to observe a banana-shaped cardiac silhouette on the lateral view. When pericardial effusion is present, it is possible to observe generalized enlargement and rounding of the cardiac silhouette on the ventrodorsal view. Radiographic evidence of cats with pulmonary edema usually has a patchy and focal distribution along the pulmonary vessels.

Definite diagnosis can be done using echocardiography. With this method, it is possible to rule out secondary causes of HCM such as congenital aortic stenosis, chronic systemic hypertension, chronic anemia and hyperthyroidism. Typical echocardiographic abnormalities found in HCM are symmetrical hypertrophy of the

left ventricular caudal wall and interventricular septum, reduced dimensions of the left ventricular chamber, and ventricular hyperkinesis.

Treatment: Medical therapy of HCM is aimed to control the clinical signs and has been adapted from that used in human medicine. To relieve signs of pulmonary edema, diuretics such as furosemide have been used. If refractory right-sided heart failure develops, a second diuretic such as hydrochlorothiazide can be used. For treatment of congestive heart failure, an angiotensin-converting enzyme inhibitor such as captopril can be used. Beta adrenergic-blocking agents (propanlol) or calcium channel blockers (dialtiazem hydrochloride) have been used for the treatment of the diastolic dysfuntion.

Medical management of thromboembolism has been mostly empirical. Heparin therapy has been used to prevent additional formation of thrombi. The use of thrombolytic agents such as streptokinase, urokinase, and tissue plasminogen activator are expensive and have not shown to be consistently effective. A potential complication of their use is uncontrolled bleeding due to their fibrinolytic effect.

Aspirin has been used to prevent platelet aggregation and thrombus formation. Follow-up studies have not shown that aspirin prevents clot formation in all cases. Some cats with long term aspirin therapy, although presenting thromboembolic episodes, have shown shorter recovery periods.

Cats with HCM have very little cardiovascular reserve and are very sensitive to stress. Caution should be observed when administering rapidly intravenous fluids since there is the potential to cause rapid decompensation and the initiation of congestive heart failure.

Pathology: HCM is characterized by hypertrophy of the left ventricle free wall ($\geq$ 0.6 cm), papillary muscles and interventricular septum. The size of the left ventricular lumen is decreased. The muscle hypertrophy can be symmetrical or asymmetrical. It is sometimes possible to find thickening of the mitral valve with enlargement of the left atria.

In a study performed by Liu, a total of 51 cats with HCM were studied. In this study, 70% of the cats presented symmetric, concentric ventricular hypertrophy with normally arranged cardiac muscle cells in the septum. In the remaining 30%, the cardiac muscle cells in the septum were disorganized. This lesions seems to be very specific to primary HCM because it was rarely found in cats with secondary HCM.

In a previous study (Van Vleet), 10 cats with HCM had gross lesions including cardiomegaly, diffuse symmetric left myocardial hypertrophy, small left ventricular cavities, and dilated left atria. The histopathology lesions found were hypertrophy and disorganization of cardiac muscle cells, interstitial fibrosis, and fibromuscular hyperplasia of small intramural coronary arteries. Other lesions observed include hypertrophy and disorganizaiton of myocytes of the left ventricular wall and septum. The endocardium, conduction system, or myocardium may present focal or diffuse degeneration, interstitial fibrosis, and chondroid metaplasia. In 50% of cats with HCM, the intramural coronary artery walls were thickened and had narrow lumens.

-by Luz Borrero-Yu, ECFVG Student
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