

Hypertrophic Cardiomyopathy in a Mixed Breed Cat Family

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ABSTRACT. A spayed female mixed cat (case 1) and its female offspring, the result of a pairing between case 1 and its male sibling, were diagnosed with hypertrophic cardiomyopathy (HCM). A pedigree survey revealed that the prevalence of HCM was at least 12.5% in the family, which was considered to be significantly higher than that in a hospital-based population (approximately 1.6%). Thus, this finding seems to support the suspected occurrence of familial HCM in this group of related cats.

KEY WORDS: feline, hypertrophic cardiomyopathy, pedigree survey.

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Hypertrophic cardiomyopathy (HCM) is one of the most common cardiac diseases in humans and cats [3]. In the majority of human cases, there are several genetic defects related to the pathogenesis of HCM [10]. Recent substantial studies have revealed that familial HCM in humans accounts for greater than 90% of cases, indicating that appropriate evaluation should be required in the patient's parents, children and siblings [2]. In cats, there is evidence that HCM is inherited as an autosomal dominant pattern in the Maine coon cat [4] and the American shorthair [9]. In addition, anecdotal evidence indicates that HCM in other breeds, including Rex, British shorthair and Ragdoll, may also be inherited [8]. However, the etiology of most feline HCM is not fully understood, and it is unknown whether familial HCM accounts for the majority of feline HCM as in humans. Thus, further information in other breeds would be required to determine the etiology of feline HCM. The present paper describes the familial occurrence of HCM in a mixed cat family.

Case presentation: Case 1 was a spayed female mixed cat, aged 14, weighing 3.1 kg, and was presented for decreased activity and anorexia. The authors (K. N. and M. K.) had already diagnosed the cat as chronic renal failure (CRF), and had conducted supportive therapy. On admission, the heart rate was 108 beats per minute (bpm), and an arrhythmia was auscultated. An electrocardiogram revealed right bundle branch block (RBBB) and 1st degree atrioventricular (AV) block with occasional escape beats (Fig. 1). Thoracic radiography showed an enlargement of both the atrium and the caudal vena cava. Echocardiography confirmed asymmetrical hypertrophy of the left ventricular free wall and intraventricular septum.

Case 2 was a spayed female mixed cat, aged 14, weighing 2.7 kg, and was presented for lethargy and anorexia. This cat was the offspring of case 1 and its sibling. The cat had been diagnosed with CRF prior to presentation, and supportive therapy had been initiated. The heart rate was 116 bpm, and an arrhythmia was auscultated. Indirect measurement of peripheral blood pressure was within reference range. The electrocardiogram (Fig. 1), thoracic radiography and echocardiography revealed the similar findings to those of case 1.

In both cases, the therapy consisted of oral enalapril malate, diltiazem hydrochloride, isosorbide dinitrate and furosemide. No treatment for the arrhythmia was given. Thereafter, the AV conduction disturbance became complete on day 28 in case 1. Both cats became unresponsive to treatment for heart failure, and died on day 138 and 320, respectively.

Pathological findings: The gross and histological findings were essentially similar in both cases. At necropsy, the hearts were enlarged and globular in shape. On the cut surface, diffuse left ventricular hypertrophy and left atrial dilatation were observed. Microscopic examination of the hearts revealed multifocal areas of strikingly disorganized cardiac muscle cells, interstitial myocardial fibrosis (plexiform fibrosis) and arteriosclerosis of small intramural coronary arteries (fibromuscular hyperplasia), indicating hypertrophic cardiomyopathy (Fig. 2). The proximal portions of the left and right bundle branches were enclosed and compressed by the fibroplastic lesions of the interventricular septal crest, resulting in partial loss and/or decrease in the number of the specialized conduction cells. The other pathological findings were congestion of the liver and the spleen, and chronic interstitial nephritis.

Pedigree survey: By reevaluating the medical records of related cats and obtaining information from the owner, an attempt was made to determine the prevalence of HCM in

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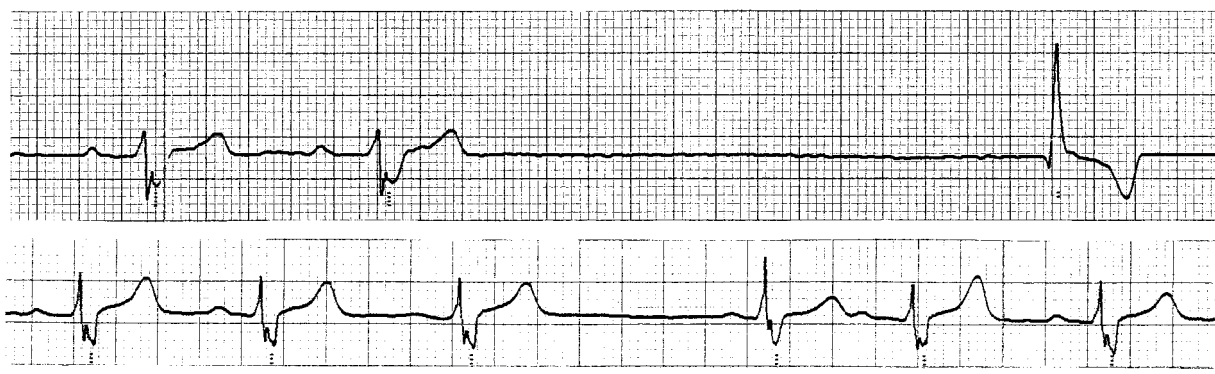


Fig. 1. Electrocardiograms (ECGs) in cats with HCM. The upper ECG was recorded from case 1 on day 5, showing right bundle branch block and 1st degree atrioventricular (AV) block with a escape beat. The AV conduction disturbance became complete on day 28 (not shown). The lower ECG was recorded from case 2, showing the same findings as in the case 1. These ECGs were taken from lead II, and vertical and horizontal lines represent 1 mV and 1 second, respectively.

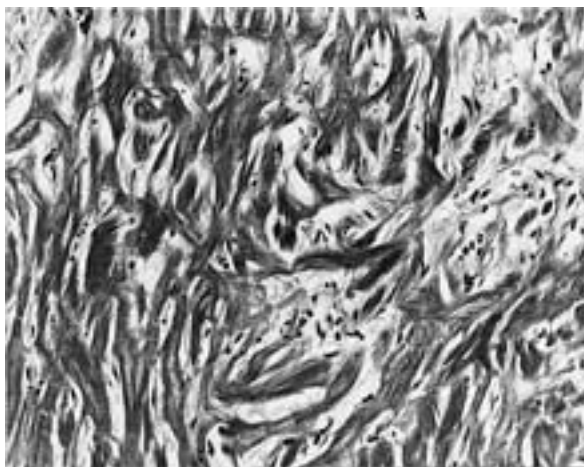


Fig. 2. Histopathological section from the left ventricular free wall in the case 1. Marked disorganization of hypertrophied cardiac muscle cells and plexiform fibrosis are present. The same findings are seen in the section of the case 2. HE. $\times 170$.

the family (Fig. 3). A male American shorthair and female mixed cat were mated, producing a litter of 5 kittens. Detail information was unavailable for three of these littermates. The remaining two cats, i.e. case 1 and its sibling, subsequently mated, producing nine kittens. For five of these kittens, detail information was not obtained. As above mentioned, case 2 was diagnosed to have HCM. Other two male cats were suspected to have HCM, because one cat had died due to aortic thromboembolism, and the other had died suddenly with considerable hypertrophy of the ventricular wall. The remaining male cat was found to be unaffected with HCM. Thus, the pedigree survey revealed that the prevalence of HCM in this family was at least 12.5% (2/16). When the suspected cats were assumed to have HCM, the prevalence would increase to at least 25%.

The present paper describes clinical and pathological

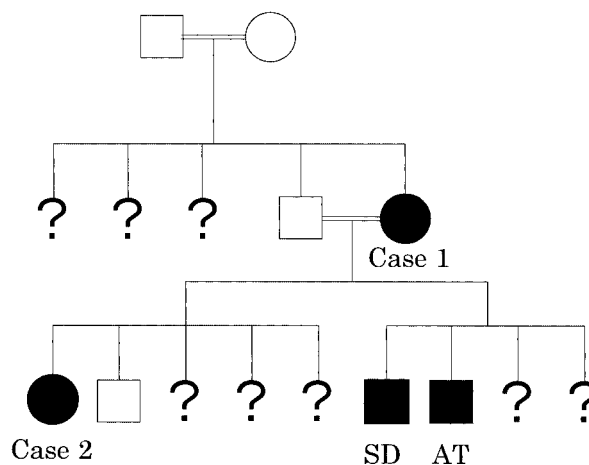


Fig. 3. Pedigree of the cats with HCM. Opened and closed symbols represent non-affected and affected cats, respectively. Square and circle indicate male and female, respectively. The sire of case 1 was an American shorthair and the dam was a mixed cat. The prevalence of HCM was estimated to be at least 12.5%. In addition, two male cats were suspected to have HCM. SD: sudden death. AT: aortic thromboembolism. A symbol '?' indicates an individual in which sex and clinical outcome could not be determined.

findings of HCM in two related cats. The prevalence of feline HCM has been reported to be approximately 1.6% in a hospital-based population [1]. Although a complete pedigree survey could not be performed, the prevalence of HCM in this family was significant. Therefore, this finding supports that there was a familial occurrence of HCM in the present cases.

A familial occurrence of feline HCM has been described, and the mode of inheritance was not determined [5]. However, recent studies have confirmed that feline HCM is inherited as an autosomal dominant trait in the Maine coon cat [4] and the American shorthair [9]. Therefore, it would

be noteworthy that the American shorthair was included as a member of the present pedigree. Because only limited cats were included in this survey, the mode of inheritance could not be determined.

In cats with HCM, the most common electrocardiographic abnormality is the enlargement of the atrium and/or ventricle. Left anterior fascicular block was observed in 11 to 33% of patients and may be a common arrhythmia associated with feline HCM [3]. Other arrhythmias including supraventricular arrhythmias, sinus bradycardia and AV block have also been described. Such arrhythmias were reported to occur in approximately 30% of cats with HCM [7]. However, the authors believe that RBBB is an uncommon arrhythmia in feline HCM, and it should be noted that this arrhythmia was recorded in both cases. Pathological changes in the cardiac conduction system of cats with HCM have been reported [6]. They were observed primarily in the AV bundle and bundle branches. These findings might explain the reason why left anterior fascicular block is commonly recorded in feline HCM. In the present cases, the lesions in the left and right bundle branches caused by fibroplasia of the interventricular septal crest might have played an important role in the occurrence of the RBBB and atrioventricular block.

In conclusion, the present feline HCM would be charac-

terized by familial occurrence, and the presences of AV block and RBBB.

REFERENCES

1. Atkins, C. E., Gallo, A. M., Kurzman, I. D. and Cowen, P. 1992. *J. Am. Vet. Med. Assoc.* **201**: 613–618.
2. Baty, C. and Watkins, H. 1998. *Q. J. Med.* **91**: 791–793.
3. Fox, P. R. 2000. pp. 869–923. *In: Textbook of Veterinary Internal Medicine*, 5th ed. (Ettinger, S.J. and Feldman, E.C. eds.). W. B. Saunders, Philadelphia.
4. Kittleson, M. D., Meurs, K. M., Munro, M. J., Kittleson, J. A., Liu, S.-K., Pion, P. D. and Towbin, J. A. 1999. *Circulation* **99**: 3172–3180.
5. Kraus, M. S., Calvert, C. A. and Jacobs, G. J. 1999. *J. Am. Anim. Hosp. Assoc.* **35**: 293–296.
6. Liu, S.-K., Tilley, L. P. and Tashjian, R. J. 1975. *Recent Adv. Stud. Cardiac Struct. Metab.* **10**: 681–693.
7. Liu, S.-K. and Tilley, L. P. 1980. *Yale J. Biol. Med.* **53**: 191–211.
8. Meurs, K. M. and Spier, A. W. 2001. pp. 261–266. *In: Consultations in Feline Internal Medicine 4* (August, J.R. ed.). W. B. Saunders, Philadelphia.
9. Meurs, K., Kittleson, M. D., Towbin, J. and Ware, W. 1997. *J. Vet. Int. Med.* **11**: 138 (abstract).
10. Wynne, J. and Braunwald, E. 2001. pp. 1751–1802. *In: Heart Disease*, 6th ed. (Braunwald, E., Zipes, D.P. and Libby, P. eds.), W. B. Saunders, Philadelphia.