Mitral Stenosis with Bacterial Myocarditis in a Cat

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ABSTRACT. An eleven-year-old female Japanese mongrel cat was referred to the Tottori University Veterinary Teaching Hospital for assessment of acute paresis and dyspnea. Two-dimensional echocardiography showed a hydropericardium. The mitral valve leaflets were thickened, the separation of the right and left leaflets was not complete. Treatments with intravenous fluids of lactate Ringer solution, furosemide, urokinase, antibiotics were initiated, but did not improve the respiratory failure. The cat died 10 days later. From pathological and microbiological examinations, this was an unusual case diagnosed as acquired mitral stenosis associated with congenital malformation of the mitral valve complex, and accompanied by secondary infectious myocarditis with Streptococcus canis.

KEY WORDS: mitral stenosis, myocarditis, Streptococcus canis.


An eleven-year-old, 4.0 kg female Japanese mongrel cat was referred to the Tottori University Veterinary Teaching Hospital for assessment of acute paresis. The history given by the owner was that the cat had sudden paresis and dyspnea three weeks before. The paresis resolved by the next day; however, the dyspnea had progressed. On the day of admission, the cat presented paresis again.

The cat was subjected to a complete clinical evaluation under oxygen inhalation. Severe dyspnea and lethargy were observed. The femoral pulse was faint and the hind limbs felt cold. The respiration and heart rates were remarkably high, 180 breaths/min and 200 beats/min, respectively. Cardiac auscultation revealed tachycardia with slight murmur. Full complete blood counts and serum biochemistry tests were performed. Leukocytosis (18,200 counts/µl), slight increases in blood urea nitrogen (40.7 mg/dl), creatine (1.3 mg/dl) and alanine aminotransferase (407 U/l), and a great elevation in creatine phosphokinase (> 40,000 U/l), were found. Serum cardiac troponin I was also high, at 0.8 ng/ml (reference range: 0.03–0.16 ng/ml [9]). From these findings, some myocardial cellular damage was suspected. Slight abnormalities were also seen in the blood coagulation profiles, including the platelet counts (120,000/µl), prothrombin time (10.6 sec), and activated partial thromboplastin time (27.9 sec). Both feline leukemia virus antigen and feline immunodeficiency virus antibody tests were negative.

Thoracic radiographs revealed a rounded cardiac silhouette with elevated pleural parenchymal density, suggesting the presence of pulmonary edema. Two-dimensional echocardiography showed a pericardial effusion, and 250 ml of effusion fluid was removed by pericardiocentesis. The specific gravity of the effusion fluid was 1.018 and the cytology detected a moderate amount of segmented neutrophils. B-mode echocardiography revealed a greatly enlarged left atrium (AO:LA ratio=1:2.1). The presence of thromboembolism was not demonstrated in any of the cardiac chambers. An echodense mass was detected on the origin of the left mitral leaflets (Fig. 1). The mitral valve leaflets were thickened and the separation of the right and left leaflets was not complete, with doming of the anterior mitral valve leaflet into the left ventricle during diastole. Narrow jet stream into the left ventricle during diastole was found by color flow-doppler echocardiography. Regurgitant mitral flow during systole was also found (Fig. 2). M-mode measurement revealed a marked hypsystole of the left ventricle (FS: 6.8%). Based on these findings, mitral stenosis was strongly suspected. Treatments with intravenous fluids of lactate Ringer solution, furosemide (1 mg/kg IV every 12 hr, and 2.5 mg/kg in intravenous fluids), urokinase (6,000 U IV over 60 min), antibiotics (ofloxacin, 5 mg/kg SC), were initiated, but did not improve the respiratory failure. On the next day, the same volume of effusion was detected again by pericardiocentesis with echocardiography.

Fig. 1. Two-dimensional echocardiogram showing the right parasternal long axis view. There is an enlarged left atrial (LA). The mitral valves (MV) are thickened, and an echodense mass was located at the origin of the left mitral leaf (arrow).
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Continuous treatments were recommended, but the owner declined this. The cat was discharged and died 10 days later.

Necropsy was performed within a few hours of the death. The lungs were diffusely congested and edematous, and partial emphysema was observed. The left atrium was severely enlarged. The left ventricular free wall side papillary muscle was extremely hypertrophied, and the chordae tendineae were not observed adjacent to the mitral valve. Both mitral valve leaflets were thickened, white-colored and smooth on their surfaces, and they were fused to each other, producing an oval and stenosed orifice (Fig. 2). Yellow- to white-colored foci, varying in sizes (2 to 5 mm), were founded in the myocardium (Fig. 2), lung, and parenchyma of the liver that were especially prominent in marginal areas (Data not shown).

Histological examinations of the ventricular and papillary myocardium revealed the characteristics of myocarditis, which were accompanied by randomly distributed multifocal areas of necrosis surrounded by inflammatory infiltrates chiefly consisting of neutrophils. In the necrotic regions, multiple Gram-positive cocci were observed, suggesting that the myocarditis was of bacterial etiology. The myocardium surrounding the focal necrosis was mildly atrophied and partially disorganized, however, fibrosis was extremely mild (Fig. 3). The mitral valve apparatus was fibrous and thickened, and accompanied by mild mucus production and collagenous proliferation. Bacterial multiplication and inflammatory infiltrations were absent in the mitral valve apparatus. Direct junctions between the mitral valve and the papillary muscles were observed (Data not shown). Obvious structures of the chordae tendineae were not present (Fig. 4). In other organs including the lungs, liver, kidneys, and spleen, there were severe supplicative inflammations characterized by bacterial multiplications and inflammatory infiltrations with neutrophils, macrophages, lymphocytes, and plasma cells. These findings indicated that this case had generalized bacteremia. Centrilobular congestion was also found in the liver. Atrophy, fatty degeneration, and coagulative necrosis were found in related hepatocytes.

Heart specimens were subjected to bacteriological examinations. Aerobic culture on heart infusion agar plates with 5 percent sheep erythrocytes yielded a heavy growth of beta-hemolytic colonies yielding a heavy growth of beta-hemolytic colonies in pure culture. These colonies were identified as *Streptococcus* species by their positive Gram staining, the absence of catalase, and the absence of tolerance of 6.5 percent sodium chloride. The Lancefield group was detected from the isolate by slide agglutination test for the identification of streptococcal group with commercial rabbit antisera (Denka-Seiken, Tokyo, Japan), and was found to be group G. PCR amplification was carried out using species-specific primers to the *sodA* gene of *Streptococcus canis* [4]. The isolate was positive for the *sodA* gene, with a specific amplicon of 363 bp (data not shown).

This case had severe left ventricular heart failure includ-
ing left atrial enlargement, pulmonary, and pericardial effusion, and these were suspected to be caused by mitral stenosis. Characteristic echocardiography findings of this cat, including thickened mitral valve, poor leaflet separation were similar to those reported in cats with mitral stenosis [10], and diastolic doming of anterior mitral valve which was found in over half of mitral stenosis dogs [6] were also detected. Although diastolic mitral filling velocity was not determined, color flow-doppler echocardiography showed narrow jet stream into the left ventricle during diastole. Mitral valvular stenosis would obstruct ventricular filling and limit cardiac output.

In cats, mitral stenosis is an extremely rare cardiac disease, and may occur as a congenital or acquired condition. Only five cats of mitral stenosis have been reported to date. They were two cats with congenital supravalvular mitral stenosis [2, 10], one cat with hypertrophic cardiomyopathy [12], and two cats of secondary acquired lesions related to congenital dysplasia of the mitral valve complex [10]. In our case, the macroscopic changes in the mitral valve com-

plex—thickened mitral valve leaflets and hypertrophied papillary muscles resulting in absence of chordae tendineae—are compatible with the anatomical lesions previously described in malformation of the mitral valve complex in cats [8]. These anatomic abnormalities would reduce leaflet mobility and advance the dynamic outflow tract obstruction or increase systolic valvular regitation secondly as same as two cats previously reported [10].

Additionally, bacterial myocarditis caused by *S. canis* was present in our case. *S. canis* is a beta-hemolytic Lancefield group G streptococcus isolated as commensal flora from the skin and mucosa of dogs [3]. *S. canis* has been isolated from a variety of animals, including dogs, cats, rats, mice, foxes, and raccoon dogs [3, 5, 7, 13]. In cats, this pathogen has been reported to cause arthritis [5], contagious lymphadenitis, pharyngitis, and submandibular edema [5, 13]. Streptococci, especially *S. canis*, have been reported to be the most common cause of infectious endocarditis, and are likely to infect the mitral valve in dogs [11]. This is the first reported case of *S. canis* myocarditis with focal necrosis in a cat. In human beings, chronic rheumatic endocarditis infected with group A beta-hemolytic Streptococcus is the most frequent etiology of acquired mitral stenosis, and fusions of the valve commissures, with or without thickening of the leaflets and chordae tendineae, are characteristic findings [1]. However, no endocarditis was recognized in the cat described in this report. In addition, there was no evidence from the histological findings to support the presence of an immune-mediated phenomenon. The random, multifocal distribution and intramyocardial location of the lesions suggested that the organisms were of hematogenous origin. Indeed, as other organs had severe suppurative lesions infected with Gram-positive streptococci, generalized hematosepsis with this organism was supported.

The size and degree of fibroplasia of the myocardial lesions may be consistent with the duration of the inflamma-
tion. The myocardial lesions might have aggravated heart function, but these lesions seemed to be comparatively newer than the ventral lesions. There was only a mild fibroplastic component in the circumferential myocardium of the focal necrosis in this case, suggesting that the hypertrophied papillary muscle and absence of chordae tendineae were not due to a result of the myocarditis. In addition, there were no inflammatory infiltrations of the junctions of the papillary muscles and mitral valve leaflets. From these features, this case was diagnosed as acquired mitral stenosis associated with mitral valve complex malformation, and accompanied by secondary infectious myocarditis with *S. canis*.

In a previous report, nine of 15 dogs with mitral stenosis were euthanatized or died by 2.5 years of age [6]. However, two cats with mitral stenosis related to mitral valve complex malformation reported previously [10] and a cat in the present report, all were over 10 years old. In cats, mitral stenosis associated with mitral valve dysplasia may develop subclinically until the terminal stage. In our case, the bacterial infection, which might be resulted from aging or less resistant to infect, developed acutely and contributed to the deterioration in this cat’s condition.

REFERENCES