Chronic Myocardial Infarction Due to Arteriosclerosis of Coronary Arteries Followed by Acute Thromboembolism of Caudal Abdominal Aorta in a Cat

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ABSTRACT. A 10-year-old cat with the paresis of hind limbs was initially diagnosed as a hypertrophic cardiomyopathy followed by acute thromboembolism of caudal abdominal aorta from the findings of the medical examinations. However, this case was proved to be an chronic myocardial infarction due to arteriosclerosis of coronary arteries by the pathologic diagnosis. In the left ventricular, the hypertrophy and the narrowing were slight, and a coagulative infarction was seen obviously. The intramural coronary arteriosclerosis showed thickening of the wall due to medial hyperplasia by fibrosis, and arterial stenosis. Myocardial infarction and arteriosclerosis are scarcely any reports of these lesions in cats. This case is valuable for an extremely rare case of myocardial infarction in the cat.

KEY WORDS: arteriosclerosis, feline, myocardial infarction.

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A 10-year-old male mixed-breed cat weighing 4.7 kg was referred to the Veterinary Teaching Hospital of Tottori University because of the paresis of hind limbs and inappetence. Physical examination revealed the dehydration, slight pale mucosa, absence of femoral arterial pulses and cold hind footpads. In neurologic examinations, the proprioceptive positioning of hind limbs was diminished. Patellar reflex did not produce extension of the stifle. The pelvic limb flexor reflex was lost. The test for pain to the femoral and back skin revealed normal responses, and the perineal reflex produced a normal reaction and the tail moved vigorously. These findings were typical signs of acute hindlimb arterial thromboembolism.

In the first medical examination, hematologic abnormalities included high white blood cell (WBC) counts (17,800/ μ l) and a slight tendency of anemia. In serum biochemical values, the creatine phosphokinase (CK) was 131,320 U/l, the lactic dehydrogenase (LDH) was >900 U/l, the alanine aminotransferase (ALT) was 349 U/l, and the aspartate aminotransferase (AST) was 860 U/l. Other hematologic and serum biochemical abnormalities were not found. Since serum concentrations of cortisol and thyroxine (T₄) were 13 ng/ml (reference range, 5 to 60 ng/ml) and 8.0 ng/ml (reference range, 12.8 to 22.1 ng/ml), respectively, hyperadrenocorticism and hyperthyroidism were excluded from the diagnosis of this case.

Cardiac disorders were found using the electrocardiography (ECG), radiography and echocardiography. In the ECG, tachycardia at the heart rate of 232 beats/min was recorded. Absence of P waves, signified atrial fibrillation, increased R wave amplitudes (1.55 mV) and slight left axis deviation in the mean QRS electrical axis of the frontal plane (40°), signified left ventricular (LV) hypertrophy, were observed. The thoracic radiograph showed LV enlargement and biatrial enlargement, so-called valentine-

shaped heart. Echocardiographic findings revealed a dilated left atrium and a stricture of LV. An echogenic area in LV wall, which size was approximately 10 × 10 mm², was observed. Increased wall thickness was observed in the interventricular septum (9.5 mm) and LV wall (8.7 mm) by M-mode echocardiography measurement, as well as diastolic failure of LV. The LV minor axis internal dimension at end-diastole and end-systole were approximately 5.1 mm and 1.5 mm, respectively. The per cent fractional shortening was about 70%. The systolic and diastolic pressures of the thoracic limb were 134 mmHg and 96 mmHg, respectively. This case was initially diagnosed as a hypertrophic cardiomyopathy (HCM) from the findings described above.

By Doppler echocardiography, the presence of thromboembolism in the caudal abdominal aorta was demonstrated. Medical treatments based on the clinical findings were carried out after the thromboembolism was extirpated surgically. Urokinase, which mediates thrombolysis by activating plasmin, was administered at 6,000 IU IV over 60 min. After the fibrinolytic therapy, a low-dose aspirin (25 mg/head PO) every 3 days was administered to prevent an initial thromboembolic event. Abnormal serum biochemical values were improved rapidly 3 days after extirpation of the thrombus.

Other findings except for the cardiac examination during the term of medical treatments included leukocytosis ranged between 18,000/ μ l and 70,000/ μ l, slight chronic anemia, mild increases of urea nitrogen (BUN) and creatinine (CRE), which increased gradually during the medical treatments, and rapid upturn of ALT and AST in the latter treatment term.

Four classes of drugs were administered to improve the cardiac performance. The drugs were as follows: diltiazem, a calcium channel blocker; propranolol, a beta-adrenergic receptor blocker; dipyridamole, a vasodilator by increasing

Fig. 1. The cross sectional finding of the left ventricular (LV). The LV hypertrophy and the narrowing of the LV outflow tract were mild. A coagulative infarction about 1.3 cm in diameter was seen obviously in LV free wall.

the adenosine levels in the extracellular space fluid; and benazepril, an ACE inhibitor. The heart rate was controlled less than 200 beats/min by these medicines. Finally, the patient recovered from the absence of femoral arterial pulses, cold hind footpads and hind limbs paresis. However the cat that became vigorous died unfortunately from a heart attack due to a hard fight with an inmate cat.

We had the chance of the pathological dissection after the cat died. Macroscopically, the LV expansion was not found although a slight biatrial dilation was seen. In the cross sectional finding, the LV hypertrophy and the LV outflow tract obstruction were mild (Fig. 1). But a coagulative infarction about 1.3 cm in diameter was seen obviously in LV free wall. It was gray and decidedly sunken on the surface of the LV wall, which likes an old infarct (Fig. 1). Other gross pathologic abnormalities were not observed in the heart including the thickening and fibrosis of the valves and the right ventricular hypertrophy.

The histopathologic changes included coagulative necrosis of the LV myocardium and arteriosclerosis of the intramural coronary arteries of the LV (Figs. 2, 4). The coagulative necrosis was chiefly fibrous and mild infiltration of inflammatory cells (Fig. 3). This finding implied that it was old infarct. In addition, the necrosis extended diffusely to the whole circumference of the left ventricular myocardium. The intramural coronary arteriosclerosis showed thickening of the wall due to medial hyperplasia by prominent fibrosis (Fig. 4). It was accompanied by arterial stenosis, but not atheromas. Although myofibers hypertrophied mildly in the interventricular septum and the LV wall, the disarray of myofibers, which is a typical finding of HCM, was not observed in the whole ventricular wall except for an extreme particle segment of the intraventricular septum. From these histopathologic findings, this case was finally determined as a myocardial infarction by arteriosclerosis of the intramural coronary arteries, although it was

Fig. 2. Microscopic finding of the coagulative necrosis in the LV myocardium. The necrosis extended diffusely to the whole circumference of the LV myocardium. H & E stain. $\times 30$.

Fig. 3. Microscopic finding of the coagulative necrosis in the LV myocardium. The coagulative necrosis was chiefly fibrous and mild infiltration of inflammatory cells. This finding implied that it was old infarct. H & E stain. $\times 152$.

Fig. 4. Microscopic finding of the arteriosclerosis of the intramural coronary arteries of the LV. The intramural coronary arteriosclerosis showed thickening of the wall due to medial hyperplasia by prominent fibrosis. It was accompanied by arterial stenosis, but not atheromas. H & E stain. $\times 465$.

diagnosed as HCM based on the results of the medical examinations.

In the findings of other organs, kidneys had a chronic suppurative pyelonephritis. Infiltration of inflammatory cells, i.e., neutrophils, macrophages and lymphocytes, extended from large areas of the renal parenchyma to the renal pelvis, and the disappearance and slight fibrosis of the glomeruli and tubules were observed. Congestions were found in the spleen and liver. In the liver, the hepatocytes and Kupffer cells included hemosiderin in the cytoplasm implied that the congestion was relatively old.

Myocardial infarction and arteriosclerosis of coronary arteries are extremely rare events in animals, although they are principal causes of acute or chronic heart failure in humans. There are scarcely any reports of these lesions in cats. In animals, myocardial infarction is almost found as secondary lesions of other disorders lead to myocardial ischemia; coronary embolism secondary to infective endocarditis of the aortic valve [11, 12], atherosclerosis observed in a few dogs with severe hypothyroidism [7], and secondary to septicemia and neoplasms [12]. In this case, these lesions were not observed except for arteriosclerosis of the intramural coronary arteries.

Arteriosclerosis is defined as chronic arterial wall change consisting of hardening, loss of elasticity and luminal narrowing. Under the general term, arteriosclerosis has three different forms: atherosclerosis which has plaques containing lipid material in arterial walls (atheromas), Monckeberg medial sclerosis or calcification, and arteriosclerosis which shows thickening of the intima due to smooth muscle hyperplasia, medial fibrosis or hyalinization, and narrowing of the lumen. Arteriosclerosis is frequent in patients with systemic hypertension in humans. In animals, systemic hypertension occurs in chronic renal disease, hyperthyroidism, hyperadrenocorticism, and pheochromocytomas. The lesions of the intramural coronary arteries in this case showed thickening of the wall due to prominent medial fibrosis and arterial stenosis. Atheromas was not observed. The arteriosclerosis in this case existed only in the intramural coronary arteries, although several findings in the kidney were seen pathologically and biochemically. Systemic hypertension, hyperthyroidism, hyperadrenocorticism, and pheochromocytomas were not observed in this case.

This case can be obviously diagnosed as a chronic myocardial infarction, not as HCM. Firstly, the LV hypertrophy, the LV outflow tract obstruction, and the left atrial dilation were slight in the pathological findings. Secondly, the disarray of myofibers in the ventricular wall, which is an important finding for the diagnosis as HCM, was hardly found in microscopically. Thirdly, the finding of wall thickening of the intramural coronary arteries in this case was obviously different from that in HCM. Although the damage to endothelium, disruption of elastic fibers, intimal edema, proliferation and migration of smooth muscle cells, and the increase in the number of collagen fibers are the major causes of wall thickness of the intramural coronary arteries in HCM [10], these were not observed in this case at

all. Also, this case is not different from restrictive cardiomyopathy (RCM) or unclassified cardiomyopathy. In unclassified cardiomyopathy, although the most characteristic findings seem to be diastolic failure of LV at several degree, hypoplasia of cardiovalvulae and the severe extension of biatrium, the diagnostic criteria in pathophysiology and pathology are not established yet [2–4].

It is supposed that the causes of the myocardial infarction in this case might be due to arteriosclerosis of the intramural coronary arteries caused by aging and slight chronic anemia. Reduction in the hemoglobin concentration may compromise oxygen supply to infarcted or ischemic myocardium, which may promote arrhythmias, worsen hypotension, and increase infarct size [5, 6, 8]. In humans, it is reported that anemia in patients with acute myocardial infarction is strongly associated with adverse outcomes and increased mortality [1, 9, 13]. Although arteriosclerosis is common in old dogs and cats, and is typically mild and usually unimportant to the health and survivals, anemia may promote oxygen deprivation of myocardial cells due to the loss of arterial blood supply by arteriosclerosis of coronary arteries. However, the exact mechanism of arteriosclerosis in this case is unknown.

This case could not be diagnosed as myocardial infarction by the medical examinations. Although the diagnosis of myocardial infarction is considered in dogs that acutely develop brady- or tachy-arrhythmias and/or ST segment deviation not attributable to other conditions, particularly when a predisposing condition such as infective endocarditis is present, the criteria of diagnosis of myocardial infarction is not established yet because myocardial infarction is extremely rare events. Also, clinical diagnosis of arteriosclerosis is difficult and early diagnosis is not usually possible, although ECG abnormalities of arteriosclerosis include atrial fibrillation, notched QRS complexes, and ST segment elevation.

Myocardial infarction and arteriosclerosis are scarcely any reports of these lesions in cats. This case is valuable for a extremely rare case of myocardial infarction in the cat and raising the establishment of the diagnostic criteria of myocardial infarction for more appropriate treatments.

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