犬における微小心筋梗塞の1例

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Myocardial Microinfarction in a Dog

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ABSTRACT. A 14-year-old female dog of Ainu breed was admitted to a veterinary hospital. It showed signs of congestive heart failure with accompanying chronic interstitial nephritis. In the pathological examination, a focal microinfarct was present in the myocardium and it was surrounded by an area of fibrosis in the left ventricular wall. Before death, there were no findings clearly suggesting the presence of this type of lesion obtained by electrocardiographic examination and serum analysis, but marked increase in CPK-MB/ total CPK activity was observed.—KEY WORDS: CPK-MB, dog, myocardial microinfarct.

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There are many reports on intramural myocardial necrosis and fibrosis in the dog [5–9, 12–14, 16, 19, 20]. Most of have been related to chronic valvular disease such as mitral insufficiency (MI) [5–7, 9, 13, 14, 16, 19], but some have been noted in congenital subaortic or pulmonary stenosis [7, 8, 16, 20]. It has been stated that this condition can aggravate the heart already suffering from valvular endocardiosis [7, 12, 19]. Such a condition, however, is not generally detected until postmortem examination. In Japan, there have been no clinical reports on this type of disease. The present paper describes the clinical data and pathological findings in a case of canine myocardial microinfarction with cardiac hypertrophy associated with chronic interstitial nephritis (CIN).

A 14-year-old spayed Ainu dog weighing 16.5 kg was referred to Veterinary Hospital, Hokkaido University because of weakness, anorexia, nausea and shortness of breath. The owner reported that she had been showed these clinical signs for about a month.

At the time of admittance, the dog was depressed and the abdomen was distended with ascites. Palpation revealed atrophied and rough surfaced kidneys. The body temperature was 38.7°C and the heart rate 160 beats/minute. Weakness of the heart sound was auscultated by a stethoscope, but no cardiac murmur was heard. Radiographs of the thorax demonstrated generalized enlargement of the cardiac silhouette. By echocardiography, a large amount of pericardial effusion, thickened left ventricular wall and interventricular septum and enlarged right ventricular chamber were noted (Fig. 1). An electrocardiographic examination with leads I, II, III, aVR, aVL and aVF revealed tracings resembling right bundle branch block (RBBB) (Fig. 2). Phonocardiograms revealed an accentuated forth heart sound. The hematological examinations showed mild leucocytosis (17,000/mm^3), a low plasma protein level (5.4 g/d*l*) and a slight increase in BUN (25.9 mg/d*l*) and Creatinine (2.16 mg/d*l*) levels. Values of serum GOT, GPT, LDH and CPK were in the normal range, but the ratio of CPK-MB activity to total CPK activity was markedly increased (Table 1). Congestive heart failure associated with CIN was diagnosed from these findings.

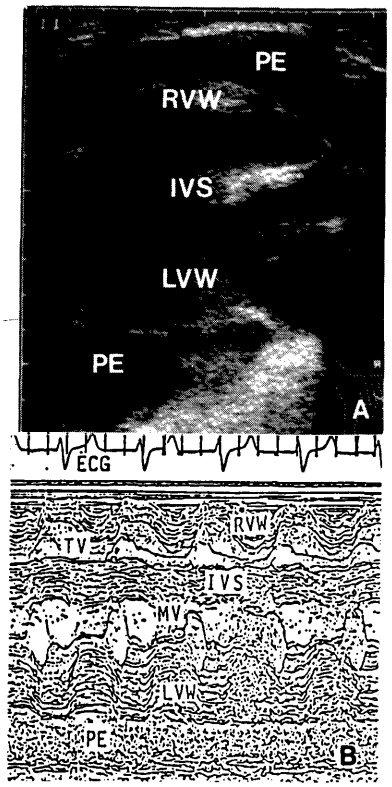

Fig. 1. (A): Two-dimensional echocardiogram. Pericardial effusion (PE) can be seen above the anterior right ventricular wall (RVW) and behind the posterior left ventricular wall (LVW). (B): M-mode echocardiogram. Note the markedly increased thickness of the septum (IVS) and the posterior left ventricular wall (LVW). The anterior right ventricular wall (RVW) is mildly thickened and the right ventricular cavity (RV) is dilated.

Table 1. Serum biochemical data

Item	Clinical course on day:			
	1	10	13	16
GOT (IU/*l*)	33.2	–	–	31.7
GPT (IU/*l*)	45.6	97.2	17.7	23.9
LDH (IU/*l*)	112.6	147.2	141.7	120.2
CPK (IU/*l*)	67.2	20.8	–	18.8
CPK-MM (%)	–	37.2	–	36.4
CPK-MB (%)	–	42.8	–	44.9
CPK-BB (%)	–	20.0	–	18.7
Na (mEq/*l*)	145.0	128.1	146.6	–
K (mEq/*l*)	5.0	4.6	3.1	–
Ca (mg/d*l*)	9.3	–	–	–

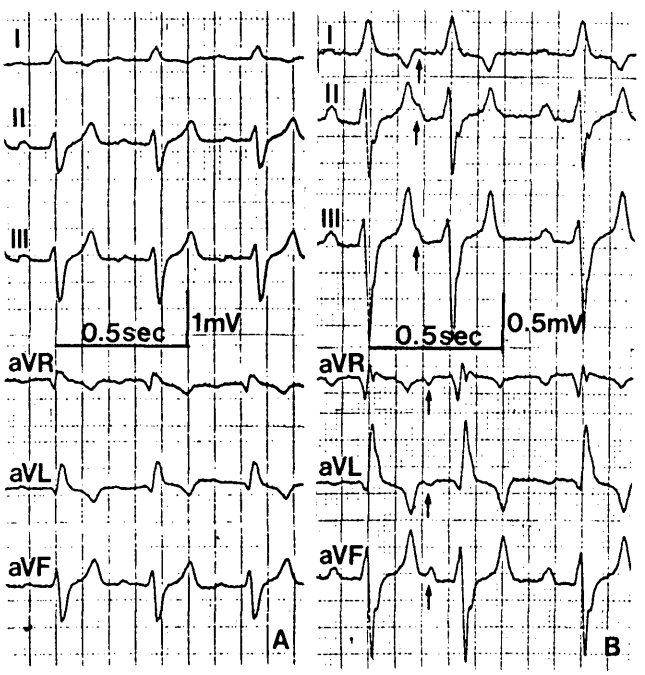

Fig. 2. (A): Electrocardiograms recorded at admittance, showing the pattern resembling right bundle branch block (RBBB). (B): In addition to RBBB, atrial premature beats (arrow) were also detected on the day of euthanasia.

The patient was treated with antibiotics, cardiotonics and diuretics. Peritoneal dialyses were made because of aggravated azotemia. Her condition, however, gradually worsened and she showed severe emaciation, depression and vomitting. The dog was euthanatized 3 weeks after admittance.

By gross examination, shrunken kidneys with rough surfaces were found. In the heart concentric hypertrophy of the left ventricle, eccentric hypertrophy of the right ventricle and dilated right atrium were observed. Moreover, many focal lesions (2 to 7 mm in diameter) of greyish-whitish in colour were

Fig. 3. Left ventricular myocardium. Myocardial fibrosis lesion. Elastica van Gieson stain. ×60.

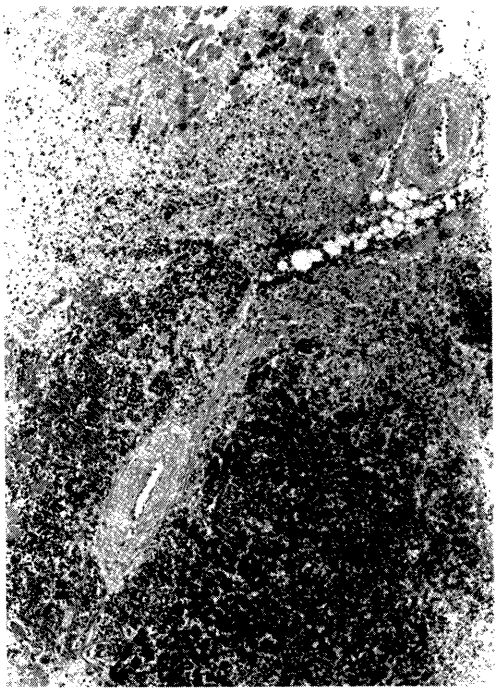

Fig. 4. Anterior papillary muscle. Myocardial microinfarct. Notice the intimal thickening and edematous and/or fibrous swelling of the wall of the arteries. HE stain. ×60.

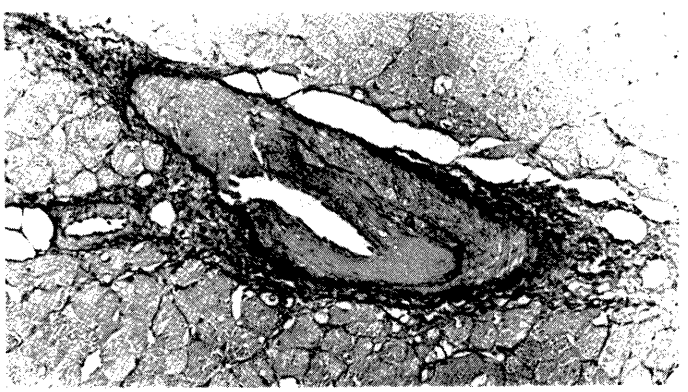

Fig. 5. Intramural coronary artery. Intimal hyaline deposits. Elastica van Gieson stain. ×150.

found throughout the left ventricular wall, and a reddish-brown patchy lesion (10× 7 mm) was apparent in the anterior papillary muscle. However, the atrioventricular valves appeared normal.

Microscopically, the greyish-whitish lesions in the left ventricular wall were of multifocal myocardial fibrosis (Fig. 3). Degenerative muscle fibers were sometimes seen within or adjacent to the fibrosis lesions. The reddish-brown lesion in the anterior papillary muscle consisted of fresh

necrosis of an area of muscle fibers, which had been atrophied, and hemorrhage in the interstitium. The lesion was considered to be an intramural myocardial microinfarct (Fig. 4).

Intramural coronary arteries throughout the left ventricle showed sclerotic changes characterized by intimal thickening due to proliferation of smooth muscle and elastic fiber, leading to severe narrowing of the vascular lumen (Fig. 4). Deposits of hyaline materials were occasionally observed in the thickened intima (Fig. 5). In addition to these changes, there were edematous and/or fibrous swelling of the walls and occasional acute septic arteritis.

There were no conspicuous changes in the regions of the right bundle branch and the sino-atrial node.

The renal lesions were characterized by marked fibrosis, loss of tubules, foci of mononuclear infiltration and hyaline substance deposits on the arterial walls.

In dogs with MI or congenital subaortic stenosis, small foci of myocardial necrosis and fibrosis were reported to be localized in the left ventricular wall and were most often seen in the anterior papillary muscle [5, 7, 8, 13, 14, 16, 19]. Regarding these myocardial lesions, arteriosclerotic narrowing of the lumen was observed in the small intramyocardial coronary arteries. The intramural myocardial infarct was considered due to ischemia brought about by reduced blood flow through the stenosed arteries [5–7, 12–14, 19].

The intramural myocaridal infarct and fibrosis in the present case were considered to be qualitatively similar to those of the previous reports [5–9, 12–14, 16, 19, 20] on the basis of the size, location and coexistence with sclerotic changes of intramural coronary arteries, but it is unique in that the myocardial lesions of this case were not associated with chronic mitral valvular disease with MI.

The incidence of coronary arterial sclerotic lesions increases with advancing age in dogs [13, 14]. In dogs suffering from CIN, hyaline deposits were found in the intrarenal arteries and occasionally in the intramyocardial branches of the coronary arteries [1, 2, 18]. Therefore, the intimal thickening with proliferation of smooth muscles and elastic fibers may have already been in existence, with no relation to CIN. But hyaline deposits in the thickened intima seem to be secondary to CIN, facilitating reduction of blood flow through the coronary arteries. These findings suggest, therefore, that the myocardial microinfarct resulted from relative underperfusion of the hypertrophied myocardium secondary to CIN.

Electrocardiographically, the arrhythmias and conduction disturbances observed in the dogs with chronic valvular disease with arterial sclerosis and myocardial disease (necrosis and fibrosis) were ventricular premature beats, atrial premature beats, atrial fibrillation, atrial flutter and left and right bundle branch block [5, 6, 16]. Ventricular extrasystole was also observed in a dog with congenital subaortc stenosis with similar lesions [16]. In the present case, electrocardiogram recordings were carried out upon admittance and on the day of euthanasia. As shown in Fig. 2, the first examination revealed a pattern resembling RBBB. In addition to this finding, atrial premature beats were also recognized at the second examination.

Patterns of RBBB have been observed in dogs with various kinds of cardiac disease including right ventricular dilatation [3, 4, 6, 11, 16], and may also occur in normal dogs [3, 5, 16]. So it is not always associated with a certain cardiac disease. Moreover, atrial premature beats occur most commonly in dogs with MI, resulting in atrial strain which causes edema and mononuclear infiltration [16]. In the present case, the dilatation of

right ventricle and right atrium was also observed. It is, therefore, considered that the electrocardiographic abnormalities in the present case have no direct connection with the myocardial disease in the left ventricular wall.

The present electrocardiogram in the limb leads did not demonstrate such characteristic changes in the wave pattern as appearance of the Q or QS wave, deflection of the S-T segment and changes in the T wave pattern, which are commonly observed with myocardial infarct in man. There are two possible explanations: First, spontaneous myocardial microinfarcts in the dog produced only vague clinical signs, so that well-timed examinations were very difficult to perform. Second, the size of infarct was smaller than those in man. In the present case, there was also the problem of obtaining abundant information, because the lead systems used in electrocardiography were limited to extremity leads. It is, however, possible that dogs may not exhibit electrocardiographic patterns similar to those obtained in man. In this regard, Pensinger [17] reports that when the normal dog's coronary arteries are experimentally ligated after preparatory ischemia, there may not be serious electrocardiographic disturbances or ventricular fibrillation.

There have been no reports on changes of serum enzyme activity to aid diagnosis of spontaneous myocardial infarct in the dog, although studies in man have revealed such changes as increase of serum GOT, LDH and CPK activity [10, 15]. In the present case, the total activities in serum GOT, LDH and CPK revealed no remarkable changes with the exception of the marked increase in the ratio of CPK-MB activity to total CPK activity. In man, the activities of GOT and CPK in serum show a rapid rise with a return to nomal levels within a short time [10, 15]. Also, there is a positive correlation between the magnitude of change in CPK, especially CPK-MB, activity and the extent of infarct size [15]. The infarct in a dog is generally substantially smaller in size than that in man. For this reason, myocardial microinfarct may have no effect on changes in the activities of serum enzymes. However, with the exception of this case, the authors have not previously observed a marked increase in CPK-MB in routine blood biochemical analyses of dogs with various diseases, and there has been no report on this change as far as we know. It is considered that the elevated CPK-MB value may suggest the existence and/or occurrence of myocardial microinfarct.

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要　　約

犬における微小心筋梗塞の1例：町田　登・山我義則・安田　準・戸尾祺明彦（北海道大学獣医学部附属家畜病院）——慢性間質性腎炎にともなううっ血性心不全を示した14歳・雌のアイヌ犬に，心電図検査ならびに血清検査を実施した結果，心筋病変の存在を明らかに示唆する所見は得られなかったが，CPK-MB の割合の著しい増加が見られた。病理学的検索では左心室壁に梗塞巣および巣状心筋線維化が認められた。