Congestive Heart Failure due to Bilateral Atrioventricular Valve Insufficiency in Two Dogs

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(Received 26 July 1995/Accepted 30 October 1995)

ABSTRACT. Congestive heart failure resulted from bilateral atrioventricular insufficiency was diagnosed in two, small breed, intact male aged dogs. In both cases, the clinical signs were mainly associated with right heart failure, while those related to left heart failure were seldom observed. Radiographic and echocardiographic examinations revealed the dilatation of the right heart due to tricuspid valve insufficiency. Mitral regurgitation was also observed without pulmonary hypertension. Both patients responded to medications of diuretics, methyl digoxin and angiotensin converting enzyme inhibitor. As congestive signs deteriorated, they became unresponsive to the treatment. Ultimately, the dogs died 9 and 13 months later from the first admission. — **KEY WORDS:** canine, heart failure, tricuspid valve insufficiency.

J. Vet. Med. Sci. 58(4): 381–384, 1996

Atrioventricular valve insufficiency (AVI) is one of the most common heart disease of aged dog of small breed. Especially mitral valve insufficiency (MVI), the major disorder of AVI, has been reported to occur preferentially in males [2]. In severe MVI, left heart failure induces pulmonary hypertension and causes right ventricular enlargement and subsequent tricuspid valve insufficiency (TVI) [3]. Thus, bilateral AVI (BAVI) is regarded as one of the end-stage symptom of MVI. In dogs with BAVI, the clinical signs associated with severe MVI and pulmonary hypertension should be observed before TVI developed in the disease process. The present paper describes right congestive heart failure due to BAVI without the obvious clinical evidences of MVI and pulmonary hypertension.

Case 1: An intact male, 15 years old, Yorkshire terrier, 4.0 kg of body weight, was presented to the Veterinary Medical Teaching Hospital of Nippon Veterinary and Animal Science University (VMTH-NVASU), with abdominal distension, anorexia, lethargy, and occasional vomiting and diarrhea lasting for 2 weeks. On admission, physical examination revealed the ascites, hepatomegaly, edema around the hind limb hock, decreased femoral pulse, bilateral cataract, gingivitis due to the accumulation of odontolith. The grade 3 and 4 systolic regurgitant murmurs were heard at the mitral and tricuspid valve areas, respectively. The lung sound was normal. Upon laboratory examinations including urinalysis, complete blood count (CBC) and serum chemistry, bacterial cystitis, mild anemia and leukocytosis with stress pattern were noted. Microfilaria was not detected in the venous blood sample. Radiographic examination revealed the severe dilatation of the heart (Fig. 1). Electrocardiogram (ECG) revealed normal sinus rhythm with notched QRS complex and the occasional intervention of atrioventricular nodal extracontraction (Fig. 2). Echocardiogram (Fig. 3) confirmed dilated cardiac chambers, especially the marked dilatation of the right atrium, since the projection of the atrial septum into left side was observed. The atrioventricular valves, especially septal leaflet of the tricuspid valve, were thickened. The

regurgitations of the mitral and tricuspid valves were detected, and other morphological abnormality was not confirmed. There was no heartworm in the right heart and main pulmonary artery. The diameter ratio of the main pulmonary artery and aorta was normal. The paradoxical motion of the ventricular septum was not observed, and the papillary muscle had a normal morphology.

Based on these results, the dog was diagnosed to have BAVI. Oral furosemide (4 mg/kg/day), methyl digoxin (0.042 mg/head/day) and enalapril (2 mg/kg/day) were initiated. As the result, the congestive signs and exercise tolerance were significantly improved, and the dog was able to run around willingly. On ECG, notched QRS complex and the extracontraction were cleared. Eight months after the first admission, decreased exercise-tolerance and weakness appeared again, and the recurrence of atrioventricular nodal extracontraction was confirmed. Medication of methyl digoxin was discontinued, but the arrhythmia persisted. The dog died 9 month after the first examination. The owner reported that the dog seemed to have died during afternoon nap. Coughing, one of the common signs of left sided heart failure, has seldom been observed in disease process.

Case 2: An intact male, 9 years old, mixed breed of terrier, 4.6 kg of body weight, was found to have been having cardiac murmur for 1 year before admission by the referring veterinarian. The patient was left untreated. The owner had not noticed any abnormal signs associated with the murmur and thus the dog was referred to VMTH-NVASU, because abdominal distension was refractory to furosemide and digoxin medication. Initial physical examination revealed the ascites, cardiac murmur and increased vesicular sound upon auscultation. The murmur was pansystolic and the most intense (grade 5) at the mitral and tricuspid valve areas. The serum chemistry revealed increased ALP and ALT activities and mild azotemia, but CBC and creatinine concentration were normal. These findings would reflected the congestion of the liver and pre-renal azotemia. Microfilaria was not detected from the

Fig. 1. The cases 1 (upper) and 2 (lower) radiograms. Left panels are dorsoventral and right are lateral views.

Fig. 2. The electrocardiograms (ECGs) of bilateral atrioventricular valve insufficiency in cases 1 and 2. Upper ECG was obtained at the first admission of the case 1. Notched QRS complexes are increased amplitude (R wave, 3.61 mV), and QRS duration is prolonged (0.06 sec.), indicating left ventricular enlargement. Arrows indicate atrioventricular nodal extracontraction. Lower ECG was recorded from case 2 when congestive signs had deteriorated. Prolonged P-Q interval (0.153 sec.) and deep Q wave (1.1 mV) indicate first degree atrioventricular block and right ventricular enlargement. The left deviation of the mean electrical axis (38°) was also observed.

Fig. 3. Two-dimension echocardiogram of atrioventricular valve insufficiency in the case 1. Marked dilation of the cardiac chambers was noted. RA ; right atrium, RV ; right ventricle, LA; left atrium, LV ; left ventricle.

peripheral blood. On the radiographic examination, all the cardiac chambers significantly dilated and pulmonary edema was not observed (Fig. 1). ECG findings included normal sinus rhythm and left axis deviation (38° on the frontal plane). Echocardiographic findings were similar to those of the case 1, and the dilatation of the right ventricle was more evident than that of the case 1. Regurgitations at both atrioventricular valves were detected, although heartworm was not observed.

Based on the diagnosis of congestive heart failure originated from BAVI, oral furosemide (4 mg/kg/day), spironolactone (2 mg/kg/day), methyl digoxin (0.042 mg/head/day) and alacepril (2 mg/kg/day) were initiated. Hyponatremic diet was also prescribed. Upon improvement in congestive signs, the doses of furosemide and spironolactone were reduced by half. During the first 7 months after initial admission, the congestive signs were controlled. One month after that period, the accumulation of the ascites became evident, and respiration and appetite were compromised. First degree atrioventricular block was observed on ECG (Fig. 2). Then, methyl digoxin was discontinued for two weeks, but failed to improve the conduction disturbance. Thus, the ascites and pleural fluid were regularly removed by the echo-guided centesis. The fluid was modified transudate. Ultimately, the dog was died 13 months after the first admission. As with the case 1, coughing has seldom been observed in the disease process.

In both cases, necropsies could not be done.

Although the present cases had BAVI, findings such as the ascites, hepatomegaly, edema and cardiac systolic murmur would mainly result from right heart failure due to TVI. In general, the causes of TVI were divided into primary and secondary TVIs. The former was associated with the myxomatous degeneration of the tricuspid valve as with MVI, while the latter was related to pulmonary hypertension due to heartworm disease, pulmonary thromboembolism and any disorder leading to left heart failure such as MVI; and idiopathic hypertension, congenital pulmonary stenosis, endocarditis, and dilated cardiomyopathy [3]. In the present cases, the secondary causes except disorders causing left heart disease could be excluded based on physical, laboratory, radiographic and echocardiographic findings.

MVI is the most common acquired heart disease in the small aged dogs. In advanced MVI, left heart failure could lead to pulmonary hypertension and subsequent TVI [3]. Thus, clinical findings related to left heart failure can be observed with the history of BAVI. Indeed, the prevalence of canine AVI in autopsied sample had been reported as: the mitral valve alone, 62%; mitral and tricuspid valves, 32.5%; and tricuspid valve alone, 1.3% [4]. Therefore, it might be expected that BAVI is the common cardiac diseases in a dog. In the present cases, the clinical signs related to severe MVI were seldom observed, and further the pulmonary hypertension secondary to MVI as the cause of TVI can be excluded, because pulmonary hypertension enough to produce right ventricular enlargement and

resultant TVI should be caused by much severer MVI with clinically apparent signs. It had been reported that tricuspid regurgitation (TR) develops most often when the mean pulmonary arterial pressure exceeds 60 mmHg in humans [1], and relatively modest elevations of pulmonary arterial pressure in dog with advanced MVI are unlikely to result in TR [6]. Although an additional examination to detect pulmonary hypertension was not performed, radiographic and echocardiographic findings were not suggestive to pulmonary hypertension. Therefore, myxomatous degeneration of the tricuspid valve may be responsible for TVI.

Although it had been reported that TVI occurs in about 50% in normal dogs [7], the clinical prevalences of congestive heart failure due to TVI alone and BAVI are not well identified [3]. Present cases were uncommon forms of BAVI without clinical findings associated with MVI. In general, myxomatous degeneration of the tricuspid valve develops in a way identical to that of the mitral valve, and usually progresses more slowly [3, 4]. In our cases, the clinical manifestations indicate that myxomatous degeneration of the tricuspid valve could develop more rapidly and more severely than that of the mitral valve. If it were the case, decrease in the cardiac output of the right ventricle due to TVI should decrease the preload to the left ventricle, reducing the clinical signs associated with MVI. This hypothesis can be a reasonable explanation that the present cases did not show clinical signs related to MVI.

Supraventricular arrhythmias were observed in both cases. In general, these arrhythmias relate to the atrial dilation due to AVI, hypokalemia, lesions such as right atrial hemangiosarcoma, and digitalis intoxication [5]. Serum potassium levels were consistently normal and digitalis intoxication could be excluded in our cases, because the arrhythmias did persist even after the discontinuance of methyl digoxin administration. Therefore, these arrhythmia might be related to the atrial dilatation due to cardiac volume overload.

In conclusion, the common features of BAVI in the present cases could be summarized as follows: 1) the dogs were small, intact male and aged; these signalments were identical to those of MVI; 2) clinical signs due to right heart failure were predominantly observed, while those related to left heart failure were seldom noted; 3) supraventricular arrhythmias were observed in the disease process; and 4) long term prognosis was thought to be poor or guarded.

Authors would thank Dr. Katsushi SUZUKI (Department of Veterinary Physiology, Nippon Veterinary and Animal Science University) for his valuable suggestion and critical reading of this paper.

REFERENCES

1. Braunwald, E. 1969. *New Engl. J. Med.* 281: 425-433.
2. Buchanan, J. W. 1979. *Adv. Vet. Sci. Comp. Med.* 21: 75-106.
3. O'Grady, M.R. 1995. pp. 944-959. *Textbook of Veterinary*

- Internal Medicine, 4th ed. (Ettinger, S.J. and Feldman, E.C. eds.), W.B. Saunders, Philadelphia.
4. Sisson, D. 1987. pp. 59–116. Contemporary Issues in Small Animal Practice, vol. 7: Cardiology (Bonagura, J.W. ed.), Churchill Livingstone, New York.
 5. Tilley, L. P. 1992. Essential of Canine and Feline Electrocardiography, 3rd eds., Lea & Febiger, Philadelphia.
 6. Wallace, C. R. and Hamilton, W. F. 1962. *Circ. Res.* 11: 301–314.
 7. Yuill, C. D. M. and O'Grady, M. R. 1991. *Can. J. Vet. Res.* 55: 185–192.