NOTE ***Surgery***

Surgical Correction of Subvalvular Aortic Stenosis Using Cardiopulmonary Bypass in a Dog

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ABSTRACT. A three-month-old male Golden Retriever had symptoms including exercise intolerance, dyspnea and syncope and was diagnosed with subvalvular aortic stenosis. Cardiac catheterization revealed a left ventricular-aortic systolic pressure gradient o f 90 mm Hg. Surgical correction of the condition was achieved using cardiopulmonary bypass. The subvalvular fibrous lesion was resected through an aortotomy. The stenosis was dilated from 8.5 mm to 12.0 mm in diameter. Postoperatively the dog was asymptomatic. Seven months after surgery, the pressure gradient decreased to 44 mm Hg. However, after another three months, the dog died suddenly without any premonitory signs. Postmortem examination revealed that pathologic changes caused by increased left ventricular pressure overlo ad were not severe.

KEY WORDS: cardiopulmonary bypass, open resection, subvalvular aortic stenosis.

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Aortic stenosis (AS) is one of the most common congenital cardiac malformations in dogs. Of the three forms of AS —supravalvular, valvular and subvalvular forms—subvalvular aortic stenosis (SAS) is the most often form observed in dogs. In SAS, left ventricular outflow tract obstruction leads to increase in left ventricular pressure overload and hypertrophy of the left ventricular myocardium. Severely affected dogs may show signs of exercise intolerance, syncope and sudden cardiac death. Since severe SAS is difficult to treat by drugs, surgical correction has been recommended. Several techniques for surgical correction of SAS have been used in dogs [2, 5, 8]. In veterinary medicine, the development of cardiopulmonary bypass (CPB) facilitates cardiotomy [7, 9, 11]. It is thought that open resection of the lesion is the most effective treatment. However, no improvement in survival of dogs following open heart surgery for SAS was reported [10].

A three-month-old male Golden Retriever, with limited mobility, was diagnosed with SAS in a veterinary hospital. Medical therapy was instituted with isosorbide dinitrate and dipyridamole, but the condition worsened. The owner requested surgical intervention and the dog was referred to the Veterinary Medical Teaching Hospital, Tokyo University of Agriculture and Technology for surgical correction of SAS.

The dog weighed 12.0 kg, with apparently normal growth. Clinical signs of exercise intolerance, dyspnea and syncope were reported. Auscultation identified a grade V~VI\VI systolic murmur with its point of maximum intensity in the region of the aortic valve. Heart rate and rhythm were normal but femoral pulses were weak. Blood examination showed no abnormal findings except for a moderate anemia (PCV of 27%). Thoracic radiography revealed enlargement of cardiac silhouette [Cardiothoracic ratio (CTR); 58.5%, Vertebral heart score (VHS); 11.5V] and dilatation of aortic arch, resulting in loss of the cranial waist and widening of the mediastinum and right tracheal deviation. Electrocardiography showed normal sinus rhythm, 180 beat/min, left axis deviation (mean electric axis of +30°), increased R-wave amplitudes (2.7 mV), extended QRS duration (0.06 sec) and depression of the ST segment (Fig. 1).

Two weeks after, no anemia was observed. Selective left/right cardiac catheterization was performed. The left ventricular-aortic systolic pressure gradient was 90 mmHg and left ventricular pressure was elevated (Table 1). Left ventricular angiocardiography showed discrete SAS, poststenotic dilatation of the ascending aorta and mild mitral regurgitation (Fig. 2-a). Right ventricular angiocardiography revealed no apparent abnormalities. Based on the above findings, the diagnosis was confirmed as severe discrete SAS.

Surgical correction of SAS using CPB was conducted. A non-blood priming CPB for open heart surgery was performed using a heart-lung machine for animal use (NAPS-III MERA: Animal Clinical Research Foundation Type, Mera, Tokyo, Japan). The dog was premedicated with atro-

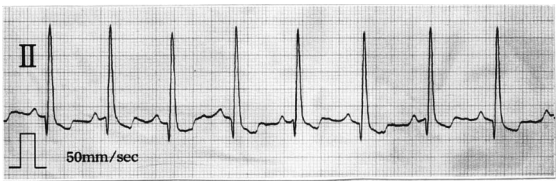

Fig. 1. Electrocardiography (50 mm/sec) showing increased R-wave amplitudes (2.7 mV), extended QRS duration (0.06 sec) and depression of the ST segment.

Table 1. Intracardiac pressure and Blood gas partial pressure values

	Preoperative				Postoperative 203 days
	Blood gas partial pressure*(torr)			Blood pressure**(mmHg)	Blood pressure**(mmHg)
	SAO_2	PO_2	PCO_2		
Left ventricle	99.8	401.5	32.8	204 / –20	134 / –5
Aorta				114 / 94	90 / 64
Pulmonary arterial wedge pressure				9 / 8	
Pulmonary artery	89.1	60.8	53	15 / 12	
Right ventricule	83.6	54.3	47.5	17 / 7	
Right atrium	91.1	65.6	44.2	3 / 2	

* under 100% O_2 inhalation
** systolic pressure / diastolic pressure

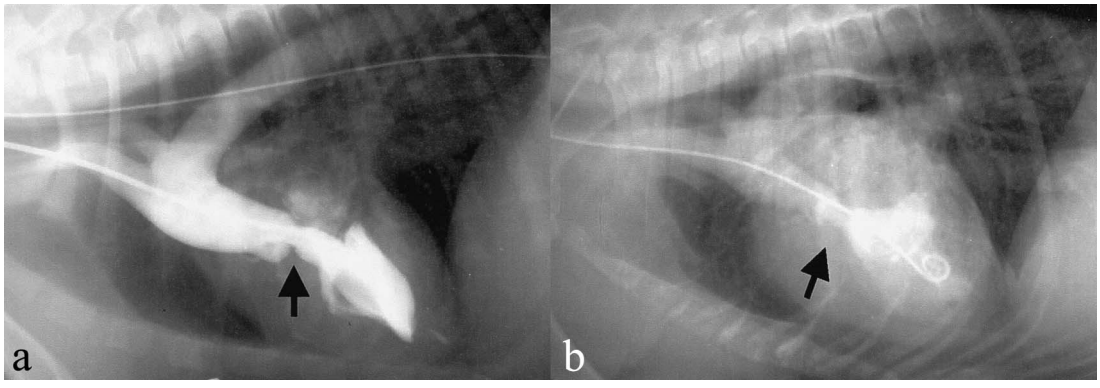

Fig. 2-a. Preoperative left ventricular angiocardiography showing subvalvular aortic stenosis (arrow), poststenotic dilatation of the ascending aorta and mild mitral regurgitation.
Fig. 2-b. Postoperative left ventricular angiocardiography showing dilation of subvalvular stenotic area (arrow).

pine sulphate (0.04 mg/kg subcutaneously) and acetylpromazine maleate (0.3 mg/kg intramuscularly). Anesthesia was induced by intravenous administration of thiamylal sodium (10 mg/kg), after which the animal was intubated. Anesthesia was maintained with a combination of isoflurane in oxygen and 0.1% ketamine hydrochloride micromini-drip administration; suxamethonium chloride was administered intermittently for muscle relaxation and respiration was controlled by intermittent positive pressure ventilation.

An electrocardiogram, arterial pressure, central venous pressure, end-tidal CO_2, arterial oxygen saturation, esophageal and rectal temperature and urine volume were measured continuously during surgery. Arterial and venous blood gas partial pressures, activated clotting time and sodium and potassium concentrations were monitored during surgery. A median sternotomy was performed and a pericardial cradle was created. After heparinization (100 U/kg intravenously), two cannulas were placed, one into the cranial and one into the caudal vena cava through the right atrial appendage and the right atrium respectively, while a blood return cannula was inserted into the right femoral artery. CPB was initiated and partial perfusion started. The hemodynamic functions of the animal were stabilized and total perfusion was initiated. The ascending aorta and pulmonary artery were cross-clamped with a vascular clamp proximal to the brachiocephalic trunk. Cardioplegia solution and myocardial protectant solution were administered antegrade via a cannula placed in the aortic root. A retrograde coronary sinus perfusion (RCSP) cannula was placed into the coronary sinus transatrially; myocardial protectant solution was administered retrogradely via the RCSP cannula during open heart surgery. An aortotomy incision was made above the coronary ostium to expose the lesion (Fig. 3). Portions of the subvalvular fibrous tissue were excised with a surgical blade (Feather scalpel No. 11, Feather Safety Razor, Osaka, Japan), avoiding damage to the aortic valve leaflets and the anterior leaflet of the mitral valve. Resection was continued around the circumference until excision was completed. The stenosis was dilated from 8.5 mm to 12.0 mm in diameter. Air was evacuated from the heart and aortic arch and the aortotomy incision was closed using 5–0 polypropylene. The RCSP cannula was extracted and the atriotomy incision was closed. The aortic cross-clamp was removed. Electrical defibrillation was used to restore the sinus rhythm. Lidocaine hydrochloride was administered as needed to help maintain a sinus rhythm and dopamine hydrochloride was given for inotropic and pressure support. Gradual cessation of the CPB was initiated while the dog's normal cardiac output was restored. CPB cannulas were removed, a chest drain tube was placed and the sternotomy

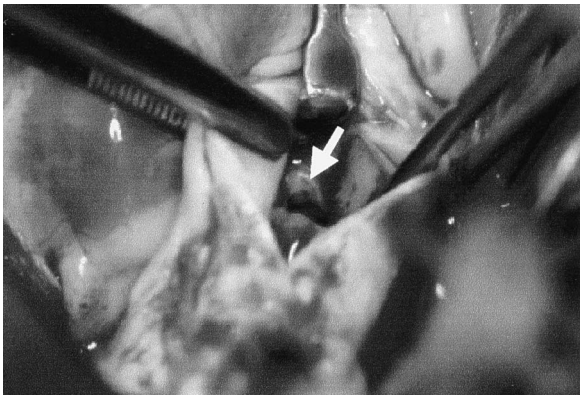

Fig. 3. The subvalvular fibrous lesion is exposed through an aortotomy (arrow).

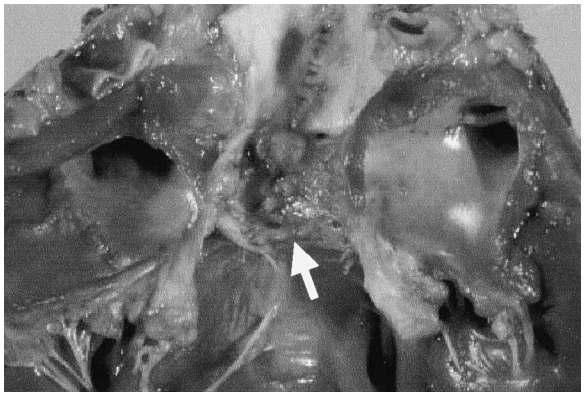

Fig. 4. A residual subvalvular fibrous ridge (arrow) and a thickened cusp of the anterior mitral valve leaflet are present.

was closed. CPB time and aortic cross-clamp time were 201 min and 62 min, respectively.

Postoperatively, dopamine and lidocaine infusions were used to stabilize the hemodynamics. Ventricular premature contraction developed for 3 days after surgery. Following removal of endotracheal tube, oxygen was administered via a nasal catheter. Thoracic drainage was maintained for 24 hr. Oral digitalization was started 2 days after surgery. Fresh whole blood was administered for correction of anemia 4 days after the operation. Twenty-three days after, the dog was assessed to be in good condition and was subsequently discharged. Oral digitalization was continued (0.01 mg/kg SID).

The dog was re-examined 203 days after surgery. The dog weighed 26.2 kg and did not exhibit any abnormal clinical signs. The cardiac murmur had decreased to grade II/VI and blood examination revealed no abnormalities. Thoracic radiography revealed mild left ventricular enlargement on the dorsoventral view and few findings except for growth change on the lateral view. [Cardiothoracic ratio (CTR); 65.3%, Vertebral heart score (VHS); 10.6V] Electrocardiography revealed no ventricular premature contraction and indicated a mean electric axis of +45°. On selective left cardiac catheterization, the pressure gradient had decreased to 44 mm Hg. Left ventricular angiocardiography showed an increased diameter of left ventricular outflow tract (Fig. 2-b).

The dog continued to show no abnormalities. However, the dog died suddenly 292 days after the operation and was necropsied. The opened left ventricle was hypertrophied. A residual subvalvular fibrous ridge and a thickened cusp of the anterior mitral valve leaflet could also be seen (Fig. 4). Histopathological examination of the heart revealed the following findings: focal and macular myocardial necrosis/fibrosis in the interventricular septum [especially the superior septum] and left ventricular posterior wall with calcification, which were by far more slight than those in other dogs with SAS. Myocardial cells had normal thickness while the interstitial myocardium was rough, atrophied and edematous. Intramural coronary arteriosclerosis was present with arterial lumen stenosis due to intimal cellular/fibrous thickening and medial collagen fibroplasia. Perivascular interstitial calcification was found. No lesions were observed in the conduction system. All other organs were found to be normal.

No official survey on the frequency of canine SAS has been reported in Japan. The Golden Retriever is the most popular canine large breed in Japan and is known to be the breed most susceptible to SAS [1, 3]. Thus, SAS is encountered most frequently in the Golden Retriever breed in Japan. However, surgical correction of SAS using CPB and follow-up has not been reported in Japan, although it has been frequently done abroad.

A retrospective analysis of 195 confirmed cases of SAS was conducted to determine the clinical course in a group of untreated dogs and to examine the relationship between the severity of the obstruction and the resulting clinical course [6]. It was found that dogs with mild SAS (pressure gradient < 35 mmHg) lived longer and tended to remain asymptomatic, while the majority of dogs with severe SAS (pressure gradient > 80 mmHg) tended to have poor prognosis, had a high prevalence of sudden cardiac death and died before three years of age. It is thought that surgical correction of SAS must be performed in severely affected pups as soon as possible.

In the present case, the dog was severely affected with SAS and had clinical signs including exercise intolerance, dyspnea and syncope. Thus, surgical correction was deemed to be necessary. During surgery, anesthesia was maintained safely and hemodynamic functions were stabilized by monitoring. Retrograde administration of myocardial protectant solution enabled safe cardiac arrest and smooth recovery of cardiac rhythm after reperfusion. Although partial ventricular septum myectomy for SAS may be performed concurrently with open resection of the subvalvular fibrous tissue, this was not necessary in the present case. During weaning from CPB and postoperatively, premature ventricular contractions, hypotension and

anemia were observed to be main complications. These were expected complications. Many studies on open heart surgery for SAS report moderate to good reduction in the pressure gradient and improvement of clinical signs [7, 9, 11]. However, in a retrospective study comparing the outcome and intermediate-term survival of dogs that underwent open heart surgical correction of SAS with those that did not undergo surgery, symptomatic treatment did not increase the survival rate of dogs that underwent surgery for SAS. Pathologic changes in the left ventricle, including concentric hypertrophy, myocardial and endocardial fibrosis/calcification and neointimal-medial coronary arterial hyperplasia, might have predisposed the dogs with SAS to sudden cardiac death. It seems likely that such pathologic changes were already present in the dogs at the time of surgery, and that these were not corrected by surgery [10].

In the present case, despite reduction of the pressure gradient and improvement in quality of life, the dog that underwent surgery died suddenly without any prior abnormal signs. However, postmortem examination revealed that pathologic changes caused by increased left ventricular pressure overload were not severe.

Although the exact mechanism of sudden cardiac death in dogs with severe SAS remains a mystery, it appears that pressure overload must be alleviated before the myocardium is damaged irreversibly. There may be possible benefits associated with administration of atenolol to dogs severely affected with SAS [1,10]. The effect of treatment with β-receptor blockers in dogs with SAS should be investigated.

During open heart surgery for SAS, not only complications of CPB should be considered but also care should be taken to avoid iatrogenic surgical injury, especially mitral and aortic valve injuries. These recurrence problems also have to be addressed. To avoid these problems, a valved apico-aortic conduit has been attempted without repairing the lesion [2]. Surgical relief of the left ventricular outflow obstruction may be difficult to achieve by conventional resection. In the future, there is a need to develop less invasive and improved surgical procedures for the management of SAS.

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