Balloon valvuloplasty for severe subaortic stenosis in a Pomeranian dog

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Abstract: A nine-month-old Pomeranian dog with exercise intolerance and syncope was presented. The dog was depressed with grade 4 systolic murmur on cardiac auscultation. Based on cardiac examination, the dog was diagnosed with severe subaortic stenosis with involvement of the anterior mitral valve. β-blocker administration was initiated and clinical signs were improved, but not fully resolved. Balloon valvuloplasty was performed and the dog survived for nearly one year without clinical sign and the cardiac troponin I level was normalized. This case describes successful management of severe subaortic stenosis in a small breed dog through balloon valvuloplasty.

Keywords: balloon valvuloplasty, canine, cardiac troponin I, subaortic stenosis, β-blocker

Aortic stenosis is one of the most common congenital heart diseases in dogs [10, 11]. According to the lesion level to the aortic valve, aortic stenosis is classified into subvalvular, valvular, and supravalvular types [1]. The subvalvular type is the most common lesion in aortic stenosis in dogs [1] and breed predisposition is well documented in large breed dogs such as Newfoundland, Golden Retriever, German Shepherd, and Boxer dogs [9].

The degree of subaortic stenosis (SAS) is typically classified as mild, moderate, or severe on the basis of the magnitude of the peak systolic pressure gradient across the area of stenosis [6]. The prognosis for dogs with severe obstruction is generally grave, with most expected to die suddenly with rarely suffering from left-side congestive heart failure while the dogs with mild or moderate obstruction is generally good, with most dogs having near-normal life expectancy [5, 6].

Medical therapy and balloon valvuloplasty is the mainstay of therapy for severe SAS, and even in some clinics, surgical resection of obstructing lesion is challenged [3, 7]. There are several reports describing procedure of aortic balloon valvuloplasty done in large breed dogs and surgical intervention, which showed varied pressure gradient reduction, but none of the reports have been reported aortic balloon valvuloplasty in small or toy breed dogs [6, 7]. As the effect of the breed or body size on response to treatment or survival time in dogs with severe SAS is unknown, the proper treatment of choice in small breed dogs with severe SAS may be confusing.

This case describes balloon valvuloplasty in a Pomeranian dog with severe SAS and successful management with cardiac biomarker monitoring. To author’s knowledge, small breed dogs were also known to have SAS but balloon valvuloplasty for SAS in these small breed has not been reported.

A nine-month-old, intact female Pomeranian dog (3.15 kg of body weight) with heart murmur was presented for further examination of exercise intolerance and intermittent syncope. The syncope episodes initiated from four month old and the episodes had been more frequent recently. In physical examination, the dog was depressed with harsh grade 4/6 systolic ejection murmur (P, point of maximal impulse) on cardiac auscultation.

On the phonocardiogram, there was a systolic ejection murmur having features of diamond shape murmur and early diastolic murmur, suggesting stenotic heart disease and outflow tract insufficiency (Fig. 1). Hematological and biochemical examinations revealed mild leukocytosis (18.51 × 10^9/L; reference, 5.05–16.7 × 10^9/L), and increased creatinine kinase (252 U/L; reference, 10–200 U/L) level. Thoracic radiography showed left-sided heart enlargement with aorta bulging. The electrocardiogram revealed normal sinus rhythm with left deviated mean electrical axis (−30'; reference, 40–100') with increased R amplitude (2.8 mV; reference, <2.5 mV) and depressed ST segment (Fig. 1), suggesting left ventricular hypertrophy and myocardial hypoxia or infarction.

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The two-dimensional echocardiography revealed stenotic left ventricular outflow tract (LVOT) with interventricular fibromuscular ridge, restrictive movement of anterior mitral valve, increased echogenicity of the subendocardial region and thickened left ventricular free wall and interventricular...
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septum with left atrial dilation (Fig. 2A). In right parasternal 5 chamber view, abnormally thickened chordae tendinae reaching stenotic annulus ring and thickened aortic valves were identified (Fig. 2B). Color and continuous wave Doppler echocardiographic studies revealed a systolic turbulent flow in aortic root (Fig. 2C) at peak velocity of 8.88 m/sec (pressure gradient, 315 mmHg) and regurgitant flow in left atrium at peak velocity of 8.53 m/sec. Aortic regurgitant flow in ventricular diastole (peak velocity of 4.53 m/sec) was also identified. In left parasternal apical 4 chamber view, we could find abnormal mitral valve motion with moderate mitral regurgitation, suggesting septal mitral valve involvement in LVOT obstructive lesion (Fig. 1). Based on these findings, severe SAS with mitral valve involvement was diagnosed. The dog was medically treated with β-blocker (atenolol, 0.5 mg/kg, per orally [PO], twice a day [BID]; Hyundai Pharm, Korea), diuretics (furosemide, 0.5 mg/kg PO, BID; Handok Pharmaceutical, Korea), angiotension-converting-enzyme inhibitor (ramipril, 0.125 mg/kg, PO, once a day [SID]; Intervet, Nederlands), pentoxyfylline (10 mg/kg PO, BID; Handok Pharmaceutical) and anti-platelet drug (clopidogrel, 3 mg/kg PO, SID; Sinil Pharmaceutical, Korea) for a week. The syncopal episode disappeared but exercise intolerance with depression persisted and balloon valvuloplasty was performed.

The patient was underwent general anesthesia with propofol (4 mg/kg; Myungmoon Pharm, Korea) after premedication with butorphanol (0.2 mg/kg; Myungmoon Pharm). The dog was supplied oxygen and maintained with isoflurane (Terrell; Piramal Critical Care, USA) through endotracheal tube. A skin incision was made over the right jugular area to expose right carotid artery. After exposing the right carotid artery, a hair-wire was inserted into the carotid artery through 18 gauge over-the-needle catheter. A 6 Fr introducer sheath of 7 cm length (Check-Flo; Cook Medical, USA) was then inserted to the right carotid artery with guidance of preplaced hair-wire. A guide-wire (Rosen guide-wire; Infiniti Medical, USA) with 5 Fr angiographic catheter (Angled; Cook Medical) was then inserted through the introducer and proceeded to left ventricle under C-arm monitoring simultaneously with electrocardiogram monitoring for fatal arrhythmia. The iohexol (Omnipaque; GE Healthcare, UK) with mixture of 1 : 2 saline was used. The obstructive lesion of left ventricular outflow tract was confirmed (Fig. 3A) and balloon dilatation catheter that matches the aortic valvular annulus size was prepared. After withdrawing angiographic catheter, the balloon dilatation catheter (8 mm × 4 cm, Tyshak I; Infiniti Medical) was then inserted and located at the stenotic left ventricular out-

Fig. 1. (A) The phonocardiogram from the dog with systolic diamond shape murmur, suggesting outflow tract obstructive lesion. There is also an early diastolic murmur indicating aortic insufficiency. (B) Electrocardiograph from dog with SAS. There are increased R amplitude (2.8 mV; reference range, < 2.5 mV) and depressed ST segment (0.3 mV; reference range, < 0.2 mV) with left deviated mean electrical axis, suggesting left ventricular hypertrophy and myocardial hypoxia or infarction.

Fig. 2. An echocardiography from the dog showing left ventricular outflow tract obstruction. (A) In right parasternal 4 chamber view in diastole, abnormal mitral valve coaptation is noted with stiff septal mitral valve movement, indicating involvement of septal leaflet of mitral valve in subaortic stenosis. There are also left atrial dilation and left ventricular concentric hypertrophy. (B) In right parasternal 5 chamber view, a fibromuscular lesion on interventricular septum and septal mitral valve is connected to the lesion with fibrous annulus ring. A thickened chordae tendinae (white arrow) is attached the annulus ring, showing severe left ventricular outflow tract (LVOT) obstruction. (C) In modified right parasternal 5 chamber view with more rotation, the fibrous annulus ring is prominent and color Doppler in a corresponding plane shows flow acceleration through the short-segment obstruction. Thickened aortic valve and post-stenotic dilation of ascending aorta is also noted.

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Flow tract lesion through the introducer. With inflation device (Sphere inflation device; Cook Medical), the balloon was inflated until the indentation of balloon disappeared and then quickly deflated while monitoring patient’s heart rate and blood pressure (Fig. 3B). This inflation procedures were repeated 5 times until the indentation from stenotic lesion (Fig. 3C) disappeared and then all devices were removed. The right carotid artery was ligated and incision line was sterilized and bandaged. The patient discharged the day after intervention without arrhythmia or severe complication and oral antibiotics (cephalexin, 30 mg/kg, PO, BID; Korus Pharm, Korea) was prescribed.

The echocardiography after the day of intervention revealed reduced aortic pressure gradient about 20% (251 mmHg from 315 mmHg) and cardiac troponin I (cTnI) monitoring revealed markedly increased (1.29 from 0.24 ng/mL; reference, < 0.2 ng/mL), indicating cardiac muscle damage or infarction from the intervention (Table 1). The clinical signs disappeared and cTnI level had been maintaining in normal range (< 0.2 ng/mL) after the intervention. The patient discharged the day after intervention without arrhythmia or severe complication and oral antibiotics (cephalexin, 30 mg/kg, PO, BID; Korus Pharm, Korea) was prescribed.

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Subaortic stenosis is a common congenital heart disease in large breed dogs [10]. As the dogs with mild and moderate SAS can live well even without clinical sign, the pressure gradient reduction through balloon valvuloplasty or surgical intervention may be the reasonable treatment of choice in severe SAS [5], which needs nearly 75% pressure gradient reduction in this patient. A reduction in severity of > 25–50% without a notable increase in aortic regurgitation is considered a successful outcome in aortic balloon valvuloplasty [11].

In this case, the velocity of aortic flow which reflects the severity of stenotic lesion was insignificantly reduced. The first possible cause may be the increased cardiac output that could be expected from improved clinical signs. In a tight stenosis with a small diameter, a very small increase in diameter and cross-sectional area could result in a large increase in flow (e.g., a 70% stenosis could allow more than five times the flow as would an 80% stenosis at the same pressure) [2]. Moreover, the pressure gradient is affected by stroke volume [6]. An increase in stroke volume consequent to reduction of the obstruction may increase the pressure gradient, so that the benefit of balloon valvuloplasty could be underestimated [6]. The other possibility would be the type of stenosis in this case. The fibromuscular ridge or ring in SAS is believed to require greater radial force to sufficiently tear and achieve an increase in effective orifice area as compared to the force required to tear fused valve leaflets as is the goal in balloon pulmonary valvuloplasty for dogs with congenital pulmonary valve stenosis [11]. Moreover, the thickened chordae tendinae continuously connecting the fibrous annulus ring and left ventricle in this case could make tearing the lesion more difficult.

In dogs with congenital SAS, high cTnI level may be an indicator of cardiac injury from microvascular disease and subsequent ischemia [8]. In this case, 2 specific findings (ST segment change of electrocardiogram and ultrasonic hyperechogenicity of the myocardium) were interpreted as indicators of ischemic damage. In both humans and animals with

**Table 1.** The serial results of cardiac troponin I (cTnI) concentration in the dog with subaortic stenosis

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Day 0*</th>
<th>Day 1†</th>
<th>8 months after day 0</th>
<th>10 months after day 0</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>cTnI (ng/mL)</td>
<td>0.24</td>
<td>1.29</td>
<td>&lt; 0.2</td>
<td>&lt; 0.2</td>
<td>&lt; 0.2</td>
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*The two days after the first visit. †Nine days after the first visit (the day of intervention).
pressure overload, mortality is likely associated with the degree of left ventricular concentric hypertrophy, development of microvascular disease, and resultant ischemia, which may be associated with sudden death or clinical signs in SAS dogs [5, 8]. Therefore, normalized cTnI in this case would suggest possible role of balloon valvuloplasty in improved clinical sign and reduced pressure overload of severe SAS despite of minimal pressure gradient reduction.

This report describes balloon valvuloplasty in small breed dog (3.15 kg of body weight) and the dog lives without clinical sign longer than previously reported median survival time of 19 months in severe SAS dogs [5]. In smaller breed dogs, the cardiac intervention can be more difficult to perform than in larger breed dogs, because of the limited size of introducer we can use. However, through the carotid artery, we can try larger introducers when comparing approach through femoral arteries like in patent ductus arteriosus occlusion. In addition, the smaller diameter balloons can provide more radial force in comparison to larger diameter balloons generally used in large breed dogs [11]. In regard to balloon valvuloplasty in severe SAS, the beneficial effect in survival time should be reconsidered despite of poor evaluation in large breed dogs [6].

In conclusion, the Pomeranian dog in this study was diagnosed with severe SAS. Medical treatment with β-blocker was initiated, but the clinical signs were not fully resolved. Through balloon valvuloplasty, this dog could have lived well with normalized cTnI level and without clinical signs for nearly one year. This case suggests that cTnI concentration can be used to evaluate the efficiency of the intervention, and balloon valvuloplasty can be useful treatment to relieve the pressure overload of severe SAS also in small breed dogs despite of lack of aortic balloon valvuloplasty in small breed dogs.

References