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VETERINARIA

Aortic balloon valvuloplasty in a dog

In this article we present a case of aortic balloon valvuloplasty in a 6-year old dog. The diagnosis of severe aortic stenosis was made by transthoracic echocardiography while the interventional procedure was monitored by transoesophageal echocardiography and fluoroscopy. Angiography was found to be a reliable method for measuring the valve annulus. A ventricular tachycardia of 220 beats/minute, induced by a pacing catheter introduced into the right ventricle, prevented dislocation of the valvuloplasty balloon during its dilation, thereby avoiding injury to the valve leaflets or surrounding tissues.

Marta Claretti
Med Vet

Danitza Pradelli*
Med Vet, PhD

Blanca Serrano Lopez
Med Vet

Alessandra Rosatelli
Med Vet

Francesca Martelli
Med Vet

Laura Mazzoni
Med Vet

Martina Angileri
Med Vet

Claudio Bussadori
Medico Chirurgo,
Med Vet, Dipl. ECVIM
(Cardiology), PhD

INTRODUCTION

Aortic stenosis is a congenital heart disease in which there is narrowing of the left ventricular outflow tract below, at, or above the valve. The subvalvular form is the most common in dogs, while the incidence of valvular stenosis is much lower[1].Valvular aortic stenosis was diagnosed in a 6-year old Boxer which was subsequently treated by valvuloplasty.

On clinical examination, a purebred Boxer had pulsus parvus et tardus and an early to mid-systolic grade III/VI crescendo-decrescendo ejection murmur with maximum intensity over the aortic valve auscultation point; the murmur radiated to the proximal third of the carotid arteries.

CASE REPORT

A sexually intact, male, 6-year old purebred Boxer, weighing 35 kg, was referred to the Gran Sasso Veterinary Centre by his veterinarian following a diagnosis of aortic stenosis. The heart murmur was detected during a routine clinical examination, after which an echocardiographic examination was requested.

At the time of diagnosis the dog, which had been adopted about a year previously, was asymptomatic for cardiac disease and, following the first examination carried out in another centre, was prescribed pharmacological therapy with atenolol at a dose of 1 mg/kg once daily.

On clinical examination at our centre, the dog had a respiratory rate of 32 breaths/minute and a heart rate of 120 beats/minute. The non-pigmented mucosal membranes that could be inspected appeared rosy; the capillary refill time, evaluated at the gingival mucosa, was <2 sec. The apex beat appeared normal, whereas the femoral arterial pulse seemed low-volume and slow-rising (*pulsus parvus et tardus*). On cardiac auscultation normal heart sounds could be heard and there was an early to mid-systolic grade III/VI murmur, with maximum intensity over the aortic valve auscultation point, radiating to the proximal third of the carotid arteries.

Two-dimensional echocardiography and Doppler stud-

Echocardiography showed a mild hypoplastic aortic annulus and fusion of the valve leaflets with systolic doming.

Clinica Veterinaria Gran Sasso (Milano)
* Corresponding Author (danitza.pradelli@gmail.com)

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ies were performed according to the procedures described in the literature[2], with the dog in the lateral decubitus position, using an Esaote MyLab Class C® ultrasound system and a phased-array, multifrequency Esaote PA240® 1-4 MHz transducer. The resulting images revealed mild to moderate concentric hypertrophy of the left ventricle, slight hypoplasia of the aortic annulus (16.3 mm), thickening of the aortic valve leaflets which were fused, systolic doming and post-stenotic dilatation of the aorta (Figure 1). Spectral Doppler analysis of the subcostal view showed accelerated anterograde flow with a peak velocity of 5.29 m/s, corresponding to a transaortic valve gradient of 112 mmHg, which is compatible with severe aortic stenosis, and a small, haemodynamically irrelevant regurgitant jet due to concomitant insufficiency of the valve.

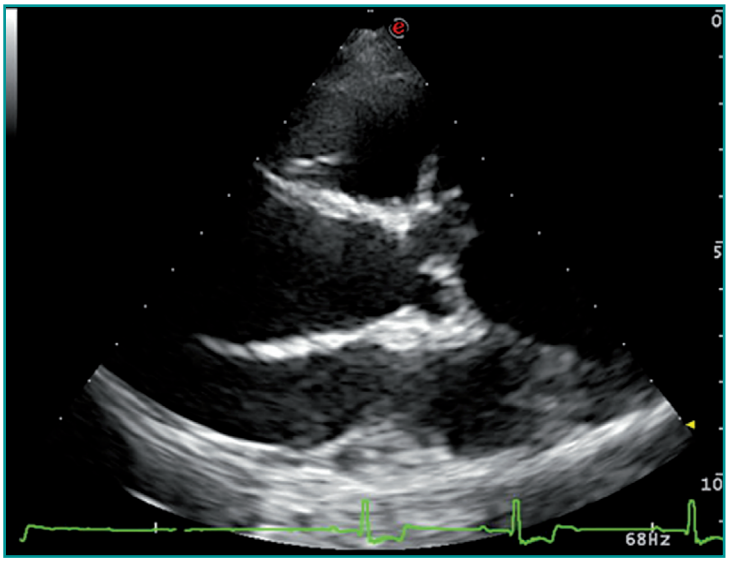

Figura 1 - Left parasternal long-axis view showing thickening of the aortic valve leaflets and dilation of the aortic root distal to the stenosis.

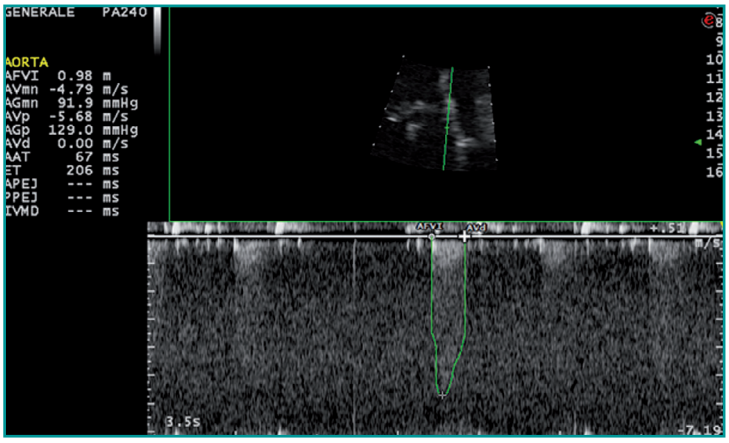

Figura 2 - Spectral Doppler of the aortic flow measured from the subcostal view. There is accelerated anterograde flow, an expression of severe aortic stenosis.

The left ventricular diastolic filling pattern was recorded from the left apical parasternal view and was found to have a pseudonormal morphology (on the basis of an E/A ratio of 1.5, an E wave deceleration time of 171 msec and an E/E' ratio of 11). Minimal mitral valve regurgitation was also detectable in the colour Doppler examination.

About 2.5 months after the initial evaluation, the dog was brought back to our centre to undergo aortic valvuloplasty. When taking the animal's recent medical history, the owners reported that the dog tired more readily and had had two episodes of syncope during exertion.

A lateral chest X-ray with the dog in right decubitus showed a dilatation distal to the stenotic valve, which was visible as a dorso-cranial prominence overlying the cranial cardiac silhouette. In the ventro-dorsal view, the cardiac outline appeared elongated due to hypertrophy of the left ventricle; the dilated aortic arch extended into the cranial mediastinum. In both projections the lung fields appeared normal.

The dominant rhythm seen during the electrocardiographic evaluation was sinus arrhythmia with a discharge frequency of between 50 and 100 beats/minute, interrupted by isolated premature supraventricular complexes, first-degree atrioventricular block and signs of left ventricular hypertrophy in the absence of intraventricular conduction delays.

Ventricular overdrive at 220 beats/minute, obtained by means of a pacing lead placed at the apex of the right ventricle, resulted in greater stability of the valvuloplasty balloon during inflation of the balloon.

The preoperative echocardiographic examination, performed prior to sedating the patient, allowed the morphology of the aortic valve to be studied again: the valve was tricuspid and the peak velocity of the accelerated anterograde flow was 5.68 m/s (corresponding to a transvalvular gradient of 129 mmHg), associated with a dynamic component (Figure 2).

Prior to the intervention, the patient was pre-medicated with butorphanol 0.4 mg/kg, atropine 0.02 mg/kg and acepromazine at a dosage of 5 µg/kg. Induction was obtained with propofol 6 mg/kg and general anaesthesia was maintained with isoflurane. After isolation of the femoral vessels and positioning of the respective introducers, heparin was administered intravenously at a dose of 100 IU/kg.

The entire intervention was followed by transoesophageal echocardiography using an Esoate MyLab

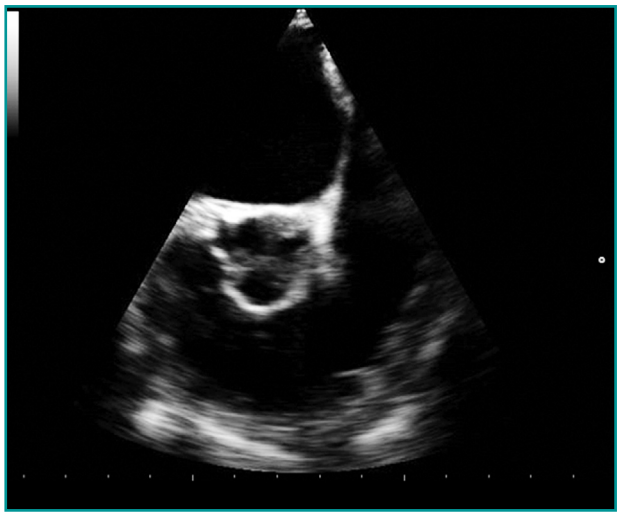

Figura 3 - Short-axis view of the aortic valve in transoesophageal echocardiography. Note the three valve cusps, which are markedly thickened.

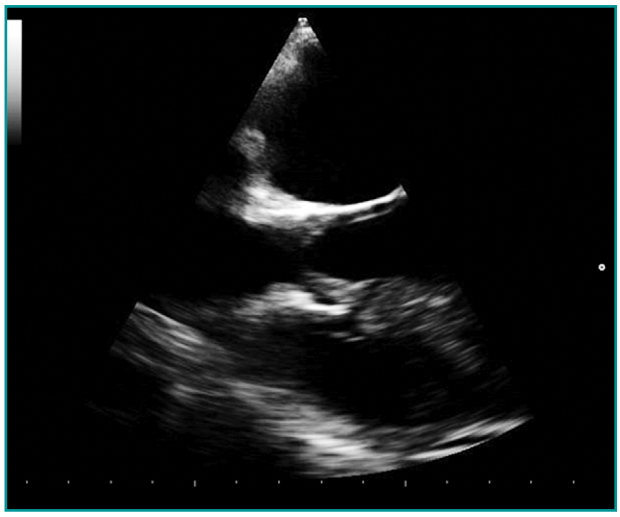

Figura 4 - Long-axis view of the aortic valve in transoesophageal echocardiography. Rudimental leaflets with reduced mobility.

30 Gold® ultrasound system with an Esaote TEE022® 3 - 7.5 MHz multifrequency transoesophageal probe and fluoroscopy (Philips Veradius Neo®). The images obtained during the transoesophageal ultrasound examination revealed the three cusps of the aortic valve and the morphology of the valve in more detail (Figure 3, Figure 4); this method was also used to measure the aortic valve annulus, which resulted of 16.2 mm.
Vascular access was secured by surgical isolation of the left femoral artery and vein, into which two introducers, respectively 10 Fr and 7 Fr (Cordis®), were inserted. Through the introducer placed in the femoral artery the left ventricle was catheterised with a 7 Fr Multipurpose catheter and a 0.035' guidewire with a straight tip, subsequently replaced by a super-stiff guidewire and a 6 Fr pig-tail angiography catheter in order to perform a ventriculography (Figure 5, Video 1), which enabled evaluation of the morphology of the stenosis and precise measurement of the aortic annulus, which had a diameter of 16 mm. Since transthoracic echocardiography had shown a dilation of the left coronary sinus (Figure 6), we then performed selective coronary arteriography through a 6 Fr Multipurpose catheter, which excluded the presence of anatomical abnormalities of this circulation (Figure 7, Video 2). Through the introducer in the femoral vein, a St. Jude Medical® bipolar electrostimulation catheter, connected to a St. Jude Medica® Merlin transmitter, was positioned at the apex of the right ventricle (Figure 8).
The super stiff Amplatz Support (Cook)® exchange wire was replaced by a super stiff Boston® 0.035' exchange wire through which a catheter was introduced with a 16 mm x 4 cm Z-Med balloon. The balloon was inflated twice in quick succession to dilate the valve (Video 3). Ventricular tachycardia was induced by stimulating the right ventricle at 220 beats/minute to maintain the catheter in place during inflation of the balloon.
The electrocardiographic changes seen during surgery

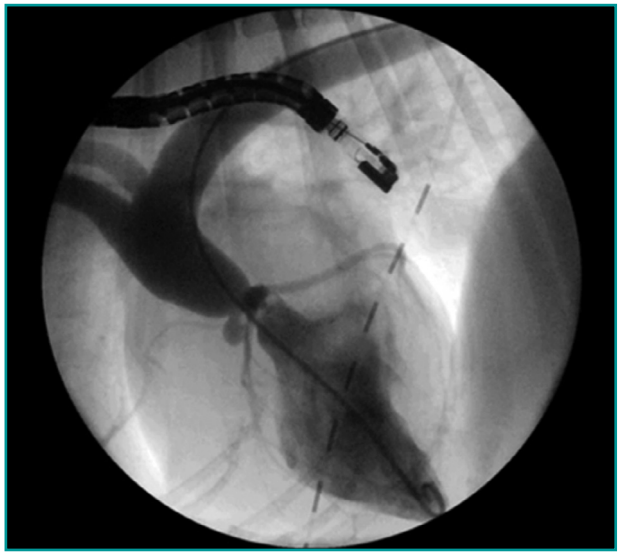

Figura 5 - Left ventriculography, performed using a pig-tail angiography catheter, shows the stenotic valve, aortic root dilatation and coronary vasculature.

Video 1. Left ventriculography performed using a pig-tail angiography catheter. The stenotic valve, aortic root dilatation and coronary vasculature can be seen.
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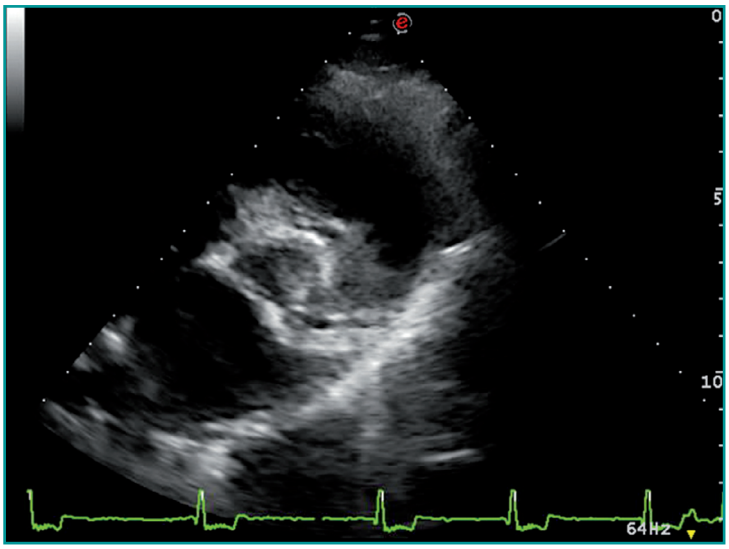

Figura 6 - Right parasternal short-axis view of the base of the heart in which the origins of both coronary arteries and dilatation of the common trunk can be seen.

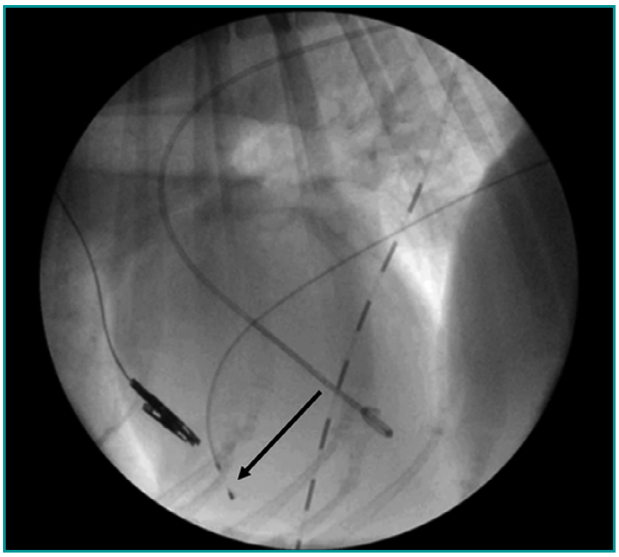

Figura 8 - Pacing lead positioned at the apex of the right ventricle (arrow).

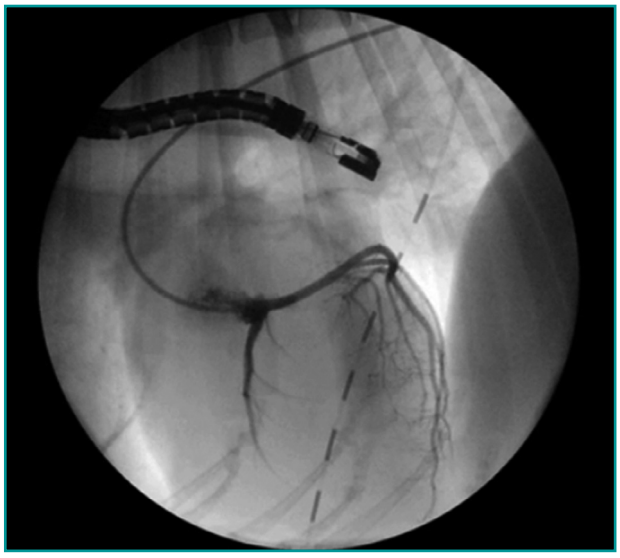

Figura 7 - Selective coronarography of the left coronary artery, performed with a Multipurpose angiography catheter, confirmed the absence of abnormalities in the vessel's course.

Video 2. Selective left coronarography. Selective angiography of the left coronary artery, performed using a Multipurpose angiography catheter, confirmed the absence of abnormalities of the course of the artery.
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Video 3. The lead positioned at the apex of the right ventricle enabled pacing at 220 bpm. Contemporaneously, the valve was dilated via catheter with a 16 mm x 4 cm Z-Med® balloon.
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The critical features of aortic valvuloplasty are the choice of the balloon and the difficulty in keeping the balloon in situ during its dilatation.

were ventricular extrasystoles and premature supraventricular complexes. At the end of the procedure the patient developed a low frequency accelerated idioventricular rhythm, which resolved spontaneously about 2 hours after regaining consciousness, the period in which sinus rhythm returned, with isolated monomorphic ventricular extrasystoles. The patient remained in our centre for 12 hours. At discharge the dog appeared alert and the electrocardiogram showed sinus rhythm in the absence of alterations. Given the patient's restlessness, only the velocity of anterograde aortic flow was measured, which was about 4.46 m/s, corresponding to a pressure gradient across the valve of 79.6 mmHg (Figure 9), with a reduction of 44%

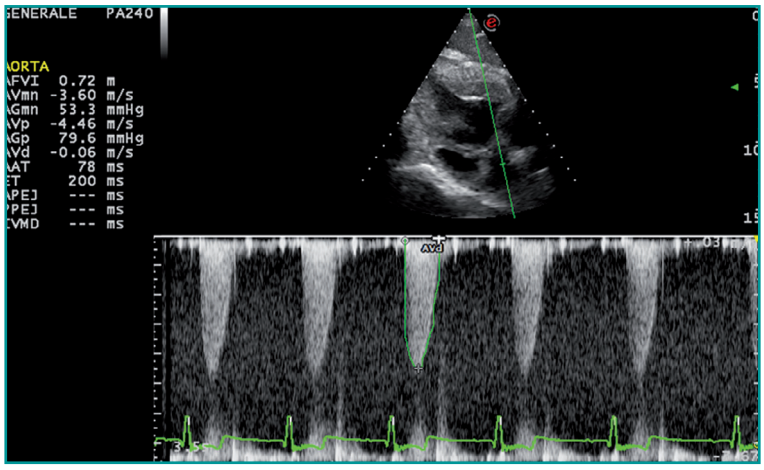

Figura 9 - Spectral Doppler of the aortic flow, performed using the subcostal view, showed an approximately 40% reduction in the transaortic valve gradient compared to that prior to the valvuloplasty.

compared to the preoperative examination.

DISCUSSION

Aortic stenosis is the second most common congenital heart disease described in dogs[1,3], with the subvalvular form being more frequent than the valvular form. A method involving the use of a cutting balloon associated with a high-pressure balloon has already been described for the treatment of subaortic stenosis in the dog[4], although there are currently no published data on the long-term outcome of this procedure. Aortic valvuloplasty was described for the first time in human medicine in 1983[5] and is the indicated therapy for this pathology. In veterinary medicine, as in human medicine, the treatment of aortic stenosis is indicated in symptomatic patients with severe stenosis and haemodynamically insignificant or mild valvular regurgitation. The critical features of this procedure are the choice of the valvuloplasty balloon and the method used to keep the balloon *in situ* while it is dilated.

It must be possible to inflate and deflate the aortic valvuloplasty balloon quickly, since the aim is to lower the transvalvular gradient as much as feasible while causing the least possible leakage at the valve itself. For this purpose, the diameter of the balloon must not exceed the diameter of the aortic annulus; indeed, in human medicine it is recommended using a balloon with a diameter 90% that of the aortic valve ring diameter[6-8], possibly increasing the diameter of the balloon by 1 mm if the pressure gradient is not reduced sufficiently and the aortic valve remains competent[9]. Angiography is decisive when selecting the size of the balloon, because it can provide an accurate measurement of the size of the valve annulus. It is of paramount importance to ensure adequate stability of the balloon during the dilatation procedure in order to avoid avulsion of the valve flaps and possible damage to surrounding tissues; furthermore, in human medicine it has been described that movement of the balloon lowers the efficacy of the procedure and leads to greater residual aortic valve regurgitation[10,11].

The difficulties in maintaining the catheter *in situ*, which are greater than those during pulmonic valvuloplasty, are due to the high left ventricular pressures and to the anatomical configuration of the outflow tract. The strategies used in human medicine involve intravenous administration of adenosine or high frequency cardiac stimulation by means of a lead placed at the apex of the right ventricle. In our patient, we chose to use the effect of ventricular overdrive at 220 beats/minute, obtained using a bipolar pacing catheter placed at the apex of the right ventricle, because adenosine has a predominantly hypotensive effect in dogs under anaesthesia and may, therefore, be more difficult to use[12].

KEY POINTS

- The diameter of the aortic valvuloplasty balloon must be equal to, or slightly less than the diameter of the aortic annulus and the valve ring must be dilated once, or at most twice.
- Ventricular overdrive, obtained by means of a pacing lead positioned at the apex of the right ventricle, ensures the balloon's stability during the dilatation procedure, thus avoiding avulsion of the valve leaflets and possible damage to surrounding tissues.

Valvuloplastica aortica in un cane

In questo articolo viene descritto il caso di un Boxer maschio di 6 anni affetto da stenosi valvolare aortica grave e sottoposto a valvuloplastica aortica presso il nostro centro.

La diagnosi è stata effettuata mediante esame ecocardiografico transtoracico, mentre tutta la procedura è stata seguita tramite ecocardiografia transesofagea (TEE) e fluoroscopia. L'angiografia si è rivelata essere una metodica attendibile per la misurazione dell'annulus valvolare. L'induzione di una tachicardia ventricolare a 220 b/min mediante una stimolazione diretta in ventricolo destro ha evitato la dislocazione del pallone da valvuloplastica durante la dilatazione dello stesso e, conseguentemente, i possibili danni ai tessuti circostanti provocati da un'eventuale dislocazione del pallone stesso.

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