Subaortic Stenosis, Aortic Insufficiency, and Patent Ductus Arteriosus in a Dog

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Abstract:

At presentation, Sadie was a 3-year-old, intact female, mixed-breed dog with a chief complaint of a heart murmur and a history of collapse after exertion. Examination revealed a 5/6 systolic murmur on the left, with a diastolic component, at times taking on a continuous character. Femoral pulses were weak, and examination was otherwise unremarkable. Radiographs revealed generalized cardiomegaly, hypervascular lungs, and dilatation of the aortic arch. Her differential diagnosis included subaortic stenosis (SAS) with aortic insufficiency (AI), and a patent ductus arteriosus (PDA).

An echocardiogram revealed a subaortic ridge, left ventricular hypertrophy, and enlargement of the left atrium, consistent with SAS. Continuous wave Doppler showed turbulent flow in the aortic outflow tract, increased outflow velocity of 6 m/s, and aortic insufficiency. The PDA was also observed, with Doppler showing continuous blood flow through the ductus. Her final diagnosis was severe SAS with AI, and a PDA.

The PDA was treated via transcatheter coil occlusion in order to resolve the PDA and the associated left ventricular volume overload which was exacerbating the stenosis. After surgery, echocardiographic evaluation showed 90% closure of the PDA, and diminished aortic outflow velocity (3.9 m/s). Her diagnosis was downgraded to mild to moderate SAS, and her prognosis upgraded to good.

History:

Sadie, a 3-year-old, intact female, mixed-breed dog, presented to the Cardiology service of the Cornell University Hospital for Animals on 8/29/02 with a chief complaint

of a heart murmur. This murmur was first observed by the owner, using a stethoscope soon after adopting the dog from a shelter approximately one year prior. A referring veterinarian also observed the murmur, and placed Sadie on Enacard (enalapril, 10 mg PO SID). In October of 2001, the owner observed an episode of collapse after playing, in which Sadie first became "wobbly," and then could not walk. Shortly after the event, evaluation by a referring veterinarian revealed no significant abnormalities apart from the murmur. The owner has also observed Sadie to enter periods of marked depression and lethargy while in heat. The owner was interested in having Sadie spayed, but a full cardiac evaluation was recommended by the referring veterinarian prior to anesthesia and surgery.

Presentation and Examination:

At the time of presentation, Sadie was still on her treatment regimen of Enacard.. Examination revealed a bright, alert, and responsive dog. Auscultation revealed a bilaterally audible systolic murmur, graded 5/6 and heard best on the left thorax. A weaker diastolic component could also be ausculted on the left side. At times, the murmur took on a continuous ("machinery") character. Sadie's femoral pulses were slightly weak. Her examination was otherwise unremarkable.

The variable character of the heart murmur allowed for an extensive differential diagnosis. The systolic component could be due to subaortic or pulmonic stenosis, mitral or tricuspid insufficiency, or a septal defect (atrial or ventricular). A diastolic component could be caused by aortic insufficiency or a mitral valve stenosis. A continuous murmur is most often associated with a patent ductus arteriosus (PDA), but may also be present in

rarer congenital disorders such as a pulmonary arteriovenous fistula.[1] Sadie's weak pulses were suggestive of a subaortic stenosis (SAS), which would commonly be associated with aortic insufficiency (AI), resulting in a systolic and diastolic ("to and fro") murmur, consistent with what was ausculted.

Diagnostics:

Survey radiographs were taken to assess any pulmonary congestion, and to observe the cardiac sillouhette. The heart was generally enlarged. No sign of congestion was observed on lateral films, but the lungs were mildly hypervascular, suggestive of pulmonary overcirculation (as would be seen in a PDA). A ventrodorsal film showed a marked dilatation in the aortic arch, a sign most often associated with SAS (post-stenotic "jet lesion") or a patent ductus arteriosus ("ductal bulge"), effectively narrowing our differential diagnosis to two disorders. Because radiographs are limited in evaluating the heart, further diagnostics were needed.[2]

An echocardiogram revealed a thickened ridge beneath the aortic valve, consistent with SAS. Mild thickening of the left ventricular free wall (concentric hypertrophy), and marked enlargement of the left atrium were also present, and are expected consequences of SAS, due to left ventricular pressure overload. Color flow Doppler showed markedly turbulent flow through the aortic valve during systole, and regurgitation of blood back through the valve on diastole (AI). Continuous wave Doppler was used to evaluate the velocity of blood flow through the aortic valve. Peak left ventricular outflow velocity was measured at 6 m/s (normal is 1-2 m/s), consistent with SAS. Based on echocardiographic evidence, a diagnosis of severe SAS with AI was made.

Further evaluation via echocardiogram demonstrated a PDA, and color flow Doppler showed marked turbulence in the area of the pulmonary artery. Continuous wave Doppler was used to measure the velocity of bloodflow through the PDA, and showed continuous flow from the aorta into the pulmonary trunk, maintained at approximately 4 m/s. This evidence allowed a diagnosis of a PDA, in addition to the previous diagnosis of SAS with AI.

Prognosis:

The prognosis for a dog with SAS is dependent on severity of the defect. The danger of SAS arises from left ventricular hypertrophy in response to pressure overload. This concentric hypertrophy results in myocardial ischemia and malignant arrhythmia, which may lead to syncopal episodes and sudden death.[1] Increased severity of the stenosis will hasten this progression, resulting in a less favorable prognosis. In cases where ischemia and sudden death do not occur, ventricular hypertrophy may still lead to congestive heart failure.[1] In general, severe cases will succumb to sudden death within the first three years of life, while milder cases tend to enter congestive heart failure later in life.[3]

Patent Ductus Arteriosus has a similarly poor prognosis. In this disorder, blood is shunted from the aorta, through the lungs, and back to the left heart, causing a left-side volume overload. Eccentric hypertrophy of the left ventricle and atrium results, ultimately, in congestive heart failure. Untreated animals generally succumb to pulmonary edema within one year of diagnosis.[1]

In Sadie's case, the presence of both defects results in a more complex pathology. Namely, volume overload of the left heart – caused by the PDA – is resulting in an additional *relative* aortic stenosis to compound the pre-existing SAS. In effect, an increased volume of blood is being passed through an already stenotic outflow tract, resulting in further pressure overload of the left heart. In an uncomplicated SAS, it is pressure overload which drives the progression of disease. Exaggerating this pressure overload will result in faster progression, and a poorer prognosis.

Management:

In veterinary medicine, there is no definitive treatment for SAS. This congenital disorder is caused by the presence of a tough, fibrous ring which encircles the left ventricular outflow tract, just proximal to the aortic valve.[1] Dilation of this stenosis using a balloon catheter has been unsuccessful, often resulting in only a transient palliative effect.[1] Open-heart surgery has been employed to correct this defect via resection or bypass, but is rare due largely to the complications of such a procedure (including hemorrhage and pneumonia).[4]

In contrast, PDA has long been treatable in veterinary medicine. Because all pathology is due to the aberrant persistence of a fetal vessel, obliteration of the vessel is immediately corrective. This has generally been accomplished via surgical ligation of the PDA. Prognosis with this procedure is excellent, and post-surgical flow through the ductus arteriosus is usually minimal if not entirely absent.[1] Although this procedure is generally curative, it does require invasive open-chest surgery and a painful recovery. An alternate new technique, called a transcatheter coil occlusion, has been developed

which circumvents this. In this procedure, a metal coil is delivered directly to the PDA using a specialized catheter, introduced from a femoral artery. The coil is lodged within the PDA, and serves as a nidus for clotting and vessel fibrosis, effectively blocking the vessel.[5] This procedure is relatively non-invasive, but technically challenging. Should the coil not lodge properly, it may relocate to occlude a pulmonary artery.[1]

Treatment and Re-evaluation:

The decision was made to proceed with a transcatheter coil occlusion, in order to cure the PDA, and to resolve the relative aortic stenosis caused by the PDA. After surgery, a repeat cardiac evaluation would be performed to assess the severity of Sadie's newly uncomplicated SAS, and to adjust her prognosis accordingly.

The coil occlusion was successfully performed on 10/14/02. On physical examination, a grade 3/6 systolic murmur was ausculted on the left. Continuous wave Doppler measured peak aortic outflow velocity at 3.9 m/s, which was considerably lower than the 6 m/s observed at presentation (but still higher than a normal 1-2 m/s). Accordingly, she was given an adjusted diagnosis of mild to moderate SAS. Doppler evaluation of the PDA revealed significantly diminished flow through the aberrant vessel, judged a 90% closure.

Sadie's adjusted diagnosis carries with it a more favorable prognosis. Although her SAS has not been cured, the effects of the stenosis have been drastically reduced by resolving the volume overload associated with the PDA. Having a mild to moderate SAS, she is still at some risk for developing congestive heart failure later in life, and will accordingly remain on her current course of Enalapril for now. Without the severe

pressure overload, Sadie's heart is expected to undergo continued remodeling in the coming months.[6] It is also possible that the small amount of residual flow through the PDA will eventually abate altogether. A repeat cardiac evaluation in three months will help to assess her progress following surgery, and to further adjust the severity of her diagnosis and prognosis.

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