



**PATIENT**

Mia Marki

**SPECIES**

Canine

**BREED**

Smooth Collie

**SEX**

Female

**AGE**

6 weeks

**WEIGHT**

6.7 lbs

**INTERPRETED BY**

Eric Lindquist, DMV,  
 DABVP, Cert. IVUSS,  
 CEO of SonoPath.com

**IMAGING PERFORMED BY**

Shari Reffi, CVT

**HOSPITAL NAME**

VCA Blairstown AH

**REFERRING VET**

Dr. Summers

**INVOICE**

74138

**DATE**

4/3/26

**PRESENTING CLINICAL SIGNS**

- BCS 5/9
- Grade V/VI heart murmur, asymptomatic.
- No current meds.

**ULTRASONOGRAPHIC EXAMINATION OF THE HEART**

The echocardiogram in this patient presented volume overload in the left heart with tachycardia, mitral and tricuspid insufficiency. The aortic valve was mildly thickened. Aortic outflow velocity was severely excessive at nearly 5.0 m/sec. This is consistent with subaortic stenosis or compensatory elevated velocity owing to the patent ductus arteriosus. The deep pulmonary artery revealed a holosystolic jet at the level of the pulmonary bifurcation. This is consistent with patent ductus arteriosus. Mild volume overload of the right ventricle and pulmonary artery was noted. No pericardial or pleural effusion was noted.

<b>CANINE CARDIAC PARAMETERS</b>	<b>MR VMAX (m/s)</b>	<b>TR VMAX (m/s)</b>	<b>LA/AO</b>	<b>LA/AO (Heart Base)</b>	<b>FS (%)</b>	<b>EF (%)</b>	<b>EPSS (cm)</b>
<b>NORMAL PARAMETER</b>	4.5-5.5	<2.7	1.3	<1.6	28-40	40-100	<0.6
<b>PATIENT</b>	5.5	2.96	1.6	2.1	45	80	0.1
<b>CANINE CARDIAC PARAMETERS</b>	<b>HR (BPM)</b>	<b>AV VMAX (m/s)</b>	<b>PV MAX (m/s)</b>	<b>BODY WEIGHT</b>	<b>LA 2D short axis Base view (cm)</b>	<b>LVIDd Avg; 2D and m-mode short axis (cm)</b>	<b>LVIDs Avg; 2D and m-mode short axis (cm)</b>
<b>NORMAL PARAMETER</b>	50-100	0.7-1.7	0.7-1.6	BELOW	BELOW	BELOW	BELOW
<b>PATIENT</b>	180	4.97	2.3	6.7 lbs	3.4	2.9	

**ULTRASONOGRAPHIC FINDINGS**

Patent ductus arteriosus with elevated LVOT velocity from subaortic stenosis and/or compensatory elevated velocity.

Concurrent volume overload of the left atrium and left ventricle.

Mitral and tricuspid insufficiency.

**INTERPRETATION OF THE FINDINGS & FURTHER RECOMMENDATIONS**

I recommend referral to a cardiologist for potential intervention and further definition of the left ventricular outflow velocity as well as potential intervention for the patent ductus arteriosus. The aortic



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valve was thickened which would fit most with aortic stenosis, however, multiple congenital defects is suspected. If any evidence of fever history is noted, then I recommend blood culture to assess for potential endocarditis, which may be complicating the presentation.

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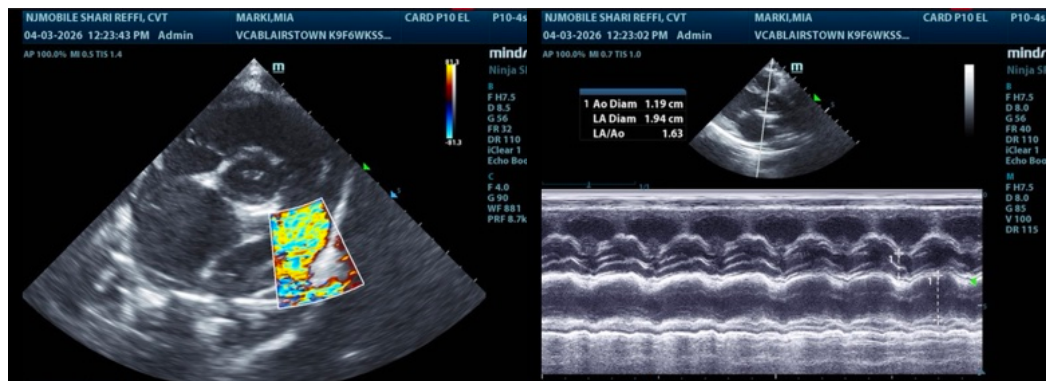
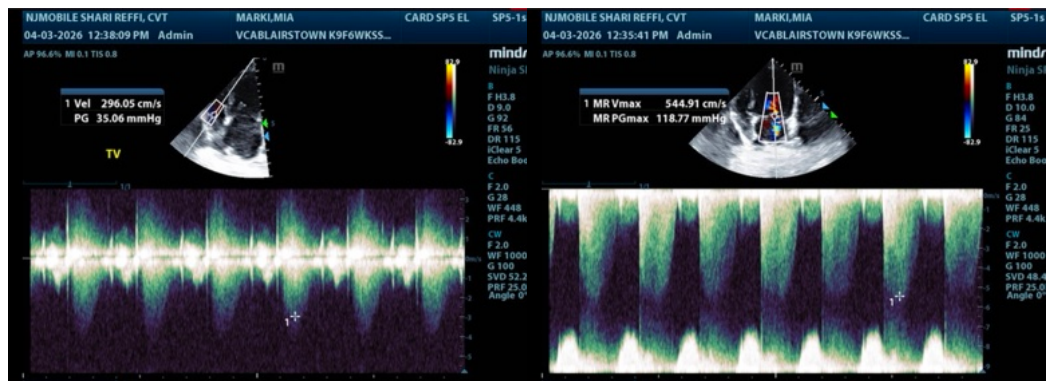
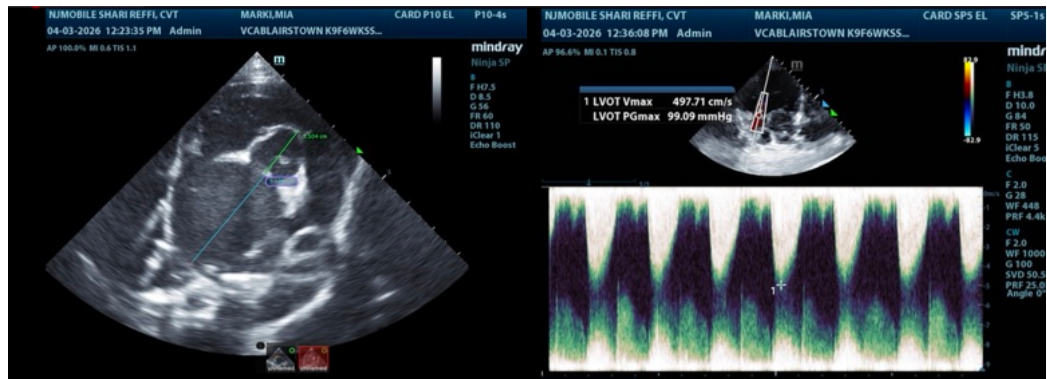
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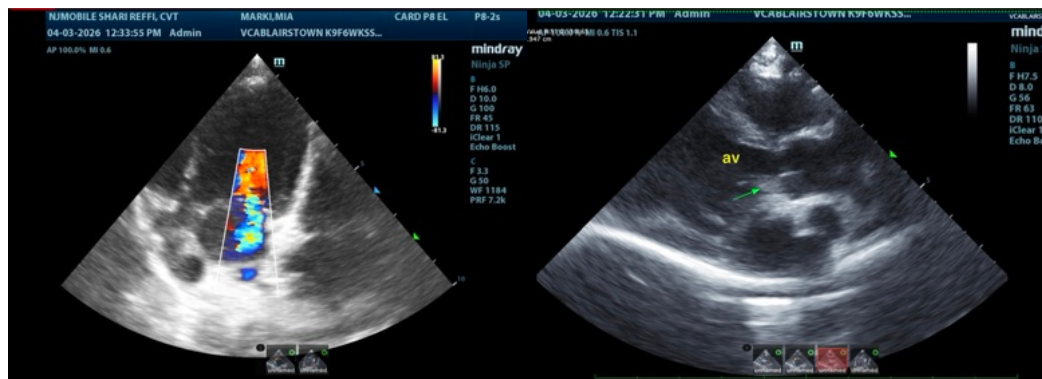
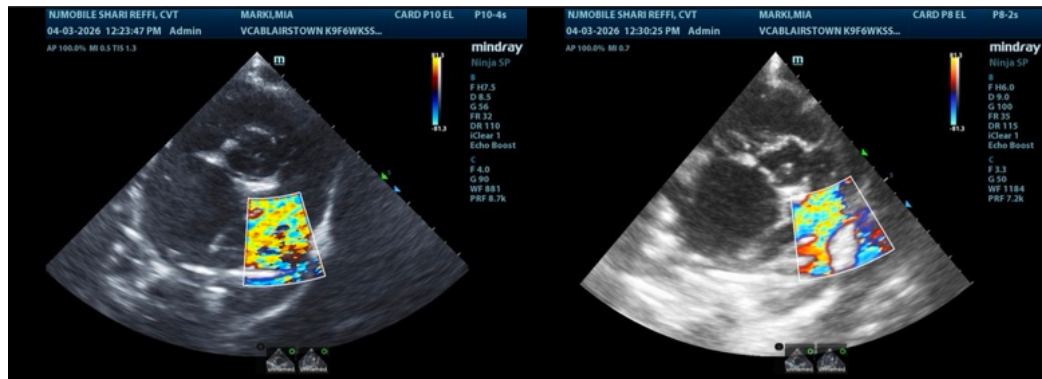
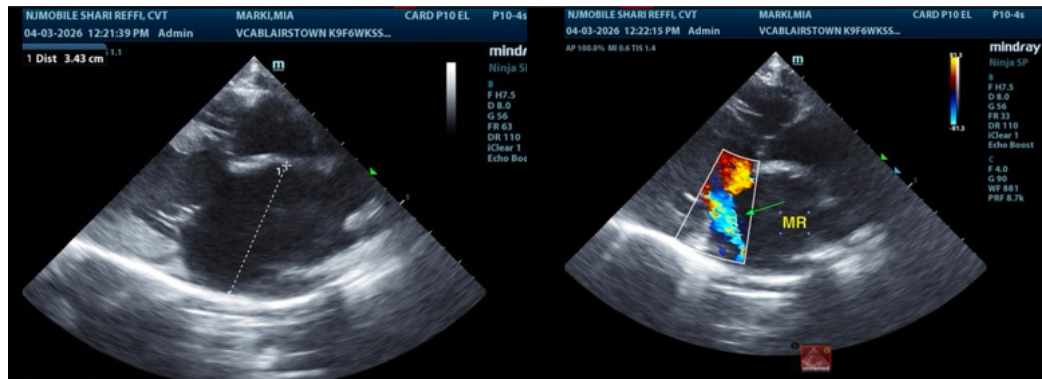
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The information and recommendations provided are based on the images presented by the referring veterinarian/sonographer. No evaluation can be communicated regarding pathology that was not visible in the image/video clips provided.

Thank you for this referral. If the clinical or image interpretation does not parallel your findings or if I can be of any further assistance please contact me.

Eric Lindquist, DMV, DABVP, Cert. IVUSS, CEO of SonoPath.com

Info@SonoPath.com



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## Patent Ductus Arteriosus

<http://www.sonopath.com/PDA>

<http://www.sonopath.com/PDAReversal>

<http://www.sonopath.com/EchoModler>

**Description:** Patent ductus arteriosus (PDA) is a common congenital heart defect in dogs and most commonly affects toy breeds. The PDA is graded on a scale of 1 to 6 based on the degree of asymmetric distribution of the smooth muscle architecture, which varies from funnel to tube-like structures. The more the smooth muscle architecture of the duct resembles the aortic wall, the higher the pathological grade. The physiologic closure of the ductus arteriosus is dictated by the cessation of prostaglandin negative feedback during partus, together with an increased oxygen tension. If the negative feedback and increased oxygen tension are prevented from occurring, the ductus arteriosus will remain open, resulting in a PDA.

**Clinical Signs:** A continuous machinery murmur located at the heart base and heard at the left 3<sup>rd</sup> intercostal space is typical for a PDA. The intensity of the murmur depends on the grade of the PDA, with a grade 6 PDA exhibiting a lower grade murmur than a grade 1 PDA. Additional signs include a mitral systolic murmur, bounding hyperkinetic femoral pulses, and a precordial thrill, which is palpated on the thoracic wall at the level of the heart base. Secondary pulmonary hypertension can develop, which can result in the murmur becoming more faint as right-to-left shunting occurs. Right-to-left reversal of the shunt results in exercise intolerance, caudal mucosal cyanosis, and polycythemia.

**Diagnostics:** Radiography may show cardiomegaly, which can stem from an enlarged pulmonary artery, left atrium, or left ventricle, and a prominent aortic arch. In some cases of PDA, the main pulmonary artery is distended and an increased vascular pattern is present. In other cases, radiographic changes may be undetectable, especially if the defect is small or volume overload is absent. In advanced stages, pulmonary edema due to congestive heart failure (CHF) can be present.

ECG findings include tall R waves consistent with left ventricular enlargement and occasionally wide P waves. In advanced cases, atrial fibrillation may be evident. NT-proBNP levels may also be elevated. One study showed a median value of 742 pmol/L in dogs with PDA compared to a median value of 333 pmol/L in healthy dogs.

Echocardiography is the best way of diagnosing a PDA, which typically shows high velocity turbulence and continuous flow in the deep pulmonary artery prior to its bifurcation on color flow and continuous wave Doppler. Occasionally, severe pulmonary hypertension results in the reversal of flow to a right-to-left shunt. If a right-to-left PDA is suspected, a bubble study of the heart and abdominal aorta can be performed. If bubbles are seen in the abdominal aorta, then a reverse PDA would be confirmed.

**Treatment:** Treatment options include surgical ligation or a less invasive procedure—fluoroscopy-guided occlusion with a coil or a duct occluder. Possible negative consequences of the former include a rupture of the duct, whereas the latter carries the risk of coil migration and embolization in the pulmonary artery. Early repair is essential for long-term survival even though some dogs will survive for years with medical management alone. Closure of the defect is the optimal and most effective solution. The best surgical survival rates are found in younger dogs before significant morphological cardiac changes occur and prior



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to the development of major arrhythmogenic activity. Medical management is best determined by echocardiographic and clinical findings. In the future, a new technique may become available that would allow for the closure of the duct via thoracoscopy.

**References:**

Broaddus KD, Tillson DM. Patent ductus arteriosus in dogs. *Comp Con Educ Prac Vet* 2010;32:1-14.

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