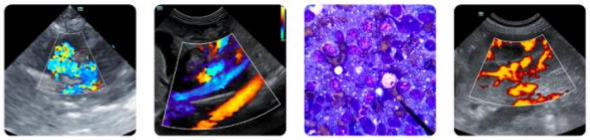

PATIENT

Stirling Proctor

SPECIES

Feline

BREED

Scottish Fold

SEX

Neutered Male

AGE

5 Years

WEIGHT

6.59 kg

INTERPRETED BY

Eric Lindquist, DMV, DABVP(CFM), Cert. IVUSS

IMAGING PERFORMED BY

Dr. Emily Kalenius

HOSPITAL NAME

Wilvet Salem

REFERRING VET

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INVOICE

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PRESENTING CLINICAL SIGNS

Presented to rDVM on 12/4. Diagnosed with UO and treated with indwelling catheterization. History of exercise intolerance but no heart murmur. rDVM treatments: simbadol convenia Cerenia Onsior Mirtazapine TD rDVM labwork 12/4: UA struvites and cocci. Normal USG. BUN 29, creat 1.6, other NSF rDVM labwork 12/5: rDVM radiograph interpretation (images not available) severely cardiomegaly no evidence CHF. RDVM TFAST: LA:Ao 2.2 Transferred to Wilvet 12/4 - has remained tachypneic and oxygen dependent. Now urinating. On maintenance rate IVF

Abnormal PE/Chem/CBC/UA Results: Intermittent gallop rhythm possible 1/5 sternal systolic murmur. Tachypneic harsh bronchovesicular sounds. BP wnl. SpO2 low 90s out of oxygen. ProBNP >1500 Radiology consult • exceptionally severe globoid cardiomegaly. • diffuse moderate bronchial change present throughout. • no obvious abnormalities in what is visible of the pulmonary vasculature, trachea and thoracic musculoskeletal system. Abdomen: no abnormalities Diffuse moderate bronco interstitial pattern. This is equivocal and could certainly indicate underlying feline asthma. Exceptionally severe globoid cardiomegaly. Comment: I cannot see any distinct pleural effusion radiographically

ULTRASONOGRAPHIC EXAMINATION OF THE HEART

FELINE CARDIAC PARAMETERS	**BODY WEIGHT** (kg)	**HR** (BPM)	**IVSd** (cm)	**LVIDd** (cm)	**LVWd** (cm)	**FS** (%)	**EF** (%)
NORMAL PARAMETER	------	150-240	0.3-0.6	1.0-2.1	0.25-0.6	35-67	80-100
PATIENT	6.59	200	0.59	1.35	0.70	53	87
FELINE CARDIAC PARAMETERS	**LA/AO** (M-mode)	**LA/AO HEART BASE** (Sisson)	**LAD** LA MAX 4 Chamber		**LVOT VEL.** (m/s)	**RVOT VEL.** (m/s)	**IVRT** (m/)
NORMAL PARAMETER	<1.5	1.6	0.7-1.7		<1.6	<1.3	40-60
PATIENT	1.0	1.1	--		1.37	1.4	NM
Adapted from June Boon,Veterinary Echocardiography,1998 Sisson D et al. JVIM 1991; 5: 232, Jacobs et al. Am J Vet Res 1985; 46:1705							

Cardiac Presentation

The cardiac presentation presented with normal volumes in the left atrium and left ventricle, however, right-sided volume overload was noted with right atrial and right ventricular enlargement. Enlarged ventricular septal defect was noted in this patient measuring approximately 1.1 cm. Slight pericardial effusion was also noted. Contractility appeared adequate. Septal free wall thickness were largely normal to slightly increased with some myocardial remodeling. Right sided over circulation is evident with right atrial, right ventricular and pulmonary artery dilation.

ULTRASONOGRAPHIC FINDINGS

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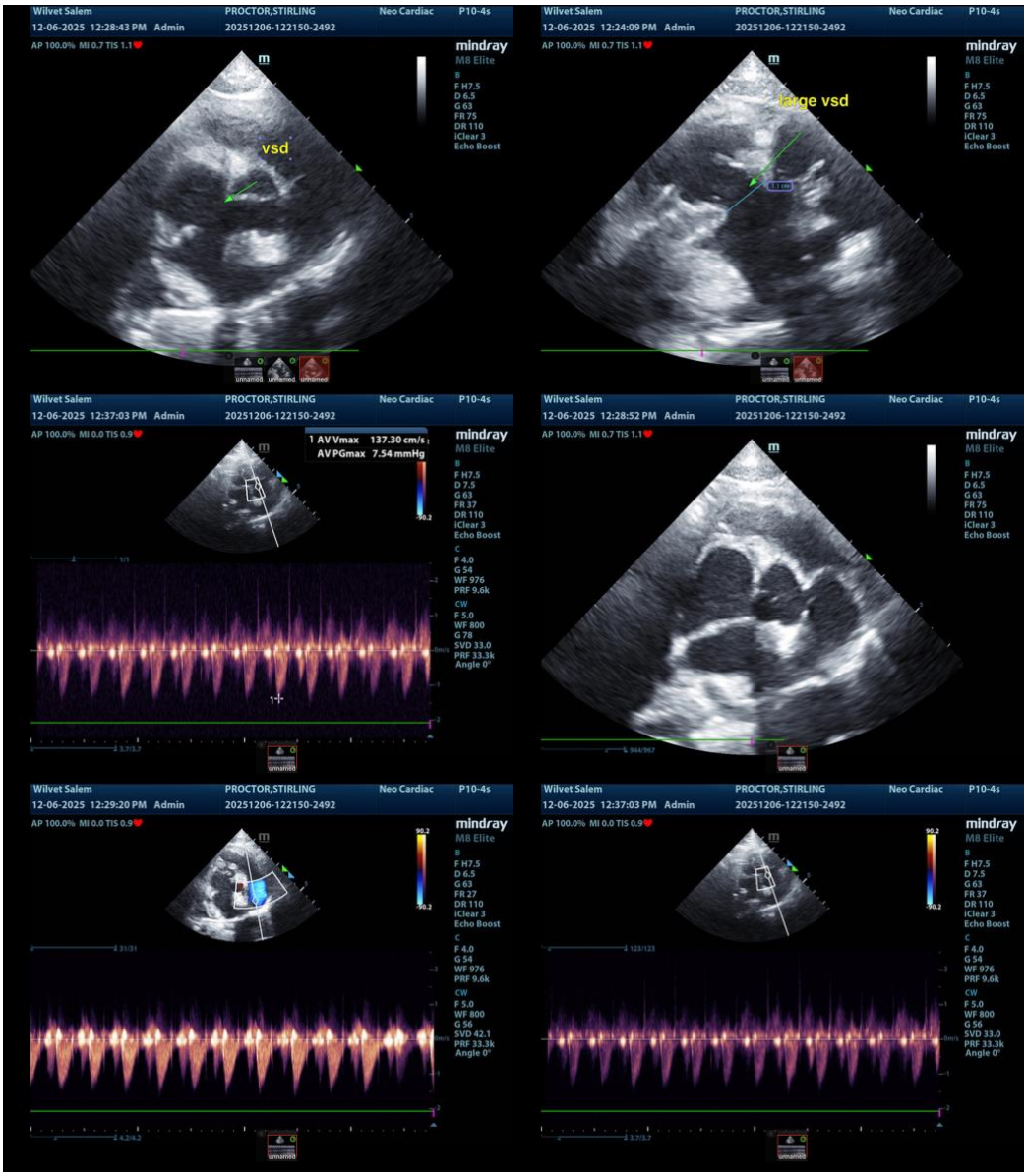
- Large ventricular septal defect with right-sided volume overload.

INTERPRETATION OF THE FINDINGS & FURTHER RECOMMENDATIONS

Abdominal sonogram is warranted to assess for evidence of passive congestion in the liver and ascites. Prognosis long term is guarded to poor. ACE inhibitor therapy and low-dose diuretic could be considered in this patient. Sildenafil is debatable in its utilization in these cases. Plavix therapy should be considered as well.



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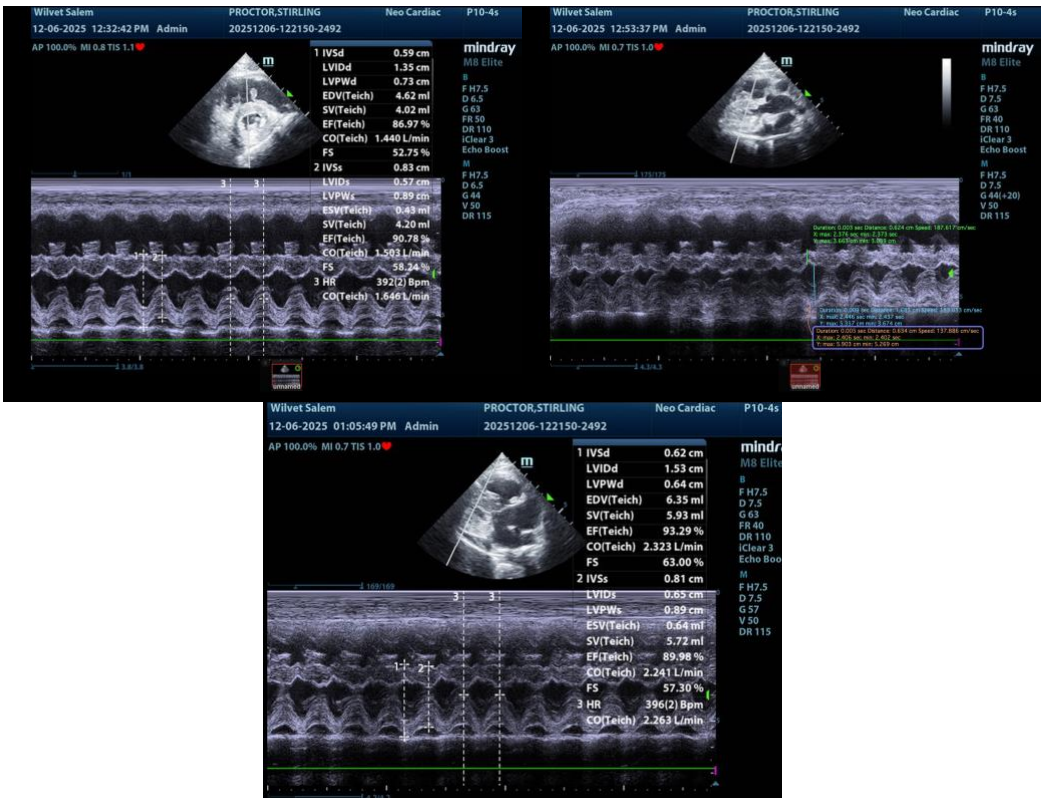


The information and recommendations provided are based on the images presented by the referring veterinarian/sonographer. No evaluation can be communicated regarding pathology that was not visible in the image/video clips provided.

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(CFM), Cert. IVUSS,

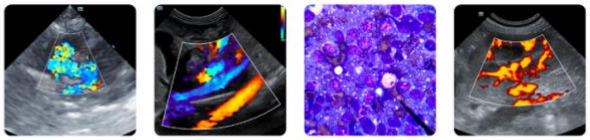
CEO, Owner, Founder -- SonoPath.com

info@SonoPath.com

Ventricular Septal Defect

http://www.sonopath.com/VSD

http://www.sonopath.com/EchoModler



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Description: Although ventricular septal defects (VSDs) account for less than 10% of congenital cardiac defects in dogs, they are far more common in cats, accounting for 56% of all cardiac defects. In dogs, the most frequently affected breeds are the West Highland White Terrier, Keeshound, Mongrel, Pinscher, French Bulldog, German Shepherd, Labrador Retriever, English Springer Spaniel, and Basset Hound.

The most common VSDs in dogs and cats are located in the basal septum. The left side of the defect is typically located just below the aortic valve, while the right side of the defect is located either in the inflow tract ("subcristal" or "perimembranous") or the outflow tract ("supracristal"). VSDs can occur in combination with other congenital abnormalities or as part of an endocardial cushion defect. Muscular defects are less common. The resulting hemodynamic consequence depends mainly on the size of the VSD. In small defects, the pressure difference between the left and right heart remains normal and the shunt flow moves at a high velocity. Depending on the shunt volume, VSDs can lead to volume overload in the left heart and pulmonary artery. A large shunt will typically result in a pressure equilibrium across both chambers; however, if pulmonary hypertension develops, then reverse shunting can occur. The latter occurs mainly in cats and only rarely in dogs. VSDs that develop in combination with other defects will have a completely different pathophysiology.

Clinical signs: Only a minority of patients with VSDs will show clinical signs. If the VSD is not too large (i.e., still resistive), a (usually loud) heart murmur can be auscultated on the right hemithorax. Large, non-resistive defects are not necessarily associated with a murmur.

Diagnostics: Perimembranous VSDs are easily seen on a right-sided apical five-chamber view just below the aortic valve. They can also be clearly seen on short axis views of the heart base. Supracristal defects are easier to see on short axis views than on regular five-chamber views. To identify muscular defects, careful tracking of the interventricular septum using multiple short axis views is necessary. They can also be seen on left apical views when the transducer is placed a bit further dorsally (i.e., not perfectly apical). In small, resistive defects, the shunt flow will be observed as a high-velocity systolic jet. The typical maximal flow velocity (Vmax) is approximately 5 m/s if normal pressure differences between the chambers are still present. Large defects do not show high-velocity flows. Slow left-to-right, right-to-left, or bidirectional flow can be displayed on a color Doppler ultrasound. In the latter case, the flow velocities are best displayed by PW-Doppler. Reversal of flow may also be demonstrated with a bubble study.

Treatment: The treatment depends on the pathophysiological status of the patient. Patients that have small defects with an insignificant flow do not require therapy; they can lead normal lives. Larger defects with marked left-sided and pulmonary arterial volume overload can be treated medically with the following: pimobendan (0.25-0.3 mg/kg PO BID); angiotensin-converting enzyme (ACE) inhibitors (enalapril at 0.5 mg/kg PO Q12-24hr or benazepril 0.25-0.5 mg/kg PO Q24hr); furosemide (1-2 mg/kg PO Q12-24hr, which can be increased incrementally, as needed); and/or spironolactone (0.25-2 mg/kg PO BID). Alternatively, they can be treated surgically via one of two options: pulmonary artery banding to increase right ventricular pressures and reduce shunting volumes, or closure of the defect via open heart surgery or a minimally invasive VSD closure.

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Bonagura JD, Lehmkuhl LB. Congenital heart disease. In Fox PR, Sisson D, Moise NS, eds. Canine and Feline Cardiology. Philadelphia, PA: WB Saunders; 1999:471-535.

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Saunders AB, Carlson JA, Nelson DA, et al. Hybrid technique for ventricular septal defect closure in a dog using an Amplatz Duct Occluder II. *J Vet Cardiol* 2013;15:217-24.