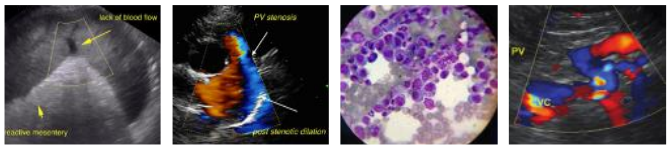
SonoPath
Clinical Sonography & Telecytology
EDUCATIONAL TELECONSULTATION SERVICES™



SonoPath.com
1-800-838-4268 info@sonopath.com

PATIENT

Moe Zisa

SPECIES

Feline

BREED

DSH

SEX

Intact Male

AGE

5 Months

WEIGHT

5.8 Pounds

INTERPRETED BY

Eric Lindquist, DMV DABVP, Cert. IVUSS

IMAGING PERFORMED BY

Dr. Travis Cerf

HOSPITAL NAME

Veterinary Center of Hardyston

REFERRING VET

Dr. Travis Cerf

INVOICE

14798

DATE

4/16/22

PRESENTING CLINICAL SIGNS

History: Palpable Thrill Employee Pet. During play will randomly lay on his side and breath very heavy (sometimes open mouth)

ULTRASONOGRAPHIC EXAMINATION OF THE ABDOMEN

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	--	NM	--	--	--	--	--

FELINE CARDIAC PARAMETERS	**LA/AO** (Boon)	**LA/AO HEART BASE** (Sisson)	**LA** 2D 4-chamber long axis AS to FW (Sisson) (cm)	**LVOT VEL.** (m/s)	**RVOT VEL.** (m/s)	**IVRT** (m/)
NORMAL PARAMETER	<1.5	0.88-1.79	0.7-1.7	<1.6	<1.3	40-60
PATIENT	1.15	--	--	--	--	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

A 0.5 cm ventricular septal defect was noted in this patient with right ventricular and right atrial enlargement, owing to secondary tricuspid insufficiency. Aortic insufficiency was also present. Mitral insufficiency was trivial yet persistent. The left atrium and left ventricle were of normal size. The ventricular septal and free wall thicknesses were fairly normal as was contractility, however, right sided pathology is significant.

ULTRASONOGRAPHIC FINDINGS

- Ventricular septal defect or tricuspid insufficiency and aortic insufficiency

INTERPRETATION OF THE FINDINGS & FURTHER RECOMMENDATIONS

Further pulmonic outflow imaging warranted to assess for pulmonic stenosis yet structurally the pulmonic valve appears unremarkable. Ventricular septal defect is non-correctable and left to right. Very guarded long-term prognosis given the secondary changes noted already in the right heart at 5 months of age. Atenolol therapy could be considered as a palliative measure; however, this patient is at risk for sudden death.

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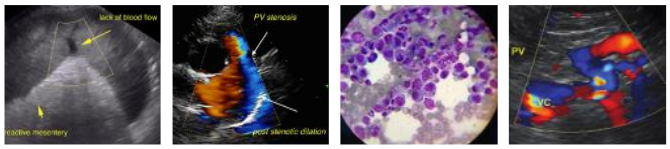

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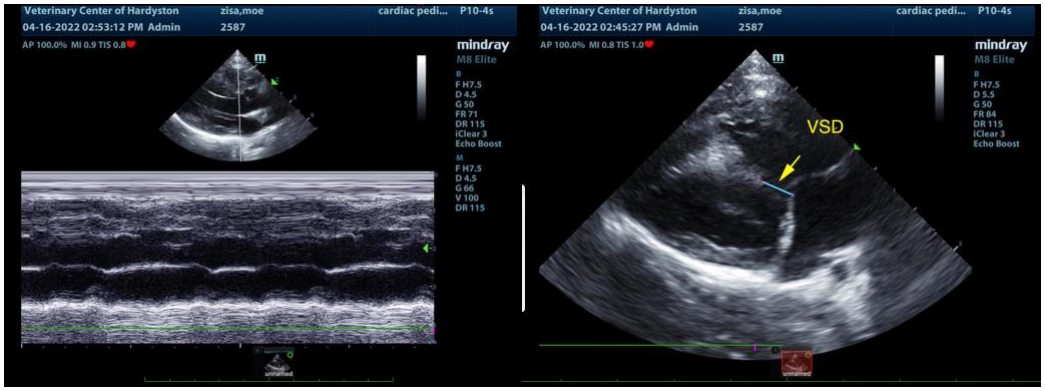

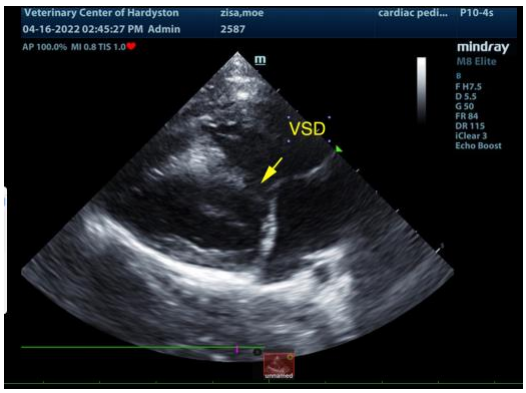

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The information and recommendations provided are based on the images presented by the referring veterinarian. No evaluation can be communicated regarding pathology that was not visible in the image/video clips provided.

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.

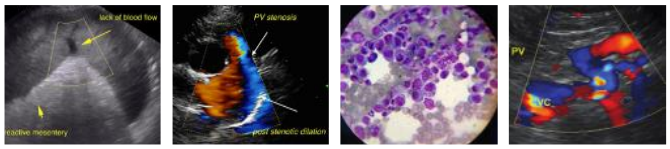
Eric Lindquist, DMV, DABVP, Cert. IVUSS, CEO of SonoPath.com
info@SonoPath.com

Ventricular Septal Defect

http://www.sonopath.com/VSD

http://www.sonopath.com/EchoModler

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



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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.

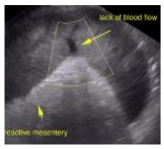
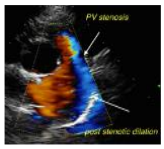
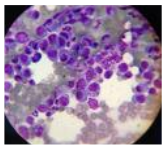
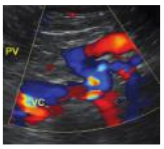
The most common VSDs in dogs and cats are located in the basal septum. The left side of the defect is typically located just below the aortic valve, while the right side of the defect is located either in the inflow tract ("subcristal" or "perimembranous") or the outflow tract ("supracristal"). VSDs 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.

References:

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