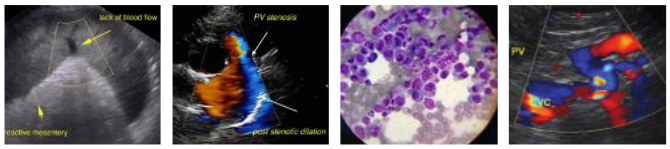
SonoPath
Clinical Sonography & Telecytology
EDUCATIONAL TELECONSULTATION SERVICES™



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

PATIENT

Kona Heckmum

SPECIES

Canine

BREED

Shiba Inu

SEX

Male

AGE

4 Months

WEIGHT

9 Pounds

INTERPRETED BY

Eric Lindquist, DMV DABVP, Cert. IVUSS

IMAGING PERFORMED BY

Dr. Jessie Evoniuk

HOSPITAL NAME

State Avenue VC

REFERRING VET

Dr. Jessie Evoniuk

INVOICE

22091

DATE

4/18/23

PRESENTING CLINICAL SIGNS

History: Referred by High Plains Veterinary Clinic with a significant history of born with a Grade 4 Heart Murmur no other concerns at home, no current meds

ULTRASONOGRAPHIC EXAMINATION OF THE HEART

CANINE CARDIAC PARAMETERS	MR VMAX (m/s)	TR VMAX (m/s)	LA/AO (Boon method)	LA/AO (Heart Base; Swe)	FS (%)	EF (%)	EPSS (cm)
NORMAL PARAMETER	4.5-5.5	<2.7	1.3	<1.3	28-40	40-100	<0.6
PATIENT	--	--	1.15	1.4	40	90	0.1
CANINE CARDIAC PARAMETERS	**HR** (BPM)	**AV VMAX** (m/s)	**PV MAX** (m/s)	**BODY WEIGHT** (kg)	**LA** 2D short axis Base view (cm)	**LVIDd** Avg; 2D and m-mode short axis (cm)	**LVIDs** Avg; 2D and m-mode short axis (cm)
NORMAL PARAMETER	50-100	0.7-1.7	0.7-1.6				
PATIENT	--	--	1.2	--	2.0	2.4	--

Cardiac Presentation

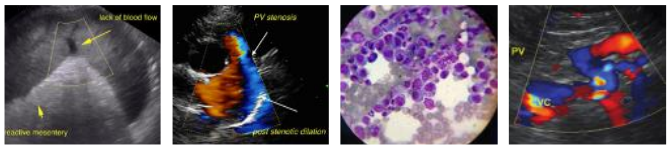
The echocardiogram in this patient demonstrated normal **left atrial** size based on 3 separate methods of LA evaluation. The cranial and caudal **mitral** valve leaflets presented normal linear structure, extension in systole, and union in diastole with normal kinesis. The **left ventricle** presented thicknesses with linear contour and was not dilated nor restricted. The **myocardium** presented normal echogenicity without subjective evidence of significant fibrotic or ischemic disease. **Contractility** of the ventricular walls was adequate and in normal range for this patient evidenced by the fractional shortening measurement and subjective evaluation of the different regions of the myocardium. The left ventricular septum in this patient revealed a membranous septal defect measuring approximately 5.0 mm with thickened irregular tissue. Color flow doppler demonstrated left to right shunting. The **right atrium** appeared mildly enlarged. No evidence of masses was noted. **Tricuspid** valve appeared slightly clubbed and a shorted septal leaflet was noted. Some level of tricuspid dysplasia may also be an issue. The **right ventricle** was unremarkable. **Pulmonary outflow** tract was unremarkable. Pulmonic outflow spectral doppler revealed a pulmonic velocity of 1.2 m/s. No visible **pericardial** or free pleura fluid was noted. The cranial **mediastinum and pericardial and extra-cardiac regions** were free of masses in the visible window.

ULTRASONOGRAPHIC FINDINGS

- Ventricular septal defect, compensated

INTERPRETATION OF THE FINDINGS & FURTHER RECOMMENDATIONS

I cannot completely rule out other congenital anomalies yet not suspected. Further definition with a



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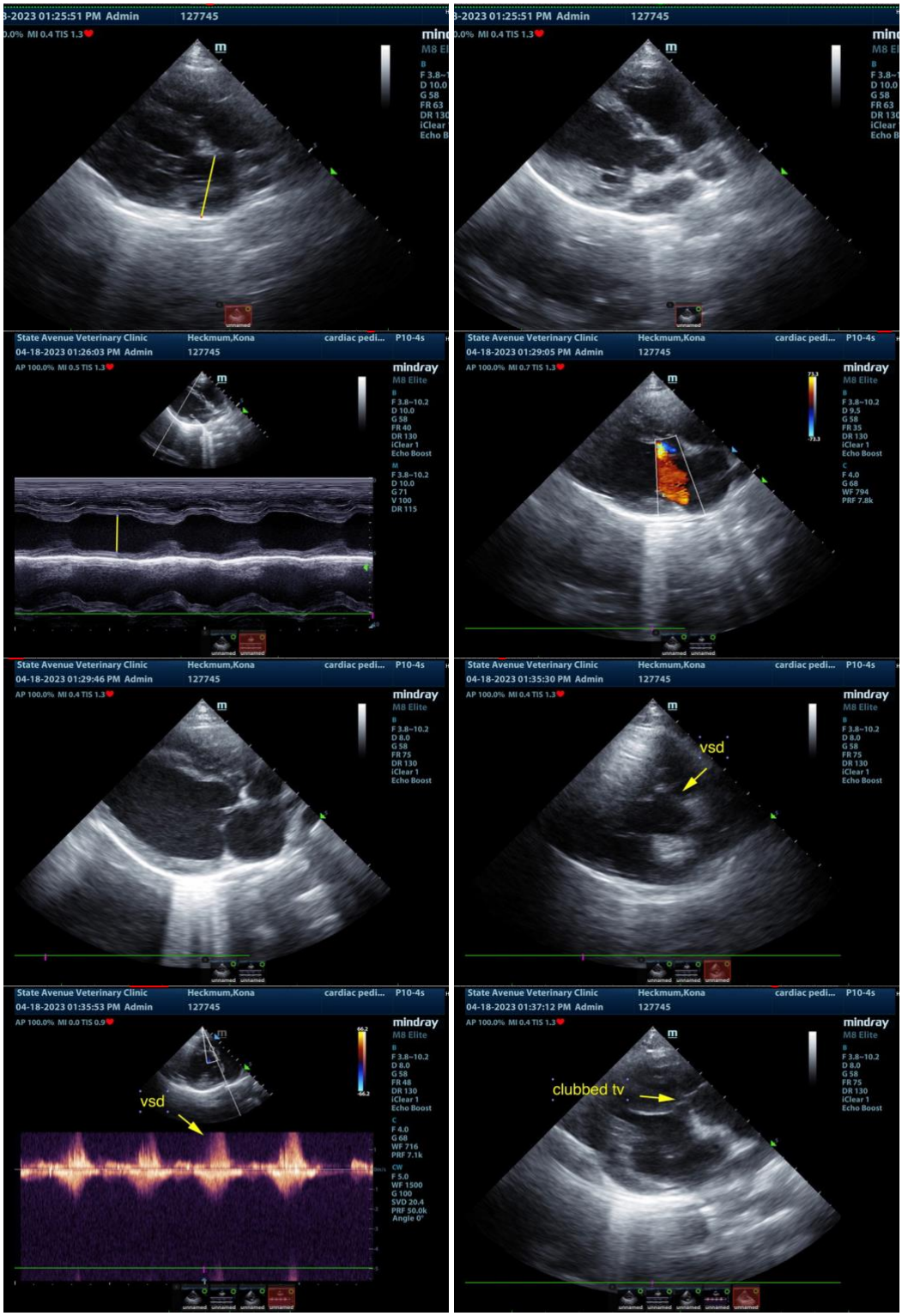
cardiologist would be ideal, however, this appears compensated at this time. The breeding line should be evaluated for similar pathology. Further doppler evaluation of the deep pulmonary artery, pulmonic valve and aortic outflow is indicated, as well as the tricuspid valve. Recheck echocardiogram in 3 months.



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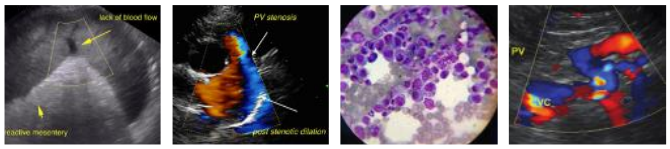

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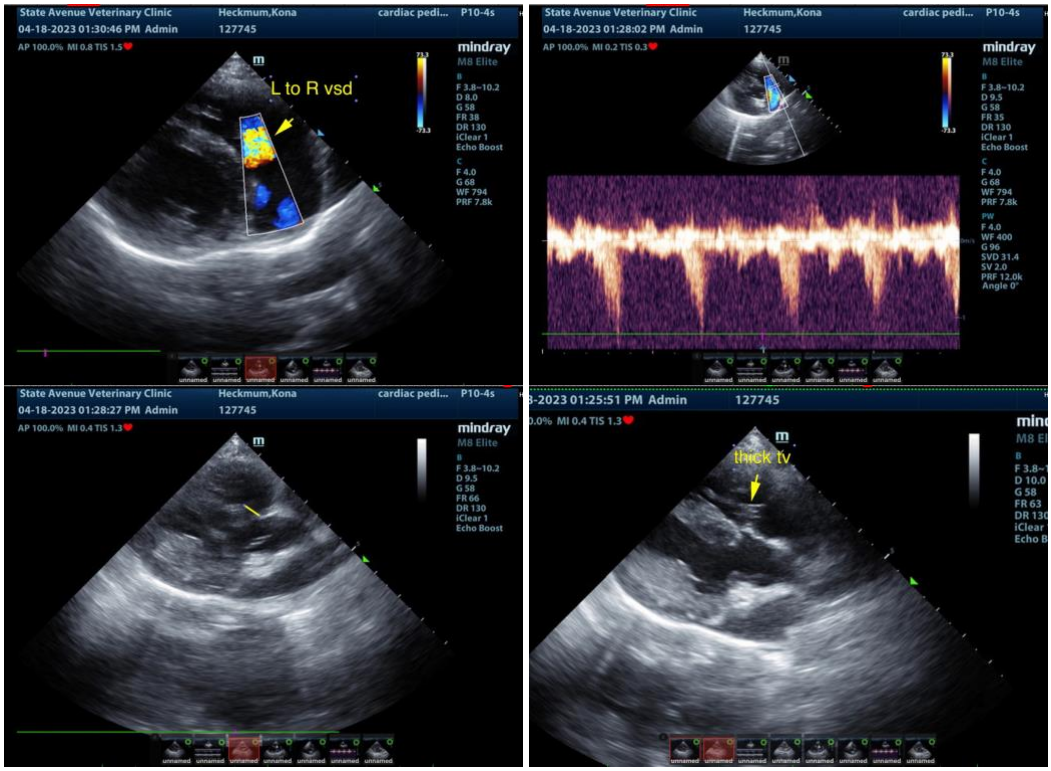

The information and recommendations provided are based on the images presented by the referring veterinarian. No evaluation can be communicated regarding pathology that was not visible in the image/video clips provided.

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 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.

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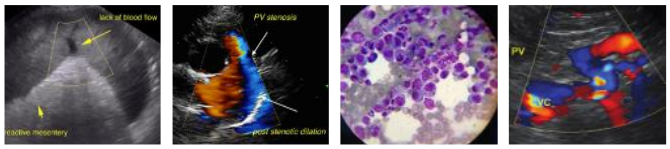

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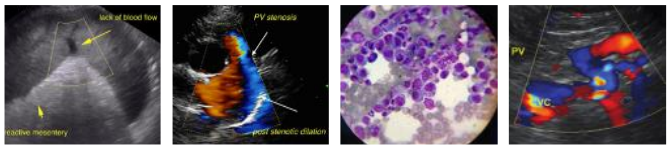
The most common VSDs in dogs and cats are located in the basal septum. The left side of the defect is typically located just below the aortic valve, while the right side of the defect is located either in the inflow tract ("subcristal" or "perimembranous") or the outflow tract ("supracristal"). VSDs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