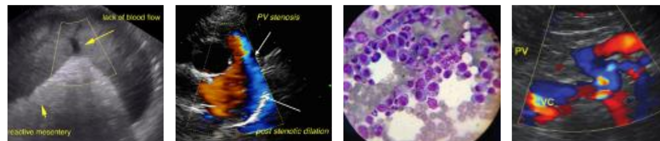
SonoPath
Clinical Sonography & Telecytology
EDUCATIONAL TELECONSULTATION SERVICES™



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

PATIENT

Mocha Samaha

SPECIES

Canine

BREED

Chawaw

SEX

Female

AGE

7 Years

WEIGHT

2.60

INTERPRETED BY

Eric Lindquist, DMV
DABVP, Cert. IVUSS

IMAGING PERFORMED BY

Doctor Valentina

HOSPITAL NAME

The Veterinary Surgery

REFERRING VET

Valentina Fresta

INVOICE

21888

DATE

4/1/23

PRESENTING CLINICAL SIGNS

History: Presented for disorexia and lethargy since two days during the estrus The patient had another similar episode last year during the season time where she exhibited disorexia, and respiratory distress. Since then, she has been on Fortekor and Pimobendan and the recovery was fast at that time with complete resolution of the clinical signs . At the clinical examination ,sternal recumbency .MMC are slightly congested and moist .The abdominal palpation reveals tense abdomen in particular on the xiphoid area .Heart reveals a murmur 2/6 on the left side of the thorax. RR 61.HR 140. the lung sound is clear. Temp 39.2

Abnormal PE/Chem/CBC/UA Results: The CBC reveals pistrinosis .The biochemistry reveals parameters in the normal range .The CPL test is negative. The x-ray reveals an enlargement of the right side of the heart .Lung pattern has increased radiopacity. After the butorphanol the patient started to feel better and ate all the food. RR now 24

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	--	--	1.0	1.5	--	--	NM
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	--	--	.70	--	--	1.2	--

Cardiac Presentation

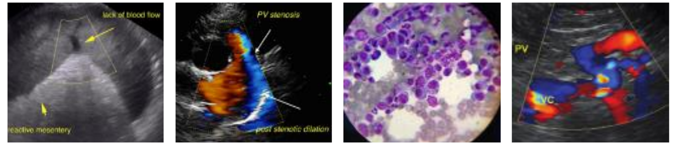
The cardiac presentation in this patient presented normal left sided volume and left sided contractility. However, a large atrial septal defect was noted in this patient, creating a right atrial and right ventricular volume overload, as well as pulmonary artery volume overload. This is present in multiple views. Hepatic vein dilation was minor.

ULTRASONOGRAPHIC FINDINGS

- Atrial septal defect with right sided volume overload, fairly compensated at this time, however, I do not recommend reproduction of this individual given that this is a congenital defect.

INTERPRETATION OF THE FINDINGS & FURTHER RECOMMENDATIONS

I recommend continuation of current protocol and reassessment of the clinical signs. Sildenafil (1



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mg/kg BID) could be considered as an adjunctive therapy, however, only if necessary. If diuretic is to be utilized, Spironolactone 1-2 mg/kg BID, could be considered, yet not likely necessary at this point.

Come join us for the SonoPath Summit on Cardiology July 10-12, 2023
https://sonopath.com/sonopath-2023-summit/

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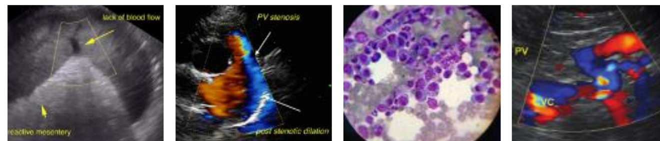

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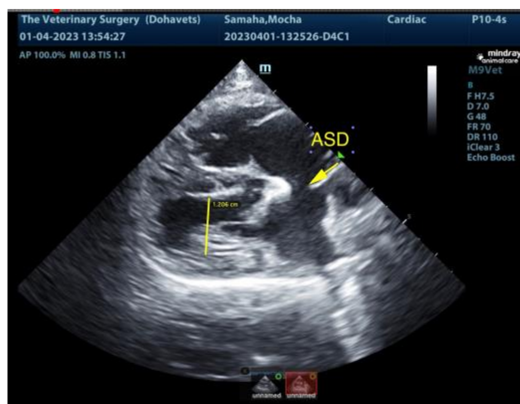


The information and recommendations provided are based on the images presented by the referring veterinarian. No evaluation can be communicated regarding pathology that was not visible in the image/video clips provided.

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

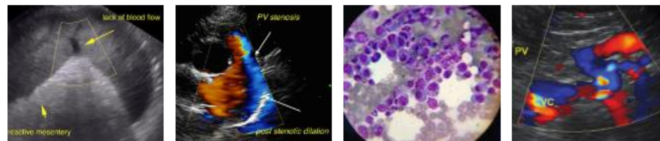
Atrial Septal Defect

http://www.sonopath.com/ASD

http://www.sonopath.com/EchoModler

Description: The atrial septum develops from the septum primum and the septum secundum. Failure of appropriate development and fusion results in a septal defect. There are four different types of atrial septum defects (ASDs): ostium primum, ostium secundum, sinus venosus, and coronary sinus. Dispositions to ASD have been reported in the following breeds: Standard Poodles, Cavalier King Charles Spaniels, German Shorthair Pointers, and Golden Retrievers, as well as shorthair cats. The ostium secundum type occurs much more frequently than the ostium primum type. The sinus venosus and coronary sinus types do not play a role in small animal cardiology.

Note: A patent foramen ovale is not classified as an ASD because it is a congenital defect caused by a significant increase in right ventricular pressure that prevents the foramen from closing.



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Under normal conditions (i.e., in the absence of concomitant defects), the flow across an ASD depends mainly on the difference in ventricular compliance. Because the right ventricle has a thinner wall, it usually fills more easily than the left ventricle. This causes left-to-right shunting across the ASD. Whereas small ASDs do not cause any hemodynamic impairment, larger defects lead to right heart volume overload and pulmonic “pseudo-stenosis” due to the additional shunting volume that passes through the pulmonic valve. In turn, pulmonary hypertension (PHT), as well as secondary tricuspid insufficiency, may develop. Increases in right ventricular pressure may result in PHT, which can lead to a reversal of the shunt.

Clinical Signs: Patients with an ASD are frequently asymptomatic, but some can develop congestive heart failure (CHF). The ASD itself does not cause a heart murmur, as the gradient across the defect is usually too low to cause turbulence. Yet, due to the pulmonic pseudo-stenosis that is associated with larger defects, a relatively soft murmur at the left heart base may be present.

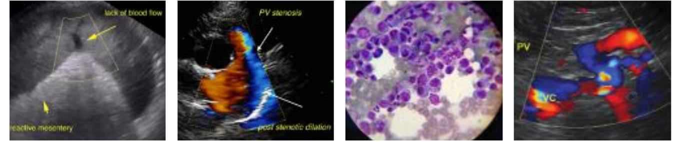
The prognosis depends on the size of the defect. Smaller defects do not cause hemodynamic impairment, whereas larger ones can result in CHF.

Diagnostics: The ASD can usually be seen on a 2D Echo using a right parasternal four-chamber view or a short axis view at the level of the heart base. Flow across the defect can be visualized using color Doppler imaging. Shunt reversal is easily visible when one employs a bubble study with agitated saline or hetastarch.

Treatment: Medical treatment is aimed mainly at reducing the right ventricular volume overload and resolving CHF; it is only indicated in patients with larger, hemodynamically significant defects. Cases are deemed hemodynamically significant when there is right heart volume overload and one can detect increased right ventricular outflow velocities. Such patients can be treated by preload reduction with diuretics and angiotensin-converting enzyme (ACE) inhibitors. If there is evidence of PHT or right ventricular systolic dysfunction, pimobendan (0.25 mg/kg BID) should be administered. Transcatheter or surgical closure may be indicated when hemodynamically significant defects are present; however, transcatheter closure is not always possible since its viability depends on the exact location and size of the defect, and whether there is sufficient tissue around the defect to keep the closure device in place. Although surgical closure is theoretically possible in all types and sizes of ASDs, it requires a cardiopulmonary bypass and carries all the risks of open heart surgery. Studies comparing open heart surgery and transcatheter closure are, however, lacking, as both procedures are rarely performed.

Small defects do not require therapy.

References:



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