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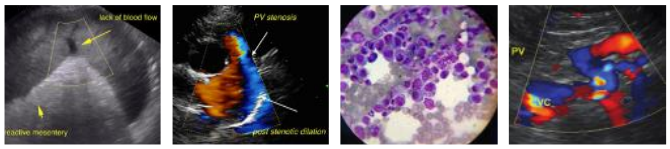


SonoPath.com
1-800-838-4268 info@sonopath.com

PATIENT

Pearl Brown

SPECIES

Canine

BREED

Terrier

SEX

Spayed Female

AGE

16 Years

WEIGHT

3.7 kg

INTERPRETED BY

Eric Lindquist, DMV DABVP, Cert. IVUSS

IMAGING PERFORMED BY

Dr. Callihan

HOSPITAL NAME

Animal Emergency Care

REFERRING VET

Dr. Loeffler

INVOICE

17743

DATE

10/16/22

PRESENTING CLINICAL SIGNS

History: Long history of coughing, has been treated with Enalapril and furosemide ~ 2 yrs, recently added pimobendan which seemed to help the cough. Owner says pt had a thoracentesis about 6 weeks ago due to pleural effusion but there is no information about that in the accompanying records. Pt presented on ER Friday for acute vomiting, lethargy and inappetence Regarding the cough, has had trials in past with prednisone and antibiotics with no notable improvement.

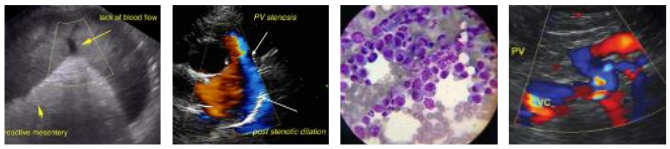
Abnormal PE/Chem/CBC/UA Results: Radiographs show consolidation in left cranial ventral lung field, no overt cardiomegaly or congestion to my eye- though rads have not been submitted for interp Bloodwork showed acute renal failure but she has responded well to therapy so far, values are improving and she is eating, and we are continuing diuresis and GI support She has remained hypotensive through hospitalization though peripheral pulses are normal at this time and with improved flow sounds on doppler. Have stopped all cardiac meds pending ultrasound report

ULTRASONOGRAPHIC EXAMINATION OF THE HEART & ABDOMEN

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	5.0	2.8	1.0	1.2	36	69	0.14
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	--	1.00	.62	--	1.54	1.57	--

Cardiac Presentation

The echocardiogram in this patient demonstrated normal **left atrial** size based on 3 different LA measurement methods. Chamber volumes and echogenicity were normal. The cranial and caudal **mitral** valve leaflets presented vegetative thickening consistent with endocardiosis. Doppler indicated fairly centralized and mild insufficiency. 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 outflow** tract demonstrated normal laminar flow and subjective structural integrity. The **right atrium** and auricle revealed normal size, structure and content. No evidence of masses was noted or chamber overload. **Tricuspid** insufficiency was noted. The **right ventricle** was of normal size



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(1/3 diameter of LV), chordae structure, myocardial echogenicity and thickness. **Pulmonic** tract assessment revealed normal valve structure, laminar flow, and diameter (approx.1:1 pa/ao ratio). No visible **pericardial** or free pleura fluid was noted. No echographically detectable evidence of infiltrative disease was visible. The cranial **mediastinum and pericardial regions** were free of masses in the visible window.

Urinary System

The **urinary bladder**, trigone, and pelvic urethra presented normal thicknesses and normal tone. The ureters were not visible which is normal. No uroliths or sediment were visualized, and anechoic urine was present. No evidence of inflammatory or neoplastic changes were noted. Ureteral papillae were normal.

The **kidneys** revealed largely normal size and structure, corticomedullary definition and ratio (cortex 1/3 of medulla) were essentially maintained with some mild age-related loss of curvilinear patterns regarding the capsule and C/M junction. The cortices presented largely uniform texture with some increased echogenicity expected for his age patient. Medullary structure differed distinctly from that of the cortex and no evidence of pelvic dilation was present. Mineralization was present in the kidneys. The left kidney measured 3.15 cm. The right kidney measured 3.83 cm.

Adrenal Glands

Both **adrenal glands** were visualized and recognized as having normal shape, size, position and echogenicity for this breed. The phrenic vasculature, glandular echogenicity and detail were unremarkable. Capsule, cortex, and medullary definition were normal for this age patient. The left adrenal gland measured 0.37 cm. The right adrenal gland measured 0.6 cm at the cranial pole and 0.4 cm at the caudal pole.

Spleen

The **spleen** revealed multifocal hyperechoic lipogranulomatous changes, not pathological.

Liver

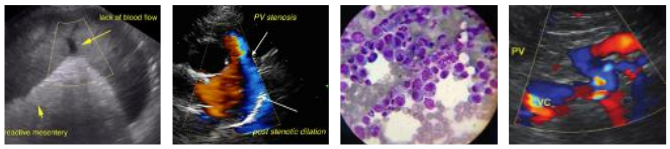
The **liver** images submitted revealed subjectively normal liver size, contour, and structure. Parenchymal echogenicity was naturally coarse and hypoechoic to the spleen. Vascular and biliary tracts were of normal volume with no evidence of congestion. The hepatic veins were not dilated. A comet tail lung pattern was noted through the diaphragm indicative of alveolar disease.

The **gallbladder** was mildly over distended with suspended and dependent debris, yet not to the level of emerging mucocele, yet sludge appears to be mildly excessive. No adjunctive inflammation was noted.

Gastrointestinal

Examination of the **gastrointestinal tract** revealed a stomach and intestine free of stasis, of normal wall thickness, acceptable curvilinear mural detail, and peristaltic activity. Small and large intestine demonstrated normal luminal chyme and stool consistency respectively. No obstructive or overt infiltrative disease was noted. No associated abnormal lymphatic activity was noted.

Pancreas



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The base and limbs of the **pancreas** were observed to be largely isoechoic to surrounding omental fat. Pancreatic duct and capsular contour were acceptably normal and parenchyma respected normal curvilinear patterns. No overt evidence of active inflammatory or neoplastic disease was noted.

Other

The right **thorax** revealed lung consolidation, consistent with pneumonitis. Mild potential for underlying thoracic neoplasia.

There is a 1.5 cm x 0.8 cm saddle **thrombus** noted in this patient.

ULTRASONOGRAPHIC FINDINGS

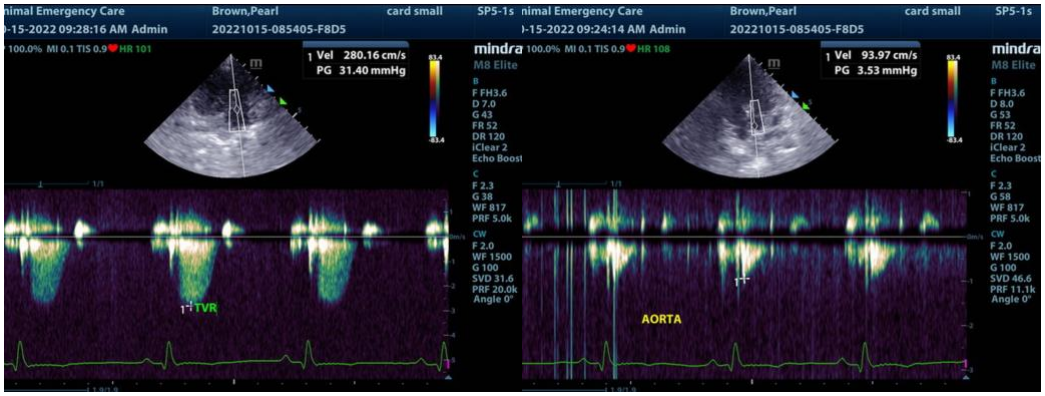
- Stage B-1 valvular disease
- Mild mitral and tricuspid insufficiency- no evidence of clinical cardiac disease
- Lung consolidation in the right thorax
- Age-related renal changes with mineralization
- Lipogranulomatous changes in the spleen
- Mild excessive gallbladder debris
- Thrombus- Full coagulation panel is warranted.

INTERPRETATION OF THE FINDINGS & FURTHER RECOMMENDATIONS

The tricuspid insufficiency velocity is borderline for early pulmonary hypertension yet not a clinical issue. The heart presents normal left atrial size and therefore, is not the cause of the cough. The respiratory disease is likely increasing pulmonary pressures, yet not to the level of treatable pulmonary hypertension. Primary respiratory protocol is recommended based on thoracic radiographs +/- thoracic CT in this patient.

No evidence of abdominal disease linked to the clinical signs. I recommend focusing on the primary lung pathology in this patient based on radiograph and/or CT findings of the chest.

Regarding the thrombus, Plavix therapy or other dissolution approach may be considered. Given the thrombus presentation, pulmonary thromboembolic disease should also be considered as a potential.



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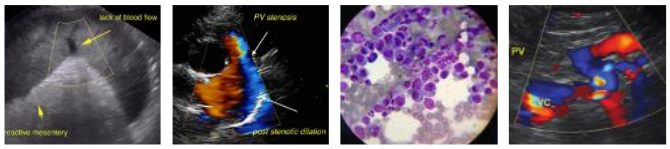

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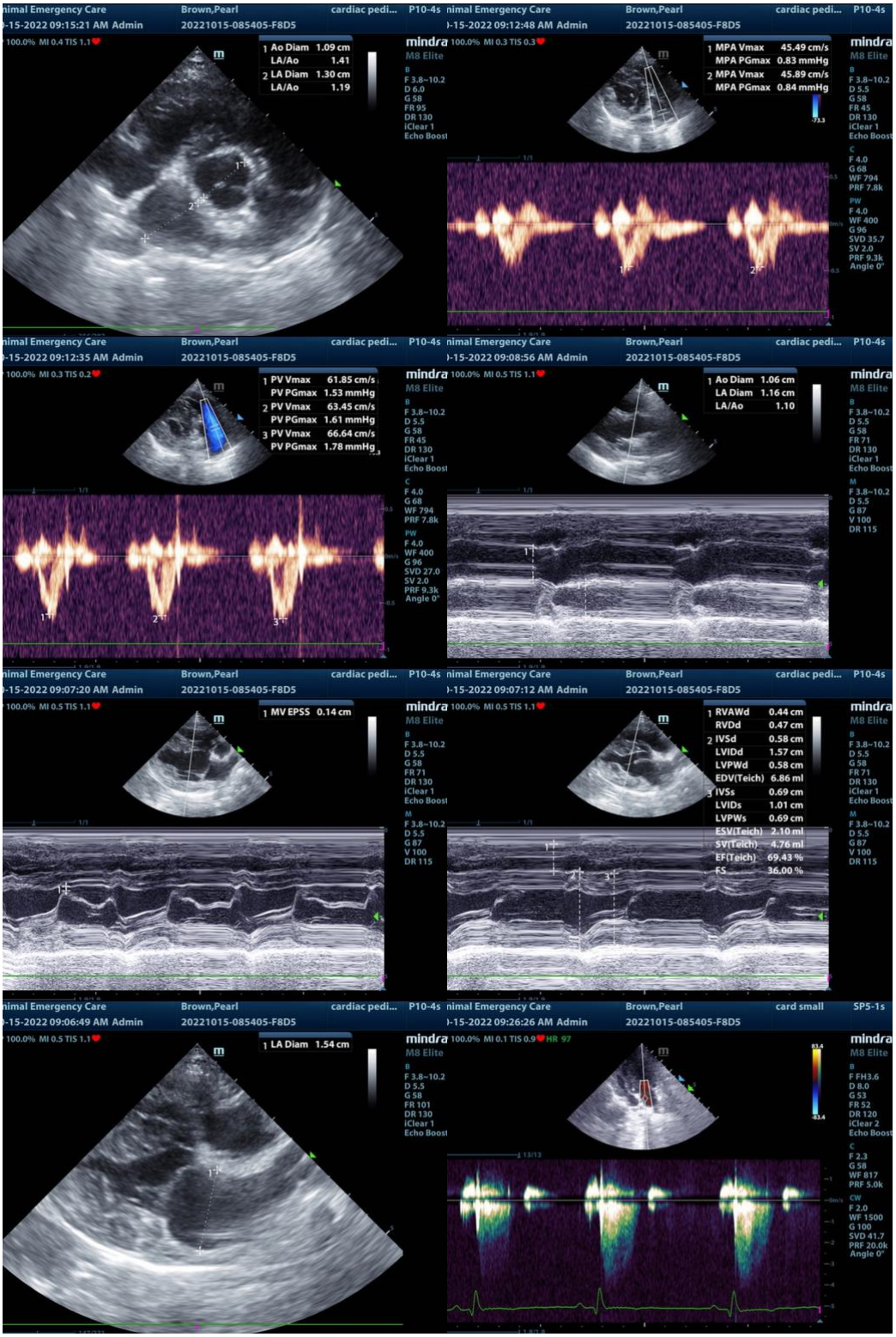

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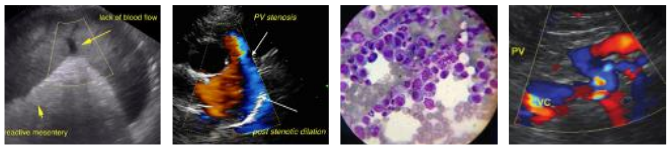

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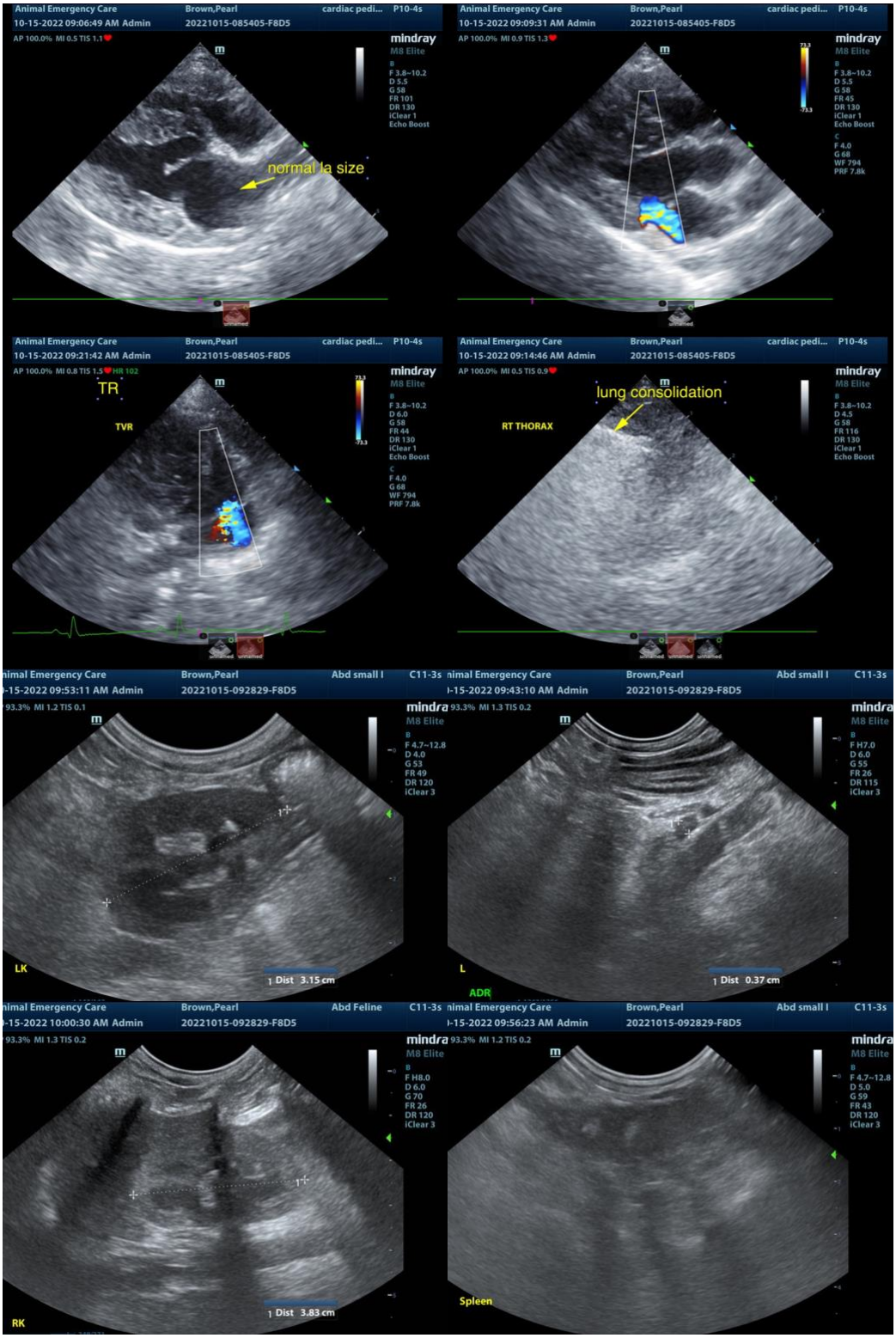

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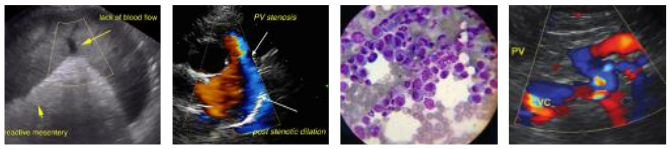

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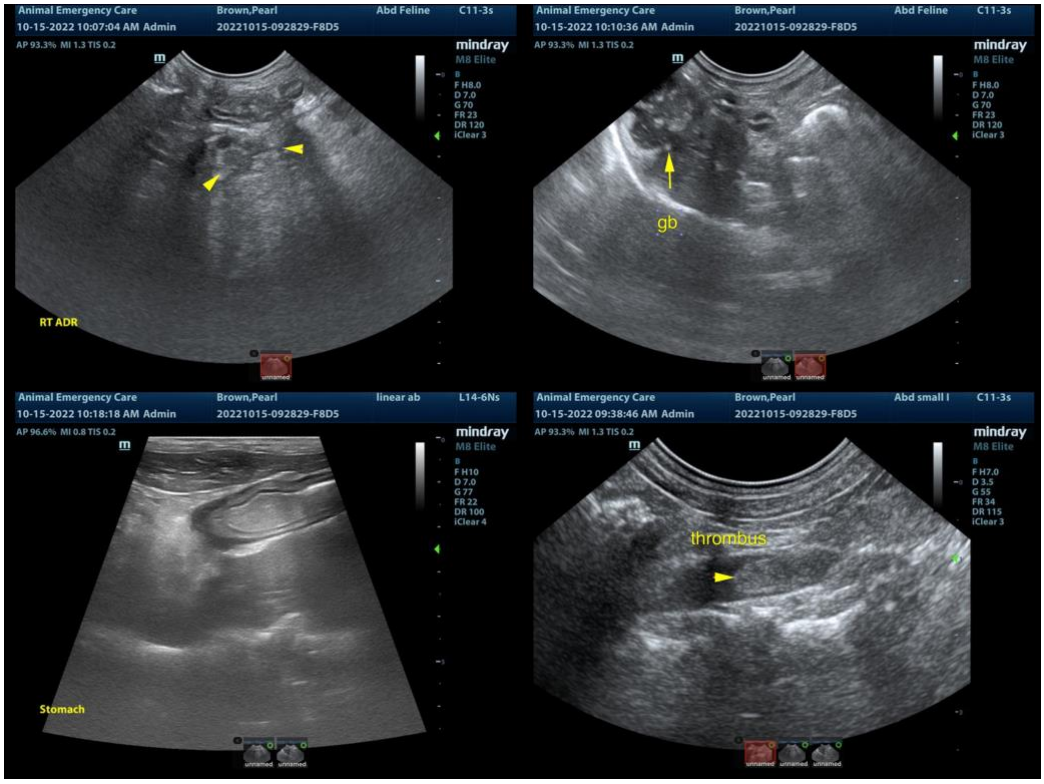

The information and recommendations provided are based on the images presented by the referring veterinarian. No evaluation can be communicated regarding pathology that was not visible in the image/video clips provided.

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

Mitral & Tricuspid Valve Disease

http://www.sonopath.com/

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SonoPath.com was founded in 2007 by Eric Lindquist, DVM, DABVP to provide a source of sonographic pathology information accessible to veterinarians of all levels of canine, feline, and exotic medicine.

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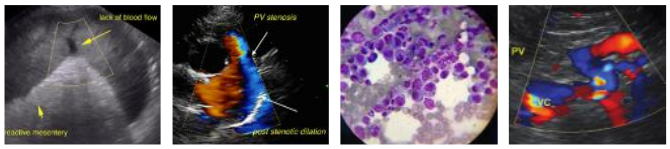

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Valve Disease

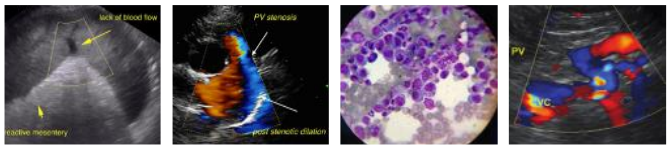
Description: Myxomatous valvular degeneration refers to a sterile degenerative disease that affects middle-aged and older dogs. The mitral valve apparatus includes the left ventricular papillary muscles, chordae tendinae, valvular annulus, and anterior and posterior leaflets. The anterior leaflet is continuous with the aortic outflow tract. The accumulation of mucopolysaccharides within the spongiosa and fibrosa layers of the leaflets creates a vegetative nodular appearance. This vegetative pathology may also be the result of bacteremia due to dental disease or a different source of bacterial pervasion. Lengthening of the chordae tendinae occurs secondary to any excessive turbulent forces that might transpire. Chondrodystrophic breeds are overrepresented and typically have collapsing trachea and intervertebral disc disease. Cavalier King Charles Spaniels are paradigmatic of this phenomenon; they develop mitral valve disease (MVD) at an early age. MVD prevalence can be as high as 33% in toy breeds over 10 years of age and is the most common cardiac disease in dogs (it accounts for 75-80% of all cases). MVD has also been reported in German Shepherds; they demonstrate a more rapid progression of the disease in comparison to other breeds.

Distortion of the mitral valve leaflets prevents normal cooptation of blood resulting in regurgitative flow of the stroke volume into the left atrium. The lengthening and rupture of the chordae can lead to leaflet prolapse into the atrium and the eventual inversion of the flail leaflet into the atrium, which will in turn exacerbate the regurgitation. Chordae rupture often occurs suddenly, leading to the rapid onset of congestive heart failure (CHF), as the myocardium does not have adequate time to hypertrophy and compensate for the defect. (A small amount of regurgitation can be well tolerated if it progresses more slowly over time.) The result is volume overload of the left atrium and left ventricle, which gives rise to eccentric hypertrophy. Eccentric hypertrophy causes dilation of the mitral annulus, which further complicates the myocardial left atrium and left ventricle stretch. Myocardial oxygen deprivation due to poor coronary perfusion (stretch and catecholamine stimulation) leads to myocardial cell death, replacement fibrosis, and myocardial dysfunction. This pathological progression is known as “overload cardiomyopathy.”

Clinical Signs: The most common sign of MVD is a progressive cough; it is due to left-sided volume overload, which in turn precipitates pulmonary edema and left atrial bulging at the left mainstem bronchus. Respiratory distress ensues gradually in chronic cases or rapidly in cases of primary chordae rupture. Exercise intolerance, syncope, ascites, weight loss, and anorexia are also commonly observed. A physical exam will often reveal an audible murmur best heard over the left cardiac apex. The murmur audibility and length into systole typically corresponds to the degree of mitral regurgitation but not necessarily to the severity of the cardiac status. Concurrent systemic hypertension may induce progression of the disease thereby necessitating systemic blood pressure measurements during medical management.

A challenging situation occurs when the practitioner must determine whether the origin of a cough is respiratory (e.g. tracheal collapse, chronic bronchitis, COPD) or cardiac (e.g. pulmonary edema, mainstem bronchus pressure). A cough history of months or years, normal to high body scores, and normal heart rates with sinus arrhythmia tend to support chronic airway disease. In cases of MVD, however, the disease tends to be progressive: the body score is lower, the heart rate is higher, and there is a possibility of pathological arrhythmias. Yet, both bronchial disease and MVD can be present simultaneously, and advanced diagnostics, such as ultrasound examination, are necessary to distinguish which culprit is inciting the clinical signs.

Diagnostics: Ascertaining NT-proBNP levels may help to determine whether dyspnea is respiratory or cardiac in origin and provide information that can assist with the early detection of cardiac disease in



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subclinical patients, especially in high-risk breeds, such as Cavalier King Charles Spaniels, Poodles, and Cocker Spaniels.

Radiographic findings may reveal a vertical or bulging caudal cardiac waist, hilar edema, generalized cardiomegaly, elevation of the left mainstem bronchus, and right-sided enlargement in advanced cases where pulmonary hypertension or tricuspid disease is also present.

Echocardiographic findings allow the practitioner to assess precisely cardiac function under pathological circumstances. Moreover, they enable an evaluation of the myocardial response at the time of the exam and help practitioners continue to quantify that response over time while patients are undergoing therapy.

Treatment: The following treatment options are based on the ACVIM consensus statement regarding MVD.

Stage A: There is a high risk of cardiac disease, but as there are no clinical signs, no specific therapy—medical or dietary—is indicated. A radiograph and an echocardiogram should be conducted in one year or earlier if the patient is a large-breed dog as MVD may progress faster.

Stage B: Heart disease is present.

B1: There is a murmur, but no chamber enlargement. Treatment is the same as for stage A.

B2: There is a murmur with left atrial and left ventricular enlargement, but the patient is asymptomatic. Start angiotensin-converting enzyme (ACE) inhibitors (enalapril at 0.5 mg/kg PO Q12-24hr or benazepril 0.25-0.5 mg/kg PO Q24hr). Consider beta blockers and mild sodium restriction.

Stage C: There are past or current clinical signs of heart failure.

Acute CHF:

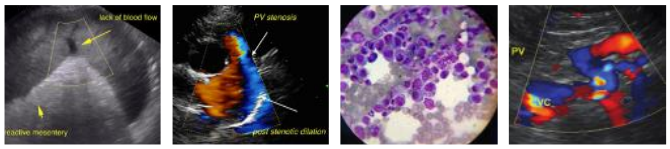
- Lasix 2 mg/kg IV or IM hourly to a total dose of 8 mg/kg until the respiratory rate has normalized. Alternatively, for cases of life-threatening pulmonary edema, administer as a CRI (1mg/kg/hour).
- Oxygen supplementation.
- Continue with the ACE inhibitor and add pimobendan at 0.25-0.3 mg/kg PO BID.

Chronic therapy:

- Lasix at 2 mg/kg PO BID and increase incrementally as needed.
- Continue with the ACE inhibitor.
- Pimobendan at 0.25-0.3 mg/kg PO BID.
- Consider adding spironolactone at 0.25-2 mg/kg PO BID to control congestion.
- A sodium-restricted diet is recommended, although some dogs will not eat it.
- Consider administering omega-3 fatty acids, digoxin, theophylline, and cough suppressants.
- If the dog is not already on beta blockers, then do not commence.

Stage D: This is the end-stage of the disease. Continue with the standard therapy of diuretics, ACE inhibitors, and pimobendan, and consider the following:

- Abdominocentesis when applicable to decrease discomfort if the patient is undergoing respiratory distress.



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- Anti-anxiety medications for sedative purposes.
- Sodium nitroprusside and dobutamine (in a critical care facility).
- Nitroglycerin.

We recommend monitoring serum urea, creatinine, electrolytes, urine specific gravity, and possibly blood pressure for 5-7 days after therapy has commenced. A repeat ECG is warranted if an arrhythmia was present during the original assessment.

References:

Atkins C, Bonagura J, Ettinger S, et al. Guidelines for the diagnosis and treatment of canine chronic valvular heart disease. *J Vet Int Med* 2009; 23(6):1142-50.

Borgarelli M, Buchanan J. Historical review, epidemiology and natural history of degenerative mitral valve disease. *J Vet Cardiol* 2012;14:93-101.

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