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Clinical Sonography & Telecytology
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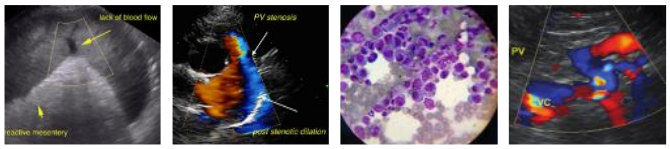


SonoPath.com
1-800-838-4268 info@sonopath.com

PATIENT

Romeo Kostelnik

SPECIES

Canine

BREED

Boston Terrier

SEX

Neutered Male

AGE

6 Years

WEIGHT

25 Pounds

INTERPRETED BY

Eric Lindquist, DMV DABVP, Cert. IVUSS

IMAGING PERFORMED BY

Diane McFadden

HOSPITAL NAME

Newton VH

REFERRING VET

Dr. Kim

INVOICE

14265

DATE

3/11/22

PRESENTING CLINICAL SIGNS

History: hypertension; IVDD on rads. On benazapril 5 mg bid, gabapentin 100mg bid-tid

Abnormal PE/Chem/CBC/UA Results: CBC/chem wnl. CPL normal BP: 189/118 (136) , 162/131 (108), 197/135 (140),

ULTRASONOGRAPHIC EXAMINATION OF THE HEART & ABDOMEN

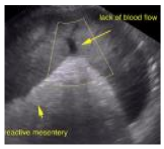
CANINE CARDIAC PARAMETERS	MR VMAX (m/s)	TR VMAX (m/s)	LA/AO (Boon method)	LA/AO (Heart Base; Swe)	FS (%)	EF (%)	EPSS (cm)
NORMAL PARAMETER	4.5-5.5	<2.7	1.3	<1.6	28-40	40-100	<0.6
PATIENT	--	--	1.15	1.33	48	82	0.27
CANINE CARDIAC PARAMETERS	**HR** (BPM)	**AV VMAX** (m/s)	**PV MAX** (m/s)	**BODY WEIGHT** (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	BELOW	BELOW	BELOW	BELOW
PATIENT	164	1.50	1.30	--	2.2	2.27	--

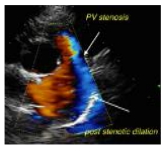
Cardiac Presentation

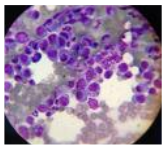
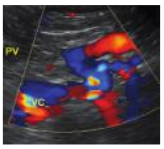
The echocardiogram in this patient demonstrated normal **left atrial** size based on 3 separate methods of LA evaluation. The cranial and caudal **mitral** valve leaflets presented normal linear structure, extension in systole, and union in diastole with normal kinesis. The **left ventricle** presented 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 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. **Tricuspid** valvular assessment demonstrated adequate linear morphology and kinesis. The **right ventricle** was of normal size (1/3 diameter of LV), chordae structure, myocardial echogenicity and thickness. **Pulmonary outflow** 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. The cranial **mediastinum and pericardial and extra-cardiac 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





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was present. No evidence of inflammatory or neoplastic changes were noted. Ureteral papillae were normal.

The **kidneys** revealed normal size and structure, corticomedullary definition and ratio for this age. The cortices presented largely uniform texture with normal echogenic relationship to liver and spleen. Medullary structure differed distinctly from the cortex and no evidence of pelvic dilation was present. The capsules were acceptably uniform without significant irregularities. The right kidney measured 4.33 cm. The left kidney measured 4.27 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 right adrenal gland measured 1.98 cm x 1.43 cm at the cranial pole and 0.54 cm at the caudal pole. The left adrenal gland measured 2.04 cm x 0.53 cm at the caudal pole and 0.51 cm at the cranial pole.

Spleen

The **spleen** presented a smooth homogeneous parenchyma hyperechoic to liver and renal cortical parenchyma. The capsule was smooth without noticeable expansion or deviation from within the spleen or adjacent pathology. The splenic vasculature demonstrated normal volume without signs of congestion or thrombosis. No sonographic evidence of acute or chronic inflammatory, neoplastic, or infarctual changes were noted.

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 gallbladder presented acceptably thin walls with primarily anechoic content. The cystic and common bile ducts were normal. No pathological hepatic lymphadenopathy was evident. No overt structural evidence of inflammatory, infiltrative or regenerative pathology was evident.

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

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.

ULTRASONOGRAPHIC FINDINGS

- Normal echocardiogram
- Normal abdomen

INTERPRETATION OF THE FINDINGS & FURTHER RECOMMENDATIONS

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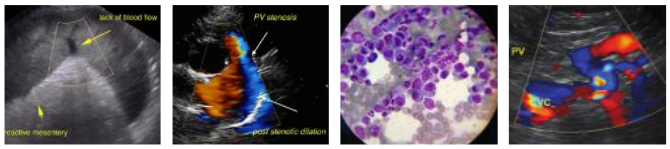

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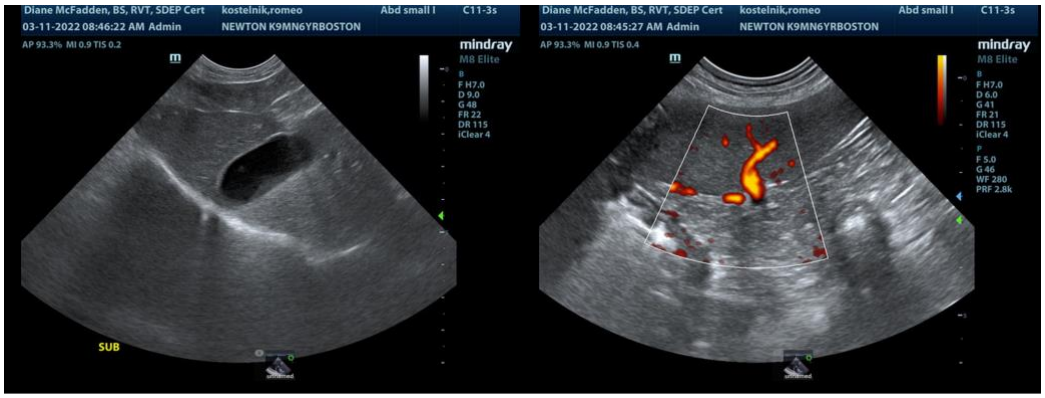
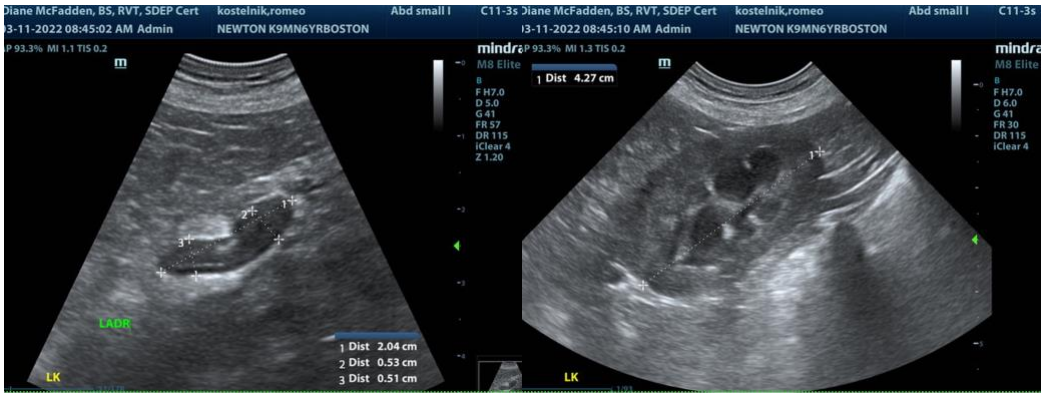
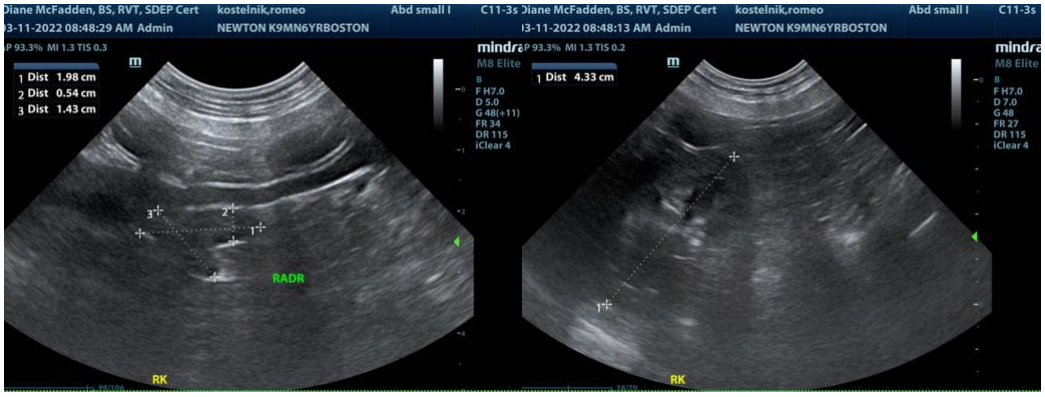
The cause of systemic hypertension is unclear in this patient. Adding Amlodipine may be the next measure to reach systolic pressure <160.







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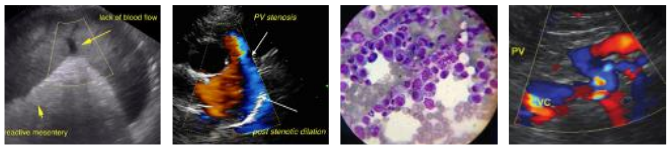

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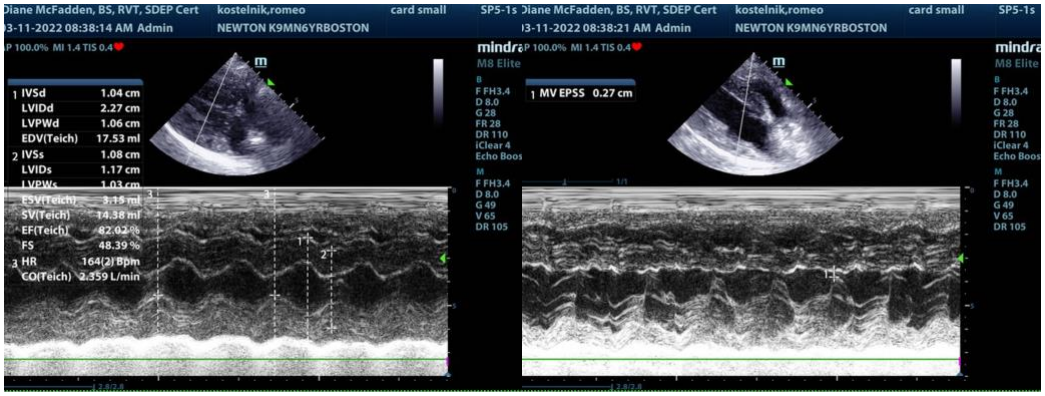
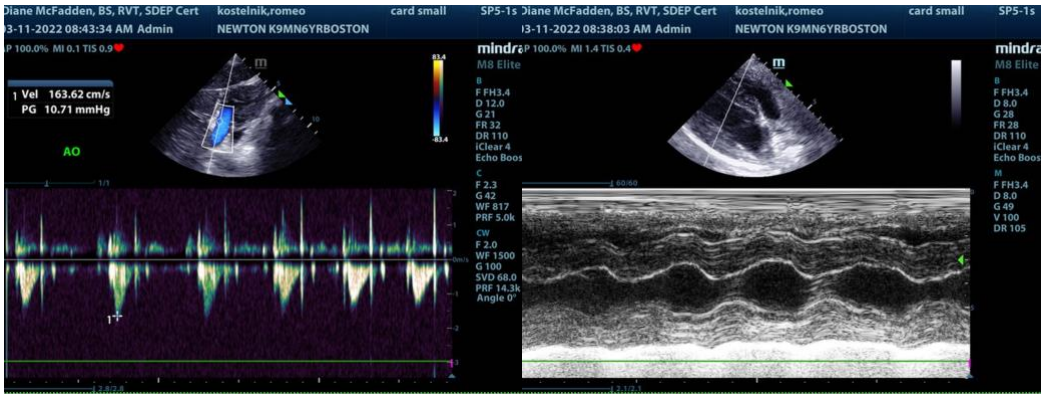




The information and recommendations provided are based on the images presented by the referring veterinarian. No evaluation can be communicated regarding pathology that was not visible in the image/video clips provided.

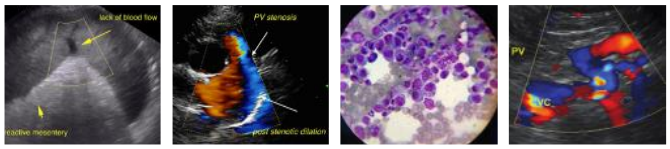
Thank you for this referral. If the clinical or image interpretation does not parallel your findings or if I can be of any further assistance please contact me.

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

Pulmonary Hypertension and Right Sided Heart Failure

http://www.sonopath.com/PHT

Description: Pulmonary hypertension (PHT) is defined as an increase in pulmonary arterial pressures and can be a significant measurable component of right-sided heart disease. Historically, this disease has been overlooked; however, it is becoming more commonly recognized due to the more widespread availability of echocardiography as well as increased clinical awareness. Therapeutic advances have also resulted in improvements in the medical management of PHT.



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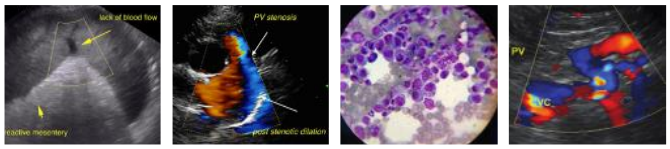
PHT is divided into two categories: precapillary and postcapillary. Causes for precapillary PHT include: hypoxemia (e.g. chronic airway disease, brachycephalic syndrome, chronic pulmonary disease, pulmonary edema, pulmonary fibrosis); inflammatory disease (e.g. chronic airway disease, chronic interstitial pneumonia, primary vascular disease, such as necrotizing arteritis and lung worms); thromboembolic disease (e.g. nephrotic syndrome, hyperadrenocorticism, DIC, neoplastic disease); obstructive disease (e.g. heart worms, compression due to thoracic neoplasia); and pulmonary overcirculation (e.g. patent ductus ateriosis, atrial septal defect, ventricular septal defect). Postcapillary PHT arises from an increase in pulmonary venous / left atrial pressures, and is mainly caused by mitral valve disease or cardiomyopathy.

Clinical Signs: Clinical signs associated with PHT include: cough, dyspnea, cyanosis, exercise intolerance, right-sided heart failure (RHF), and syncope. Stridor or abnormal lung sounds may be auscultated, and a heart murmur may be present, particularly in cases of postcapillary PHT, marked tricuspid regurgitation, or concomitant (non-related) cardiac disease. Sometimes, only a soft systolic or diastolic heart murmur will be present. Thus, the absence of a heart murmur in a cyanotic patient does not rule out the presence of a shunt. A split-second heart sound can also sometimes be heard. Patients with chronic respiratory disease, heartworms, cyanosis, severe exercise intolerance, and suddenly acquired RHF, as well as those with left-sided CHF for whom traditional therapy is not working, should always be screened for PHT. Patients that experience the sudden onset of PHT, such as those with acute respiratory distress syndrome and pulmonary thromboembolic disease, are the most difficult to treat. In these cases, the heart does not have time to adapt and compensate as it does with chronic respiratory disease or idiopathic PHT. Therefore, one should always evaluate patients that present with acute respiratory crisis for possible secondary PHT.

Diagnostics: PHT is diagnosed with echocardiography using color flow and spectral Doppler. A tricuspid regurgitant velocity greater than 2.8 m/s and/or a pulmonic insufficiency velocity greater than 2.2 m/s are considered abnormal and consistent with PHT. The pressure gradient is then calculated using the modified Bernoulli equation, $P = 4V^2$, where V = velocity. A normal pressure gradient is considered to be less than 30 mm Hg. Thus, mild PHT is characterized by a pressure gradient that falls between 31-50 mm Hg; moderate PHT is assigned when the gradient is between 51-75 mm Hg; and severe PHT occurs when the gradient is above 75 mm Hg. The gold standard for confirming PHT is via pulmonary arterial catheterization, which allows for the direct measurement of pulmonary arterial pressures; however, this is not commonly undertaken. Thoracic radiographs are critical for evaluating the pulmonary parenchyma and identifying pulmonary vascular abnormalities due to thromboembolism or heartworm disease. One can evaluate pulmonary functions using arterial blood gas assessments or pulse oximetry; however, the latter is a less sophisticated testing method. If thromboembolic disease is suspected, measuring a patient's pulse oximetry while s/he is both on and off oxygen is a crude way to assess diffusion capabilities. If the pulse oximetry reading is very low and does not improve with oxygen therapy, one should suspect a ventilation-perfusion (VQ) mismatch, which typically occurs in the face of severe pulmonary thromboembolism (PTE). If the pulse oximetry reading normalizes with oxygen supplementation, then one should suspect a diffusion disorder, which often occurs with parenchymal disease. Ventilation perfusion scanning using nuclear scintigraphy is considered the gold standard test for PTE.

Cor pulmonale is typified by right heart enlargement secondary to various primary pulmonary disease states that result in pulmonary hypertension. RHF, when accompanied by ascites, can develop acutely if there is



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an abrupt change in pulmonary arterial pressure due to any of the following: acute severe thromboembolic disease, lung lobe torsion, sudden acute respiratory distress syndrome (SARDS), acute inflammation, or rapidly progressive neoplasia. Alternatively, RHF can develop slowly over time due to chronic diseases, such as neoplasia, chronic inflammatory disease, asthma or bronchitis, pulmonary fibrosis, and brachycepahlic syndrome.

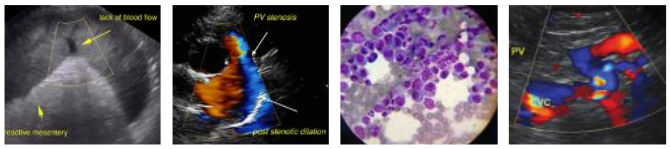
Further clinical workup should always include a CBC, urinalysis, D-dimers, abdominal ultrasound, tests for hyperadrenocorticism, and heartworm tests.

It has been reported that NT-proBNP increases markedly in cases of PHT. For this reason, an elevation in NT-proBNP in a dyspneic patient with a heart murmur does not automatically indicate the presence of congestive left-sided heart failure (LHF). When treating dyspneic patients, one should never rely uniquely on NT-proBNP testing.

Treatment: Treatment should initially revolve around the primary inciting cause. In the event of primary left-sided heart disease with severe left atrial enlargement, which results in increased left-sided pressures and ultimately PHT, management entails the use of diuretics, angiotensin-converting enzyme (ACE) inhibitors, and pimobendan. These medications are also indicated for treating RHF in the absence of left-sided disease. Pimobendan is an inodilator with positive inotropic properties. It is also a PDE3 inhibitor and thus exerts vasodilating effects in the pulmonary vasculature, resulting in a reduction of PHT. This is the first choice for PHT in the face of LHF. The standard dose of pimobendan, 0.25-0.3 mg/kg PO BID, can also be increased to three times daily, as needed, to manage severe PHT. The use of theophylline (5-10 mg/kg BID), a non-specific PDE3 inhibitor, can be additionally beneficial, particularly in patients with airway disease. Sildenafil, a phosphodiesterase V inhibitor, should be added in the face of CHF. The target dose is 1-2 mg/kg PO BID-TID, but the effective dose varies with each individual. Based on the severity of the clinical signs, one can begin by administering a low dose (0.5 mg/kg PO BID) for the first 2 weeks, increase it to 1 mg/kg PO BID for the next 2 weeks, and then raise it again to 1.5 mg/kg PO BID for the following 2 weeks (one can go up to 2 mg/kg PO BID and even increase it to TID, as necessary).

In patients with acute PHT and tricuspid regurgitation velocities greater than 4.5 m/s or pulmonic regurgitation velocities greater than 4 m/s, oxygen therapy and abdominocentesis is required if ascites is present. Additional therapy includes the administration of pimobendan and sildenafil, as per above.

Prognosis: The prognosis for PHT is guarded, although it has improved somewhat due to increased use of sildenafil and pimobendan. Subsequent to treatment, it is necessary to monitor clinical signs, respiratory rate, and effort, and follow up with periodic echocardiographic exams. Yet, even if follow-up echocardiograms show marked improvements in clinical signs, it is possible that the velocity of the tricuspid insufficiency jet may not have changed significantly. It is also recommended to track BUN, creatinine, electrolytes, radiographs, and systemic blood pressure in follow-up appointments, depending on the underlying disease process and course of medical management.



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Goggs R, Benigni L, Fuentes V, et al. Pulmonary thromboembolism. *J Vet Emerg Crit Care* 2009;19:30-52.

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