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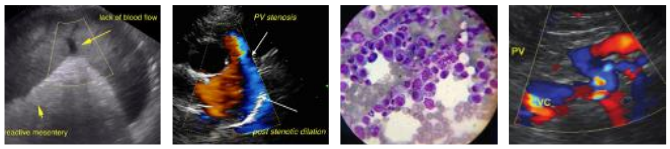

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1-800-838-4268 info@sonopath.com

PATIENT
Tito Guerra

SPECIES
Canine

BREED
French Bulldog

SEX
Neutered Male

AGE
11 Years

WEIGHT
18 kg

INTERPRETED BY
Eric Lindquist, DMV DABVP, Cert. IVUSS

IMAGING PERFORMED BY
Dr. Belan

HOSPITAL NAME
Silverado VC

REFERRING VET
Dr. Marahar

INVOICE
21670

DATE
3/17/23

PRESENTING CLINICAL SIGNS

History: Lethargic with good appetite and a pendulous abdomen has formed over the last few weeks. On amoxicillin and doxycycline. Scan abdomen 54 images echo 56 images total 110

Abnormal PE/Chem/CBC/UA Results: Moderate elevation of liver enzymes

ULTRASONOGRAPHIC EXAMINATION OF THE HEART & ABDOMEN

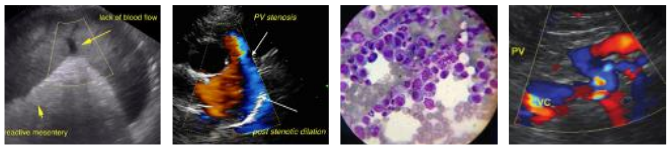
CANINE CARDIAC PARAMETERS	**MR VMAX** (m/s)	**TR VMAX** (m/s)	**LA/AO** (Boon method)	**LA/AO** (Heart Base; Swe)	**FS** (%)	**EF** (%)	**EPSS** (cm)
NORMAL PARAMETER	4.5-5.5	<2.7	1.3	<1.3	28-40	40-100	<0.6
PATIENT	--	4.18	1.2	1.68	50	84	0.1
CANINE CARDIAC PARAMETERS	**HR** (BPM)	**AV VMAX** (m/s)	**PV 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	117	0.84	--	--	--	1.94	--

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 septal flattening owing to volume and pressure overload of the right ventricle. Paradoxical septal motion was noted. The calculations may be erroneous owing to paradoxical septal motion interference. The **myocardium** presented normal echogenicity without subjective evidence of significant fibrotic or ischemic disease. Aortic velocity was subnormal, likely underestimated. The right ventricle was severely enlarged. The right atrium was severely enlarged in this patient with a 2.5 – 3:1 right atrial to left atrial ratio in four chamber long axis. Complete filling of the right atrium was noted on color flow doppler. Severe tricuspid insufficiency was noted, consistent with severe pulmonary hypertension. Ascites was noted, consistent with right sided heart failure owing to tricuspid insufficiency and pulmonary hypertension. The pulmonary artery was dilated. The pulmonic valve was unremarkable. No evidence of heartworm. No pericardial or pleural effusion noted.

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.



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The **kidneys** revealed largely normal size and structure, corticomedullary definition and ratio (cortex 1/3 of medulla) were essentially maintained with some minor 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. The right kidney measured 5.94 cm. The left kidney measured 6.0 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 0.51 cm at the cranial pole and 0.49 cm at the caudal pole. The right adrenal gland measured 0.38 cm at the caudal pole and 0.35 cm at the cranial pole.

Spleen

The **spleen** revealed minor heterogenous parenchymal changes. The spleen was relatively normal in size with mild vascular congestion.

Liver

The **liver** revealed increased portal markings, heterogenous parenchymal changes and dilated hepatic veins, consistent with passive congestion owing to right sided heart failure. A 5.2 cm x 3.98 cm parenchymal mass was noted in this patient, appeared to be deriving from the left caudal liver.The gallbladder and common bile duct were unremarkable.

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

- Right sided heart failure
- Chronic passive congestion liver with inflammatory pattern and left caudal liver mass with enhanced surrounding mesentery
- Nodular spleen
- Age-related renal changes
- Ascites

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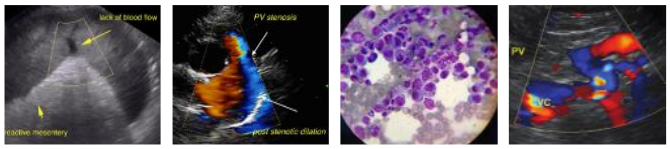

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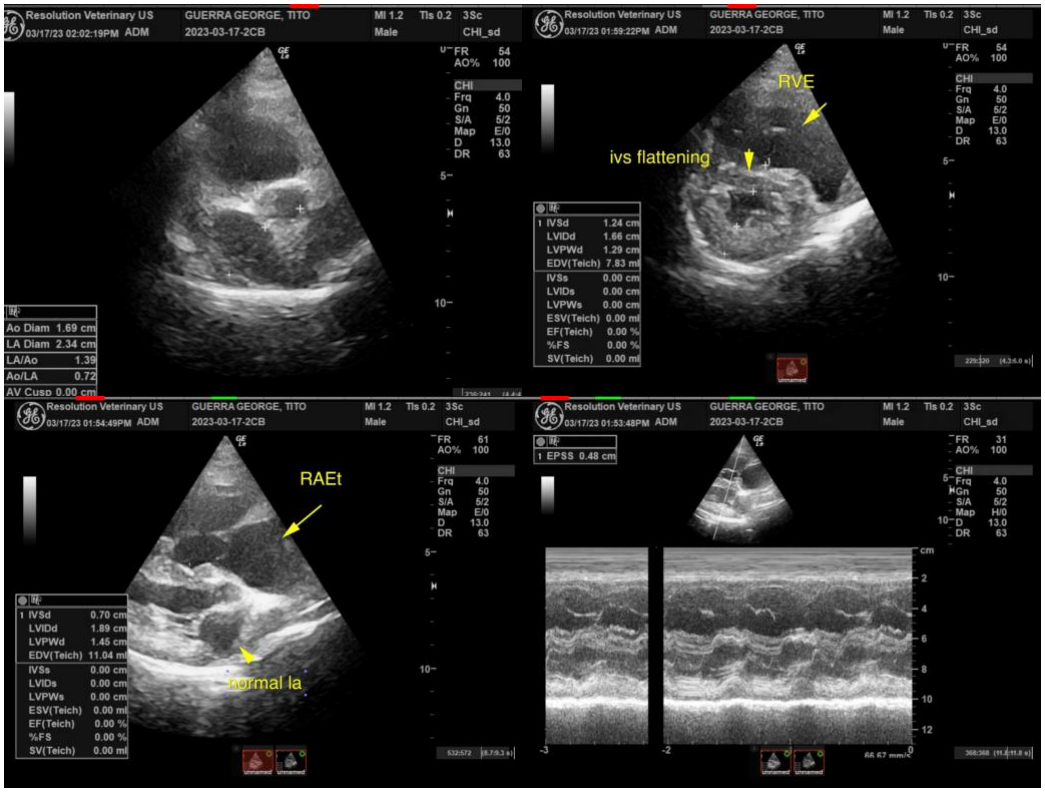
INTERPRETATION OF THE FINDINGS & FURTHER RECOMMENDATIONS

There are two separate issues occurring in this patient, one is right sided heart failure causing passive congestion and ascites, the second is left caudal liver mass and chronic liver disease with nodular splenic changes. Abdominocentesis is recommended, if not already performed, to ensure that this is a transudate or modified transudate. Ideally, stabilization of the right sided heart failure would be performed with left liver lobectomy. However, the comorbidities are challenging to manage. Regarding the right sided heart failure, recommend Sildenafil 1 mg/kg BID, increasing to 1.5 mg/kg BID after two weeks. Ace-inhibitor therapy could be justified 0.5 mg/kg SID, progressing to BID and Spironolactone 1-2 mg/kg BID. If the patient is stable, recheck echo in one month. Monitoring blood pressures, BUN and clinical signs is recommended. Staging for eventual left liver lobectomy would be indicated, however, further disease should be evaluated with 25-guage aspirates of the general hepatic parenchyma, hepatic mass and splenic nodules. If Sildenafil is not available, then second level treatment for right sided failure can involve Pimobendan 0.3 cm/kg BID, but I do not recommend utilizing both of them, simultaneously, at this time.



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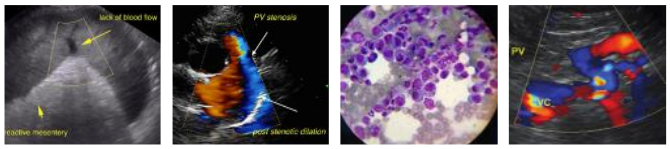

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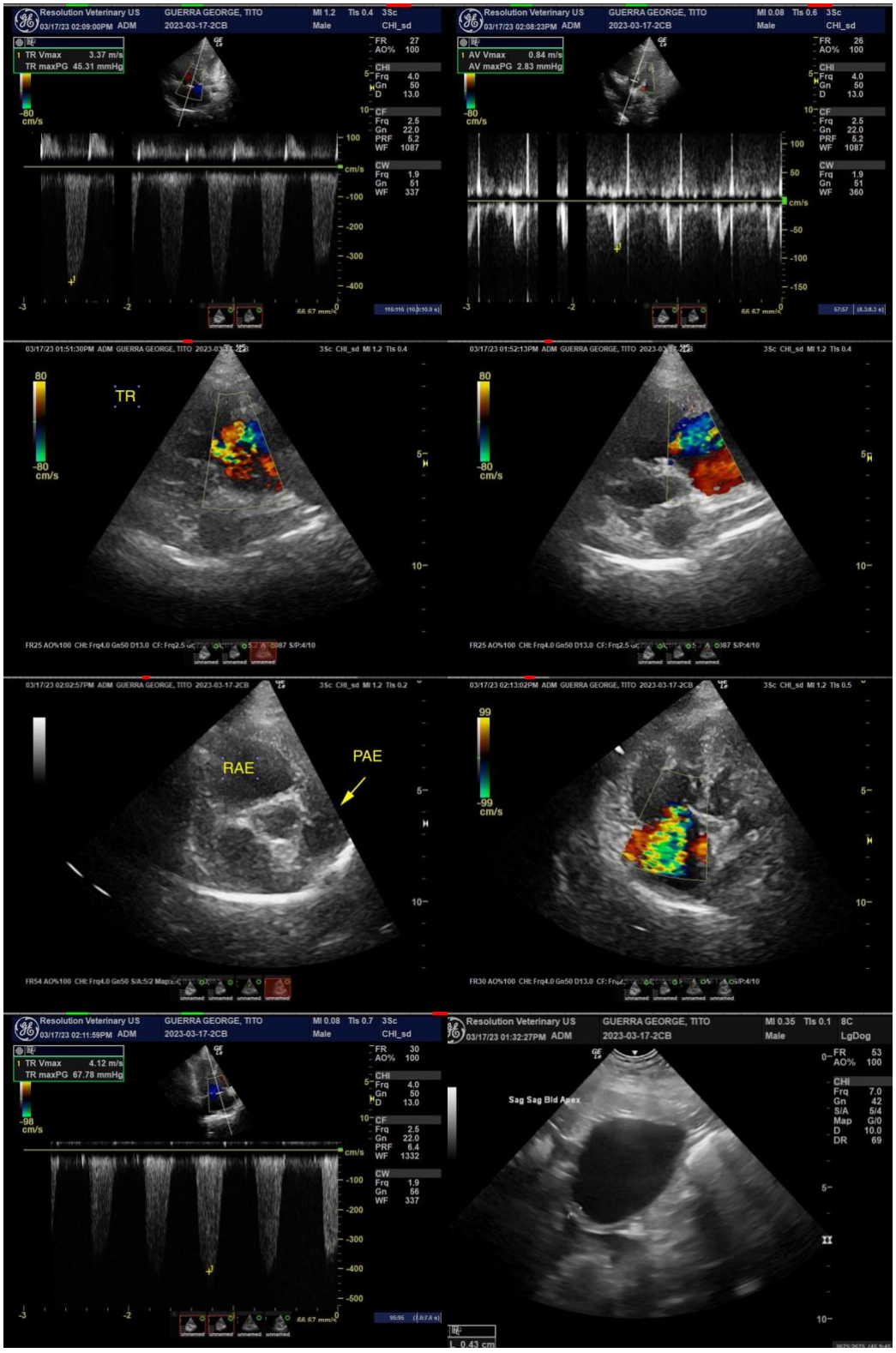

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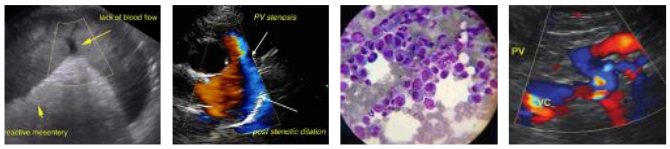

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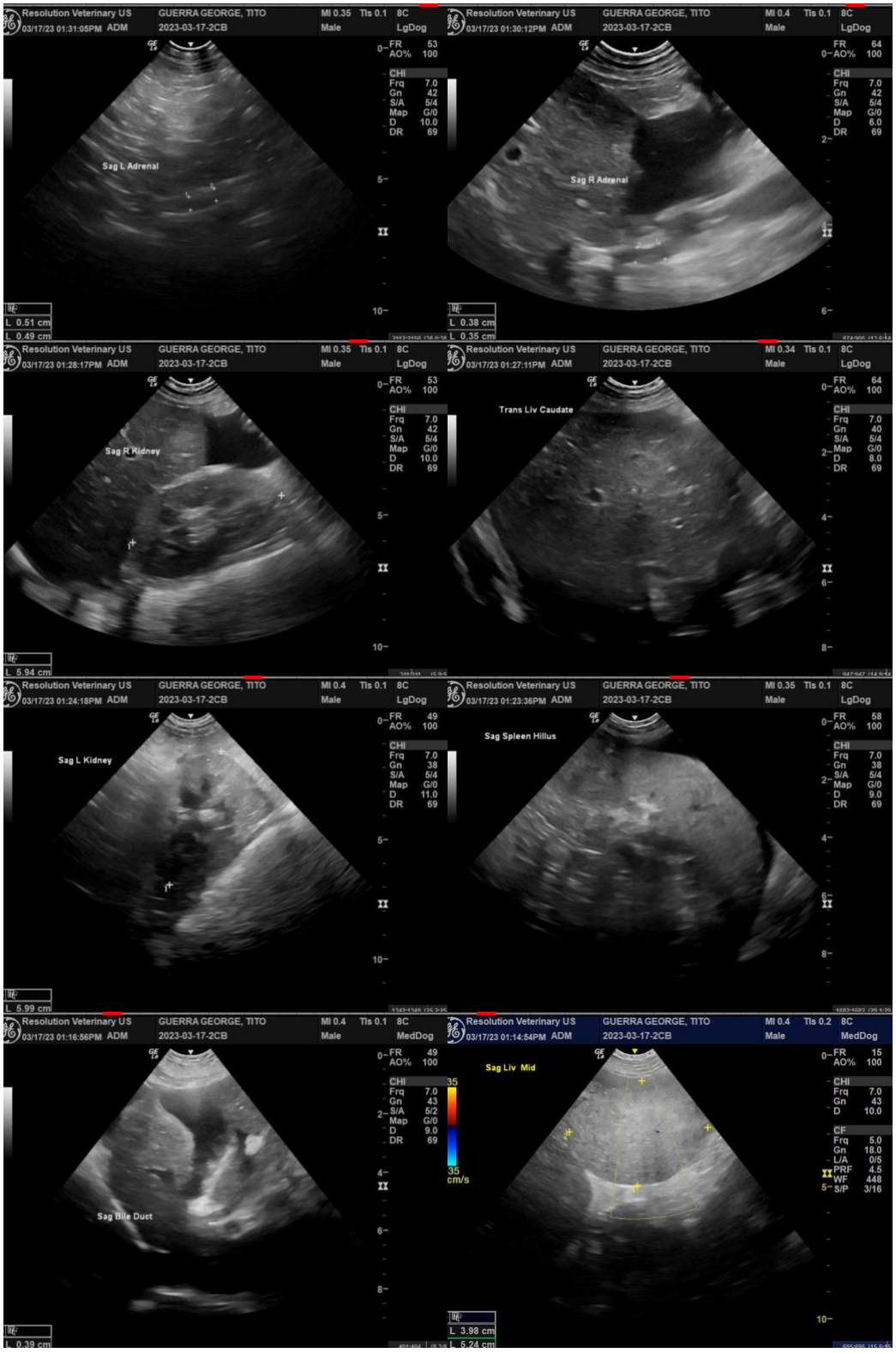

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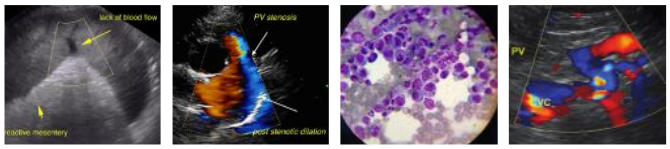

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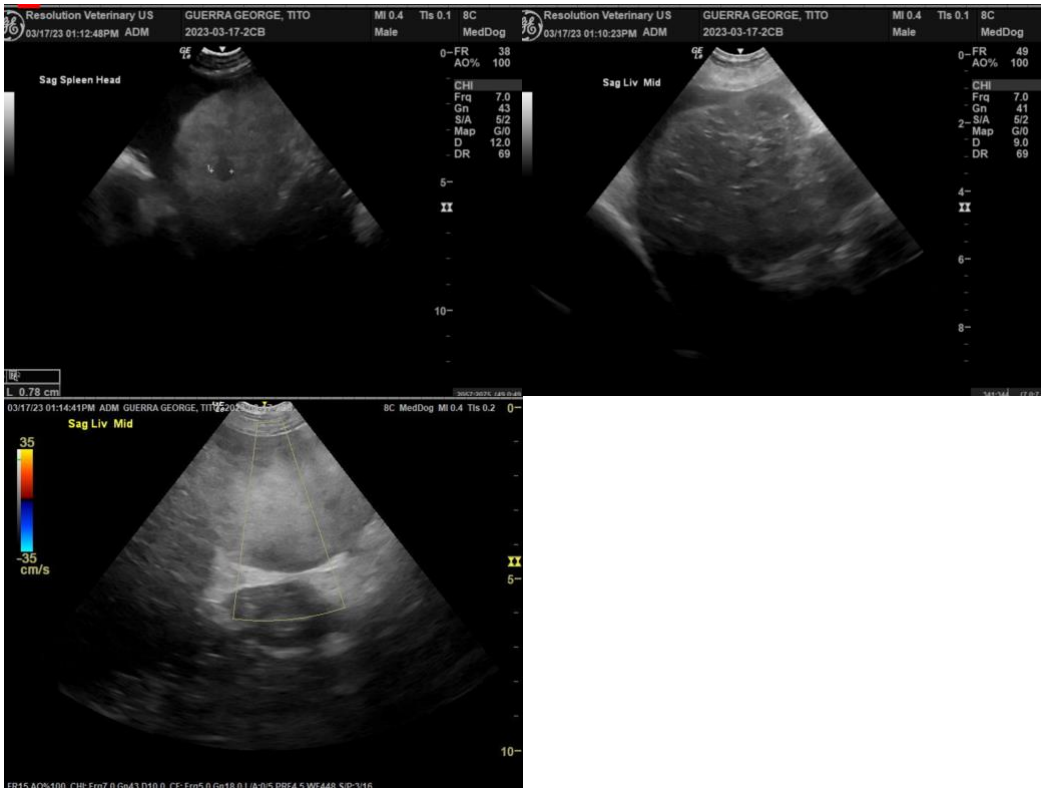


The information and recommendations provided are based on the images presented by the referring veterinarian. No evaluation can be communicated regarding pathology that was not visible in the image/video clips provided.

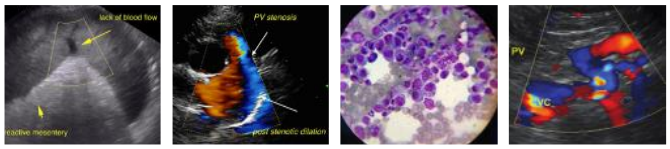
Thank you for this referral. If the clinical or image interpretation does not parallel your findings or if I can be of any further assistance please contact me.

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

Right Heart Disease-General Considerations

http://www.sonopath.com/RightHeartDisease

Description: Right heart disease is often an incidental finding, which can be either cardiogenic or secondary to respiratory or systemic disease. The coughing patient with right heart disease may present with primary respiratory disease (i.e., bronchial collapse, collapsing trachea, pneumonitis) and suffer from secondary pulmonary hypertension (PHT). Concurrent mitral valve disease and chronic left-sided congestive heart failure (CHF) might also lead to PHT. The dyspeic patient with right heart enlargement might have pulmonary



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hypertension due to airway disease, chronic CHF, parenchymal lung disease (e.g. pulmonic fibrosis), or a cardiac shunt with secondary PHT and shunt reversal.

Primary cardiac causes of right heart enlargement include: tricuspid dysplasia/degeneration; pulmonic stenosis; pulmonic insufficiency; atrial or septal defects; patent ductus arteriosus; right auricular masses; and pericardial peritoneal diaphragmatic hernias. The second most common cause of right-sided enlargement is secondary PHT, which results in high-velocity tricuspid insufficiency (TR vel.>2.8 m/sec) and pulmonic insufficiency due to diseases that cause increased pulmonary vascular resistance or increased pulmonary wedge pressures. The most common cause of secondary PHT is left-sided heart failure (LHF), which presents radiographically as a more globoid-shaped heart with marked left atrial and ventricular enlargement. There are also signs of left-sided CHF as opposed to a simple prominent cranial waist or reverse D radiographic presentation.

Secondary, non-cardiac causes of PHT include: acute or chronic respiratory disease; pulmonary thromboembolic disease; thoracic neoplasia; excessive thoracic fat deposition (e.g. Pickwickian syndrome, which leads to chronic hypoxia); brachycephalic syndrome; high altitude disease; heartworm disease; and primary vascular disease.

Clinical Signs: The most common presenting symptoms of right heart disease are collapse, syncope, intermittent or constant acute respiratory distress (e.g. thromboembolic disease), and exercise intolerance.

Diagnostics: Physical examination may reveal a right-sided apical heart murmur and/or a cranial left heart murmur, a split S2, jugular distension, ascites, and signs consistent with respiratory disease (i.e., cough, wheeze, tracheal collapse, tachypnea). Radiographic findings may reveal an enlarged right atrium, right ventricle, and/or primary/secondary branches of the pulmonary artery. In cases of PHT, an enlarged or engorged pulmonary artery is often present. Tortuous arteries or those that suddenly terminate can indicate the presence of thromboembolic disease or heartworms. An interstitial pattern might indicate the presence of pulmonary parasitism or primary interstitial lung disease. Pulmonic stenosis is suspected if the pulmonic segment is enlarged. ECG findings include tall P and S waves with a right axis shift.

Treatment: Please refer to the chapter “Pulmonary Hypertension” for therapeutic recommendations.

References:

Oyama MA, Rush JE, Rozanski EA, et al. Assessment of serum N-terminal pro-B-type natriuretic peptide concentration for differentiation of congestive heart failure from primary respiratory tract disease as the cause of respiratory signs in dogs. *J Am Vet Med Assoc* 2009;235:1319-25.

Rozanski E. Interstitial lung disease in small animals. Proceedings from American College of Veterinary Internal Medicine Forum, Denver, CO, June 15-18, 2011.

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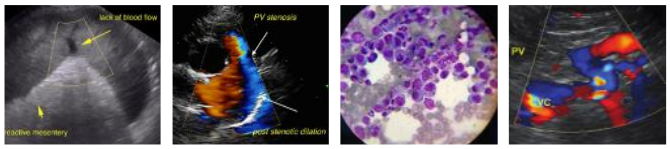

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Zoia A, Augusto M, Drigo M, Caldin M. Evaluation of hemostatic and fibrinolytic markers in dogs with ascites attributable to right-sided congestive heart failure. *J Am Vet Med Assoc* 2012;241:1336-43.