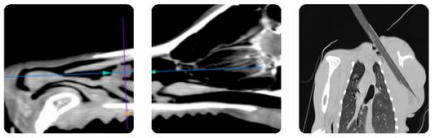


PATIENT

Milo Pooches

SPECIES

Canine

BREED

Bulldog Mix

SEX

Male

AGE

1Y, 1M

WEIGHT

17.7kg

INTERPRETED BY

Tilde Rodrigues Froes, DMV, MSc., Dr. Med Vet., Dipl. CBraRVet

IMAGING PERFORMED BY

Mobile Pet Imaging

HOSPITAL NAME

Mobile Pet Imaging

REFERRING VET

Novoa

INVOICE

73960

DATE

2-26-26

PRESENTING CLINICAL SIGNS

- Milo has a history of elevated liver enzymes since puppyhood. Previous diagnostics confirmed significant liver dysfunction, Bile acids (4/26/25) (Pre 53.2 umol/L, Post 119.9 umol/L) and a low Protein C level (8/9/25) (46%). Over the past week, was reported an acute onset of lethargy and significant weight loss (from 50 lbs to 38 lbs). A week prior, Milo had an episode of regurgitation that contained paper towels. He was vomiting before admission in the hospital (2/19/26). He has been receiving Denamarin and was recently started on Lactulose. The physical exam revealed that he was quiet but alert and had a decreased body condition. A new heart murmur was noted and some dark stool was noted. Blood Work (2/19/26): Revealed BUN 74 mg/dL, Creatinine 2.4 mg/dL, SDMA 29 µg/dL, indicating kidney dysfunction. Liver enzymes were severely elevated (ALT 2,311 U/L, ALP 2,068 U/L), consistent with hepatopathy. A mild, microcytic, hypochromic non-regenerative anemia and low albumin (2.5 g/dL) were also present. Urinalysis (2/19/26) showed USG 1.045, protein 3+, and rare cocci bacteria, suggesting urinary tract or kidney infection. Urine Cystatin B (2/19/26): 652 ng/mL, confirming kidney injury. Abdominal Radiographs showed a small liver and some retained food material in the stomach, but no evidence of an intestinal obstruction. Abdominal Ultrasound revealed fluid-filled intestines, a large gallbladder containing debris, and abnormal liver vasculature. CT requested to rule out Liver Shunt.

Abnormal PE/Chem/CBC/UA Results: Blood Work (2/20/26): After approximately 24 hours of IV fluid therapy, the kidney values remained largely unchanged (Creatinine 2.5 mg/dL, BUN 67 mg/dL). Liver enzymes showed mixed changes (ALT decreased to 2,074 U/L, AST increased to 815 U/L, ALP increased to 2,474 U/L), and total bilirubin increased to 2.1 mg/dL. PE (2/26/26): T100.1 F, HR 112, RR 30, BCS 3/9, MM pink, CRT <2 seg, H/L WNL, Dental Calculus 3/4

COMPUTED TOMOGRAPHIC STUDY OF THE ABDOMEN

A pre- and post-contrast CT study of the abdomen are provided for review totaling 3 series. One pre-contrast series of the abdomen, bone algorithm. Two post-contrast series of the abdomen, soft tissue algorithm.

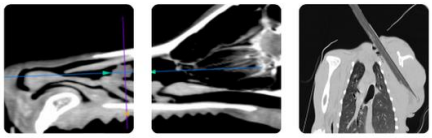
COMPUTED TOMOGRAPHIC FINDINGS

The liver is markedly reduced in volume, with homogeneous soft tissue attenuation, regular margins, and uniform contrast enhancement.

The portal vein and all portal tributaries are severely dilated. In the right cranial abdomen, there is a complex conglomerate of markedly enlarged, tortuous vascular structures, with both intrahepatic and extrahepatic components. The extrahepatic portion appears to communicate with a large vascular aneurysmal dilation.

Within the intrahepatic vasculature, there are regions of heterogeneous vascular contrast enhancement, with apparent contrast retention in a smaller-caliber vascular complex contiguous with the tortuous anomalous vessels, suggestive of abnormal hepatic arterial–portal communications.

The extrahepatic portal vasculature demonstrates severe dilation of all tributaries. Additionally, there are multiple, extensive dilated small vessels distributed throughout all abdominal quadrants, apparently interconnected. The cranial mesenteric vein, caudal mesenteric vein, left colic vein, splenogonadal vessels and the entire mesenteric venous chain are diffusely dilated and associated with small shunting vessels (acquired portal collaterals).



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Within the large tortuous vascular complex in the right cranial abdomen - extra-hepatic, there is an intraluminal filling defect within a larger vessel, adjacent to the described aneurysmal dilation, consistent with suspected thrombosis.

The caudal vena cava is dilated. The azygos vein is also dilated.

The gallbladder is not visualized.

The stomach is moderately to severely distended with heterogeneous hypoattenuating fluid-ingesta content and gas.

The gastrointestinal tract is otherwise normally positioned. The colon is moderately distended with gas and heterogeneous fecal material; wall thickness is within normal limits.

The right pancreatic lobe is mildly enlarged but maintains normal contour and attenuation. The left pancreatic lobe is unremarkable.

The abdominal lymph nodes and both adrenal glands are within normal limits.

The kidneys are normal in size, shape, and attenuation. The renal pelves are unremarkable. The ureters are poorly visualized due to absence of excretory contrast phase and the presence of numerous anomalous retroperitoneal vessels.

The urinary bladder is moderately distended with homogeneous hypoattenuating fluid. Wall thickness is normal.

The prostate is normal in size, shape, and attenuation.

At L2–L3, there is complete bridging vertebral endplate spondylosis deformans. At this same level, the vertebral endplates are presenting osteolytic foci and irregular, and there is widening of the corresponding intervertebral disc space.

COMPUTED TOMOGRAPHIC DIAGNOSIS

- Severe, complex vascular malformation characterized by marked dilation of the portal vein and all portal tributaries, extensive intrahepatic and extrahepatic tortuous vascular networks, a large aneurysmal vascular dilation in the right cranial abdomen, diffuse dilation of the mesenteric venous system, and splenogonadal vessels (small shunting vessels). Findings are most consistent with a congenital hepatic arteriovenous malformation (high-flow anomalous portal–systemic vascular connections) with concurrent portal hypertension.
- Intraluminal filling defect within a major anomalous vessel adjacent to the aneurysmal dilation, highly suspicious for concurrent vascular thrombosis.
- Concurrent dilation of the caudal vena cava and azygos vein.
- Marked microhepatia.
- Non-visualization of the gallbladder, consistent with possible gallbladder agenesis.
- Moderate to severe gastric distension, compatible with delayed gastric emptying and retained heterogeneous ingesta and fluid content.
- Mild enlargement of the right pancreatic lobe, possibly associated with pancreatic edema.
- L2–L3 spondylosis deformans with irregular vertebral endplates and widening of the intervertebral disc space. The vertebral endplates at this level demonstrate osteolytic foci and irregular margins. Differential diagnosis includes discospondylitis.

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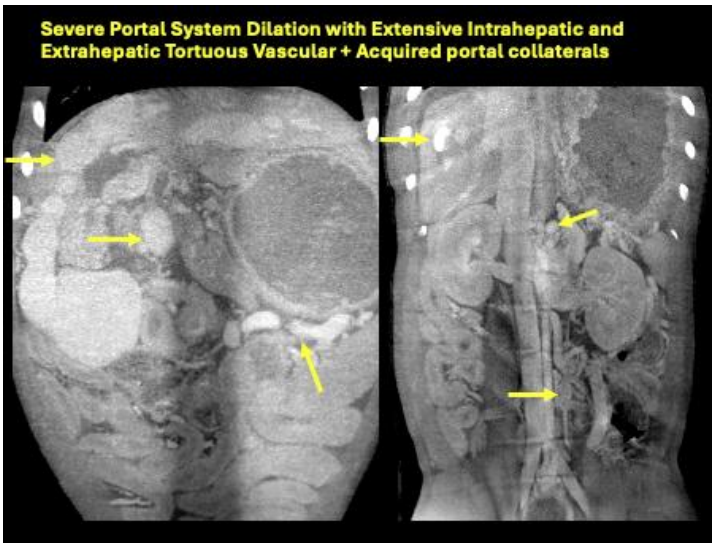
INTERPRETATIONOFTHEFINDINGS&FURTHERRECOMMENDATIONS

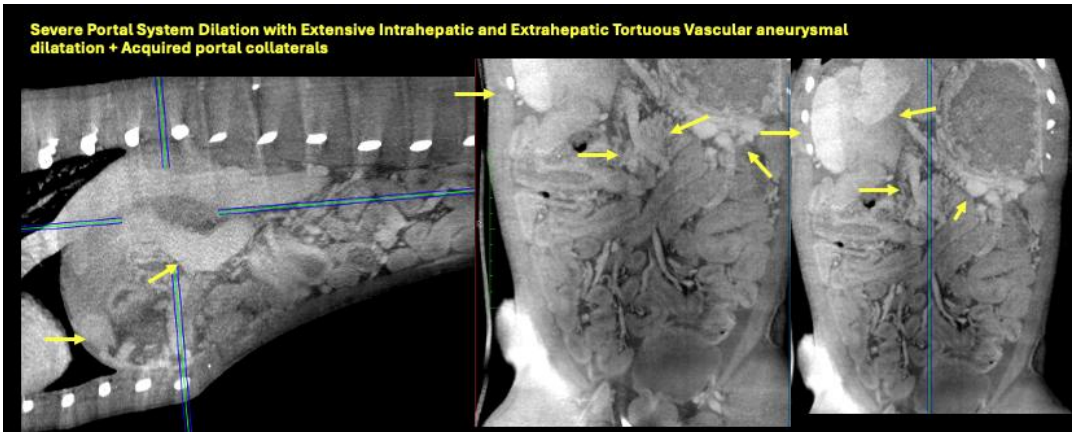
The tomographic findings demonstrate a severe, diffuse, high-flow vascular anomaly involving the portal system, with both intrahepatic and extrahepatic components, associated with aneurysmal dilation and suspected secondary thrombosis. The marked dilation of the portal and mesenteric venous systems indicated secondary portal hypertension.

The presence of a filling defect within a major anomalous vessel is concerning for thrombosis, which may further compromise portal hemodynamics of the region.

The lesion demonstrates a complex, high-flow vascular malformation. The prognosis is considered guarded due to the extent of the vascular abnormalities and the suspected concurrent thrombosis. Consider coagulation profile and assessment for hypercoagulability given suspected thrombosis.

At the level of L2–L3, the irregular vertebral endplates and widening of the intervertebral disc space raise concern for possible discospondylitis. Correlation with clinical signs (e.g., spinal pain, fever) is recommended, also consider an empirical treatment.





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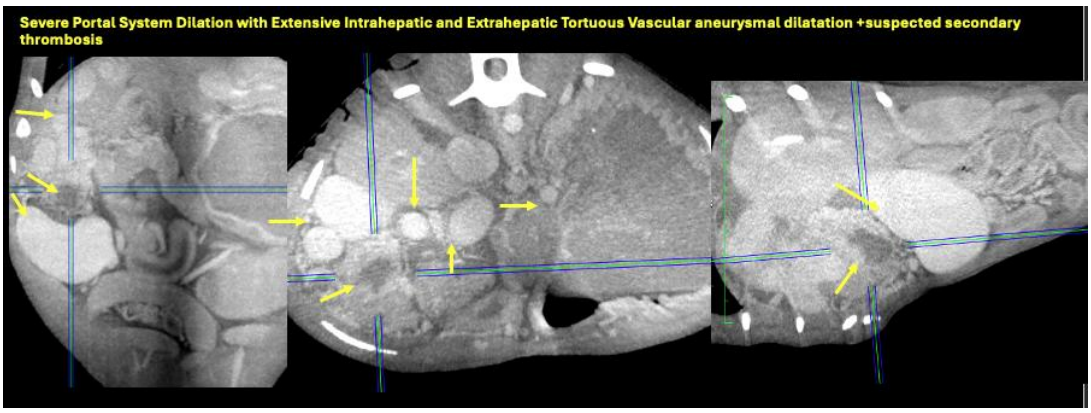

The information and recommendations provided are based on the images presented by the referring veterinarian. No evaluation can be communicated regarding pathology that was not visible in the image/video clips provided.

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.

Tilde Rodrigues Froes, DMV, MSc., Dr. Med.Vet., Dipl.CBraRVet
info@sonopath.com