



PATIENT

Winnie Gross Grubbs

SPECIES

Canine

BREED

Chihuahua/Poodle

SEX

Spayed female

AGE

11 years

WEIGHT

14.6 lbs

PRESENTING CLINICAL SIGNS

History: Winnie was presented for follow-up evaluation of presumptive pancreatitis. Over the preceding 24 hours, anorexia recurred and a single episode of vomiting was reported, following an initial period of clinical improvement. The owner described Winnie as bright, alert, and responsive. On physical examination, jaundice was evident, characterized by yellow discoloration of the skin and sclera. Winnie was afebrile at the time of presentation. Progression of acute severe pancreatitis was identified, now complicated by the development of icterus. The presence of jaundice, in conjunction with vomiting and anorexia, raises concern for concurrent hepatobiliary involvement or possible biliary obstruction. Abdominal ultrasonography demonstrated a swollen pancreas, significant hyperechoic peripancreatic fat, minor abdominal free fluid, and gallbladder sludge without marked distension, supporting the diagnosis of progressive acute severe pancreatitis with associated hepatobiliary dysfunction.

Differentials for the observed clinical signs include hepatobiliary disease, biliary obstruction, or infectious/inflammatory etiologies affecting the liver or pancreas.

Abnormal PE/Chem/CBC/UA Results: RBC 6.74 5.65 - 8.87 HCT 44.6 37.3 - 61.7 HGB 1.1 13.1 - 20.5 LOW MCV 66.2 61.6 - 73.5 MCH < 1.6 21.2 - 25.9 LOW MCHC < 2.5 32.0 - 37.9 LOW RDW 17.3 13.6 - 21.7 %RETIC 0.1 RETIC 5.4 10.0 - 110.0 LOW WBC 26.89 5.05 - 16.76 HIGH NEU 78.1 LYM 8.1 MONO 12.9 EOS 0.7 BASO 0.1 NEU 21.01 2.95 - 11.64 LYM 2.19 1.05 - 5.10 MONO 3.47 0.16 - 1.12 EOS 0.19 0.06 - 1.23 BASO 0.03 0.00 - 0.10 PLT 409 148 - 484 MPV 13.2 8.7 - 13.2 PDW 16.2 9.1 - 19.4 PCT 0.54 0.14 - 0.46 GLU 454 70 - 143 SDMA 9 0 - 14 CREA 1.5 BUN 44 7 - 27 BUN/CREA 29 PHOS 8.1 2.5 - 6.8 CA 10.4 7.9 - 12.0 TP 7.3 5.2 - 8.2 ALB 3.4 2.2 - 3.9 GLOB 3.9 2.5 - 4.5 ALB/GLOB 0.9 ALT 395 10 - 125 ALKP > 2000 23 - 212 GGT 6 0 - 11 U/L TBIL 0.6 0.0 - 0.9 CHOL 328 110 - 320 AMYL 900 500 - 1500 LIPA 1555 200 - 1800 QPL 449 0 - 200

INTERPRETED BY

Eric Lindquist, DMV
DABVP, Cert. IVUSS

ULTRASONOGRAPHIC EXAMINATION OF THE ABDOMEN

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 was 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 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 this age patient. Medullary structure differed distinctly from that of the cortex and no evidence of pelvic dilation was present. The left and right 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 0.5 cm.

IMAGING PERFORMED BY

Dr. Hornbuckle

HOSPITAL NAME

Golden Isles AH

REFERRING VET

Dr. Hornbuckle

INVOICE

78136

DATE

5/29/26



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Spleen

The **spleen** was enlarged and congested with minor heterogenous parenchymal changes. Enhanced mesentery was noted throughout the cranial abdomen owing to the ascites, yet also potential for inflammation.

Liver

The **liver** was swollen and mildly irregular with slight heterogenous changes with a right cranial 2.8 cm mass adjacent to the gallbladder. This may be non-neoplastic. There is a potential for abscessation, nodular hyperplasia, round cell neoplasia, and carcinoma are all possible. If accessible SDEP 12 approach to FNA is indicated. The portal vein was not visible. Given the ascites I cannot rule out a portal vein thrombosis causing a prehepatic portal hypertension and secondary ascites. 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

The upper **gastrointestinal tract** revealed envelopment by the pancreatic pathology with some anechoic stasis and duodenal spasming. The distal small intestine and colon were unremarkable other than minor thickening.

Pancreas

The **pancreas** revealed extensive, mixed hypoechoic and hyperechoic changes on the right and left base of the pancreas.

Free Abdomen

A moderate amount of mildly echogenic ascites was noted throughout the abdomen.

ULTRASONOGRAPHIC FINDINGS

Swollen, irregular liver with heterogenous changes with a right cranial liver mass.

Upper GI tract was enveloped by the pancreatic pathology with anechoic stasis and duodenal spasming.

Extensive mixed hypoechoic and hyperechoic pancreatic changes.

Ascites.



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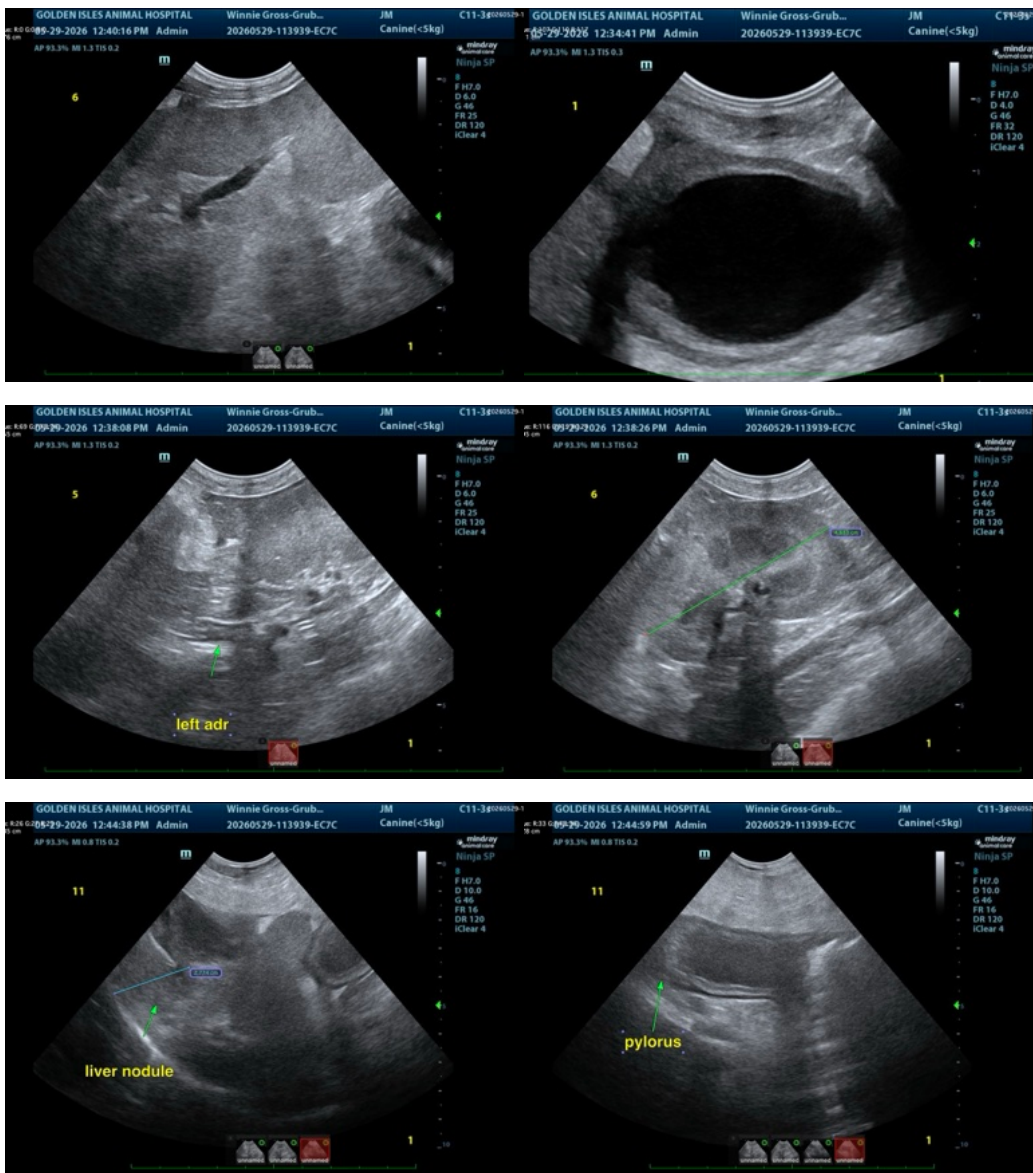
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INTERPRETATION OF THE FINDINGS & FURTHER RECOMMENDATIONS

I strongly recommend ultrasound-guided FNA of the general liver, liver nodule if accessible, pancreas and abdominocentesis and cytospin of the free fluid.

I recommend treating for pancreatitis until further diagnostics further define the patient and outcome. Recheck sonogram is recommended in 48-72 hours.





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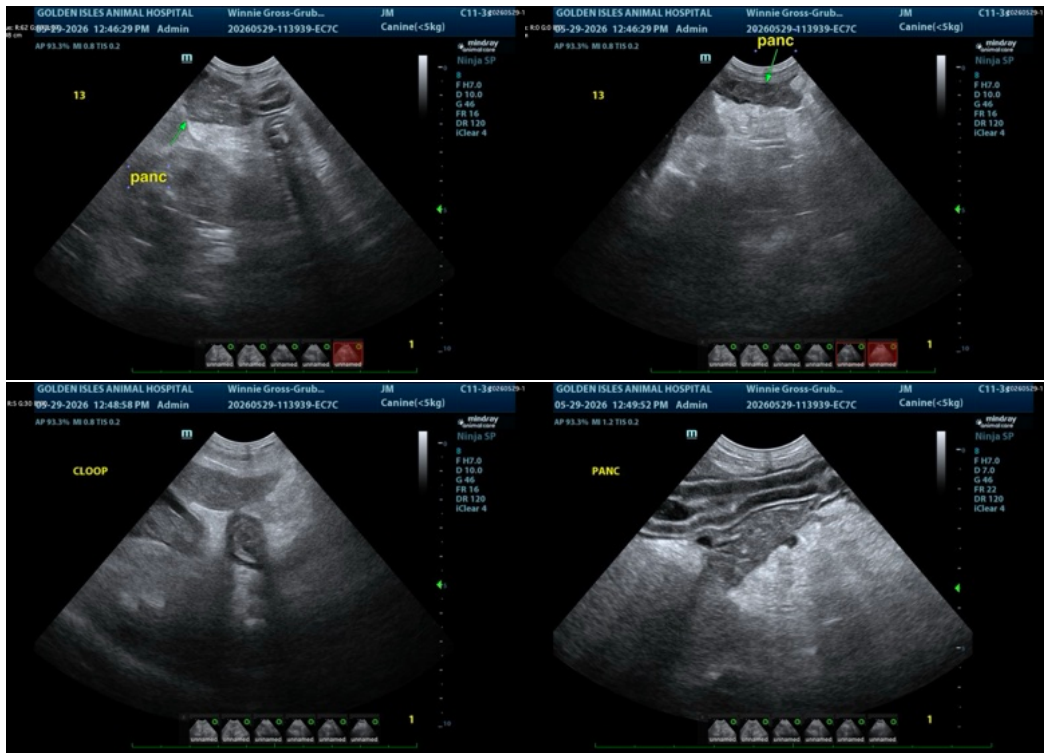
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The information and recommendations provided are based on the images presented by the referring veterinarian/sonographer. No evaluation can be communicated regarding pathology that was not visible in the image/video clips provided.

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 (CFM), Cert. IVUSS, CEO of SonoPath.com

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