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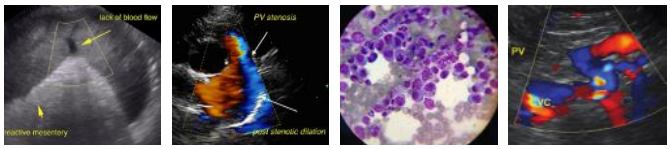

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1-800-838-4268 info@sonopath.com

PATIENT

Oyster Norris

SPECIES

Feline

BREED

DSH

SEX

Neutered male

AGE

7 years

WEIGHT

10.1 pounds

INTERPRETED BY

R. McKenzie Daniel, DVM, DABVP (Canine and Feline)

IMAGING PERFORMED BY

Nicole Gotfredson

HOSPITAL NAME

Buffalo Veterinary Clinic

REFERRING VET

Dr Garry Gotfredson

INVOICE

DATE

3/21/2022

PRESENTING CLINICAL SIGNS

History: Chronic vomiting. Currently not wanting to eat.

Abnormal PE/Chem/CBC/UA Results: CBC/Chem- WNL

ULTRASONOGRAPHIC EXAMINATION OF THE ABDOMEN

Urinary System

The urinary bladder, trigone, cystourethral junction, and visible pelvic urethra to a depth of 2 cm exhibited normal thickness and tone. Anechoic urine was present in the lumen with no uroliths or sediment. The ureteral papillae were normal. The ureters were not visible which is normal. No evidence of inflammatory or neoplastic changes were noted.

Normal size and margination were present in the kidneys. A normal 1:3 cortex / medulla ratio and normal corticomedullary definition were maintained. The echogenicity of the cortex was similar to or slightly less than normal liver parenchyma while the medulla echogenicity was hypoechoic to the cortex with no evidence of pelvic dilation. The left kidney measured 3.8 cm in length, the right kidney measured 4.0 cm in length.

The area of the aortic trifurcation was free of pathology.

Adrenal Glands

The left adrenal gland was uniform in size and contour with a uniformly hypoechoic parenchyma. The left adrenal gland measured 0.33 cm width. No evidence of pathology in the area of the right adrenal gland was noted.

Spleen

The spleen exhibited a finely textured and homogenous parenchyma which was hyperechoic to the liver and renal cortical parenchyma. The capsule was smooth and regular without apparent expansion. The splenic vasculature at the hilus was normal in volume with no evidence of congestion or thrombosis. Acute to chronic inflammatory, neoplastic, or benign parenchyma changes were not noted. The spleen measured 0.65 cm in width.

Liver

The liver was subjectively normal in size, structure, and contour. The liver parenchyma was uniform and hypoechoic to the spleen with a mild coarse echotexture. The hepatic and portal vasculature were normal in appearance without signs of congestion. The gallbladder was non-distended in size with thin walls and primarily anechoic luminal content with mild luminal debris primarily along the inner luminal wall. No evidence of gallbladder or peripheral inflammation. The cystic and common bile ducts were normal.

Gastrointestinal

The stomach presented intact wall layering with a normal wall layer ratio. The lumen of the stomach contained mild retained chyme and luminal gas with no signs of ileus, obstruction or foreign material. The gastric body wall measured 0.21 cm.

The small intestine presented intact wall layering with 1:3 muscularis/mucosa ratio. The lumen of the small intestine was empty with no signs of ileus, obstruction or foreign material. The duodenum wall measured 0.25 cm. The jejunum wall measured 0.25 cm.

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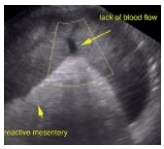
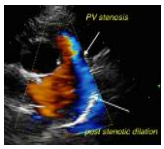
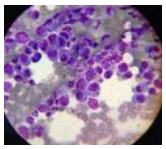
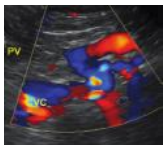

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Normal visible colon wall layers were present with apparent formed feces in lumen.

Pancreas

The pancreas exhibited generalized mild enlargement most notable in the left pancreatic limb with mildly swollen to asymmetrical pancreatic contour. Hypoechoic parenchyma with evidence of subtle peripancreatic reactive mesentery was noted.

Free Abdomen

No omental lymphadenopathy, masses or peritoneal effusion was observed.

ULTRASONOGRAPHIC FINDINGS

- Pancreatitis.
- Overtly normal gastrointestinal tract with possible mild gastric hypomotility.
- Minor gallbladder debris-likely incidental given lack of reported hepatic enzyme elevations or cholestasis.

INTERPRETATION OF THE FINDINGS & FURTHER RECOMMENDATIONS

The pancreatic presentation is suggestive of moderate active pancreatitis and most likely cause of the patient's clinical signs. A potential flare up of chronic pancreatitis is possible. Structurally insignificant inflammatory bowel or inflammatory hepatopathy i.e. triad disease could be considered if persistent gastrointestinal signs, evidence of weight loss or previous history of hepatic enzyme elevations. Empirical therapy for moderate active pancreatitis with monitoring of clinical response would be reasonable.

A GI panel to include PLI/TLI/Cobalamin/Folate for further assessment of the pancreas and to rule out occult intestinal disease is recommended.

Feline Pancreatitis & Pancreatic Neoplasia

http://www.sonopath.com/FelinePancreatitis
http://www.sonopath.com/FelinePancreaticCarcinoma

Description: Feline pancreatic disease is typically comprised of two major subtypes: chronic lymphoplasmacytic pancreatitis and a more acute necrotizing form of pancreatitis. Both forms may be associated with inflammatory bowel disease (IBD) and cholangitis due to the unique anatomical location of the feline pancreatic duct as it enters the duodenum adjacent to the common bile duct (triaditis).

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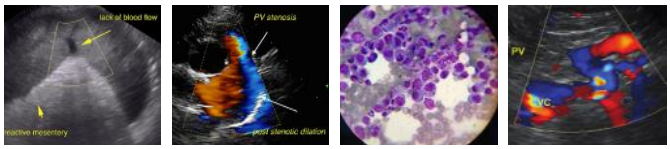

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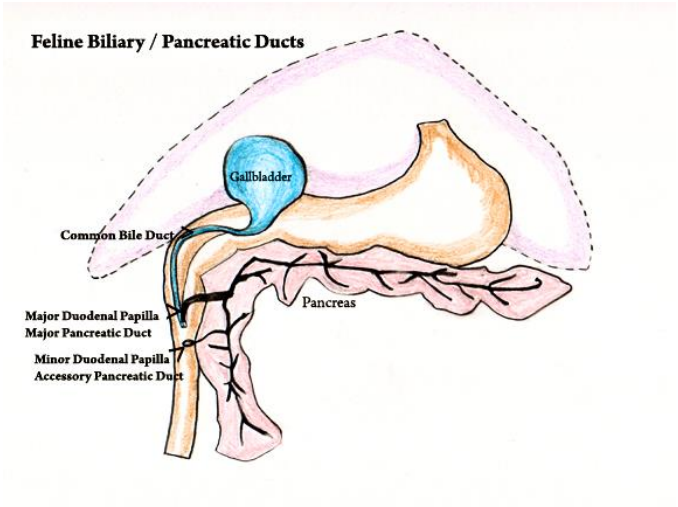
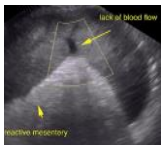
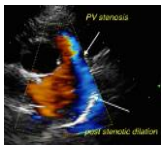
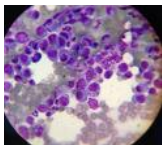
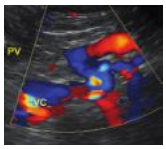


Figure Design: K. Vazquez & E. Lindquist

Clinical signs: Clinical signs are often vague. Cats typically present with lethargy and anorexia that may or may not include abdominal discomfort. Vomiting is only seen in approximately one third of all cases. Concurrent disease, such as cholangitis or hepatic lipidosis, may result in icterus, which can also be caused by post-hepatic pancreatic inflammation. In patients with liver involvement, coagulopathies may also develop. In the acute form, ascites may occur, and in the chronic form, concurrent nephritis may exist. Both forms can predispose the patient to diabetes mellitus, which may turn out to be temporary or permanent. Animals with concurrent liver involvement often have increased ALT, ALP, and bilirubin, and may also have elevated WBC counts. In all cases, low albumin, calcium, and magnesium are poor prognostic indicators.

Diagnostics: A diagnosis of pancreatitis is based on supportive information gathered from lab work and ultrasonography. The feline pancreatic-specific lipase assay (fPLI) is designed to evaluate for elevated blood lipase levels that are of pancreatic origin. Because increased levels of fPLI are indicative of pancreatic inflammation, the test is useful for monitoring patient progress and recurrence. B_{12} and folate levels should also be assessed, as patients with pancreatitis are often deficient in vitamin B_{12} secondary to chronic IBD; this deficiency is thought to propagate chronic pancreatitis.

Ultrasonography offers a noninvasive means of evaluating of pancreatic anatomical changes. It can be used to help distinguish between severe forms of inflammation (i.e., hypoechoic pancreatic nodular changes with concurrent hyperechoic peripancreatic fat, with or without ascites) and more chronic forms (i.e., focal or diffuse moderately thickened hypoechoic pancreatic limbs, with or without evidence of fibrosis or calcifications) as well as differentiate among other pancreatic conditions, such as neoplasia, cystic structures, and abscesses. Because pancreatic carcinoma and lymphoma can resemble pancreatitis sonographically, ultrasound-guided or surgical sampling is critical and strongly recommended in most cases. Pancreatic carcinomatosis is a relatively frequent finding in middle-aged to geriatric cats and can also resemble pancreatitis. In cases of pancreatic carcinomatosis, one may observe on ultrasound coalescing nodular omental changes, which are often accompanied by progressively accumulating ascites and potential hepatic or lymphatic metastatic lesions. Cytospin and the immediate slide preparation of sediment derived from the free fluid may allow for a more reliable identification of exfoliating carcinoma cells than submitting simple fluid for analysis; the latter often consists of potentially degenerative neoplastic cells that may be unrecognizable to a cytologist. Alternatively, fine needle aspiration (FNA) of the hypoechoic portions of the omental-pancreatic mass is an effective approach for confirming pancreatic neoplasia to the exclusion of pancreatitis or necrosis. Furthermore, ultrasonography allows for the assessment of other abdominal organs that may be concurrently affected in cases of triaditis or nephritis.

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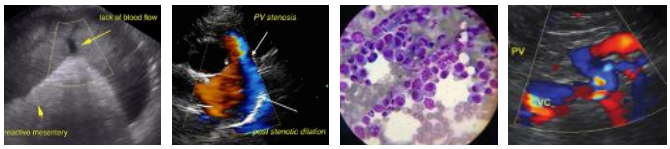
The general consensus among practitioners and researchers is that abdominal ultrasound combined with fPLI testing comprises the most sensitive and specific diagnostic screening currently available. For cats, this combination has been shown to be more sensitive and specific than abdominal CT scans. Upon sonography, however, it is important not to confuse chronic or age-related changes in the feline pancreas with those that are specific to pancreatitis, such as increased pancreatic width, pancreatic duct dilation, and echogenic alterations. Thus, if clinically appropriate, sampling via ultrasound-guided fine needle aspiration (FNA) or core biopsy should be performed when necessary to help differentiate active disease from chronic or age-related changes. Recent studies show that endoscopic ultrasound is ideal for identifying the pancreas in obese patients where increased intra-abdominal fat can influence the echogenicity of the pancreas; however, changes seen in the peripancreatic adipose tissue via transabdominal ultrasonography are also indicative of pancreatic inflammation.

Treatment: Fluid volume maintenance, pain management, treatment and prevention of infection, and nutritional support are the key components to therapeutic success. Treatment recommendations are as follows:

1. It is important to continue feeding feline patients, as there is a high risk of hepatic lipidosis; one must also encourage adequate enterocyte nutrition. If necessary, an esophagostomy or a PEG tube placement is recommended. One should not administer a protein-restricted diet unless signs of hepatic encephalopathy are present. Patients that are prone to vomiting can often still receive nutrition via a nasogastric tube that will permit the trickle-feeding of a concentrated diet, such as CliniCare® Feline Liquid Diet (Abbott Animal Health). Recent research suggests that the trypsin cascade is not reactivated by further introduction of dietary fat; therefore, simply getting these patients to eat is initially more important than what they eat. Appropriate antiemetics can be utilized; however, metoclopramide is not recommended, as there is a risk of compromising pancreatic perfusion (see below for anti-emetic recommendations).
2. Administer IV fluid therapy that consists of a balanced electrolyte solution supplemented with B vitamins and potassium chloride (20mEq/L or as necessary based on serum K levels) and magnesium sulfate (0.3-0.5mEq/kg/day as a CRI in D5W) if hypomagnesemia exists. Colloids, such as hetastarch (10ml/kg in LRS; up to one third of the dose can be given as a slow bolus), or plasma transfusions can be administered as deemed necessary, especially in patients with low albumin, ascitic fluid, concurrent hepatic disease, or coagulopathies. Vitamin B_{12} injections (0.25-1mg SQ/IM weekly for 4 weeks) should be given if this vitamin has been found to be deficient.
3. Administer broad-spectrum antibiotics if a suppurative form of pancreatitis is of concern (ampicillin at 20mg/kg IV TID and/or enrofloxacin at 2.5 mg/kg BID PO, IV, or IM). Note: Baytril yields very infrequent side effects in cats, including blindness, and ideally should be administered orally once vomiting has been controlled. Most cases of pancreatitis are not bacterial, but many afflicted patients may also have other underlying bacterial diseases, such as cholangitis.
4. Analgesia is imperative to facilitate healing and encourage eating. Abdominal palpation does not often elicit a typical pain response in a cat with pancreatitis and should not be relied upon to determine whether pain management should be pursued. A fentanyl patch or oxymorphone (0.1-0.2 mg/kg IM, SQ, or IV QID) works best; however, if these are not available, then butorphanol (0.4 mg/kg IM or SQ Q4hr) can be utilized, although it does not control well for visceral pain. One should not use nonsteroidal anti-inflammatory drugs (NSAIDs), as this class of drugs will decrease blood flow to the pancreas, which can exacerbate the disease.
5. One should administer H_2 receptor blockers (famotidine 0.5mg/kg IV Q12-24hr or PO BID) or a proton pump inhibitor (omeprazole 0.7 mg/kg PO Q24hr; however, it can be difficult to dose in cats).
6. Control of vomiting can be achieved with maropitant (1 mg/kg PO or SQ once daily for up to 5 days), mirtazapine (¼ of a 15 mg tablet PO Q3 days), dolasetron (0.6 mg/g IV Q24hr),



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odansetron (0.1-0.15 mg/kg slow IV push BID-TID PRN), or chlorpromazine (0.5mg/kg IM, SQ TID).

7. Dietary supplements: Denosyl (SAM-E) (90mg/day on an empty stomach as an antioxidant) and carnitine (250mg/day). If hepatic involvement is evidenced from lab work or ultrasound, one can supplement with Marin (a combination of milk thistle, zinc, and vitamin E) and Actigall (10-15mg/kg PO Q24hr) if biliary stasis or cholangitis is suspected. These supplements can be mixed into a slurry for tube feeding.
8. In very severe cases, dopamine (0.5mcg/kg/min as a CRI) can be initiated to assist in pancreatic (mesenteric) and renal microcirculation; however, it must be given very early in the disease process to be of benefit. This rate must be strictly controlled; infusing at a rate faster than the one recommended can lead to complications, such as increased systemic peripheral resistance. This situation should be monitored at a 24-hour facility due to the possibility for a multitude of adverse side effects.
9. In the case of lymphoplasmacytic or other immune-mediated forms of pancreatitis, as confirmed by aspirate or biopsy, prednisone at 0.5-1 mg/kg PO Q24hr on a weaning schedule may be utilized. Prednisone should not be used, however, in cases of suppurative pancreatitis or hepatic lipidosis.
10. Insulin therapy should be implemented in cases where patients become diabetic; however, blood glucose must be strictly monitored, as the insulin requirements may decrease or halt once the pancreatitis has resolved.

Treatment can take days to weeks, depending on the severity of the case at hand. Serial fPLI testing may be of use in monitoring the progression of the acute aspects of the disease. It is important to treat all facets of pancreatic conditions, as they occur most frequently in conjunction with intestinal and hepatic disease. These diseases are often chronic and need to be managed as such.

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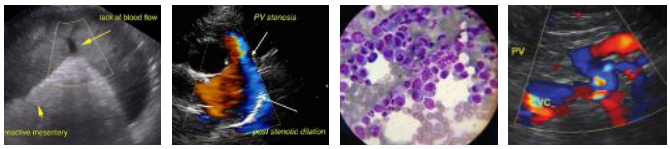

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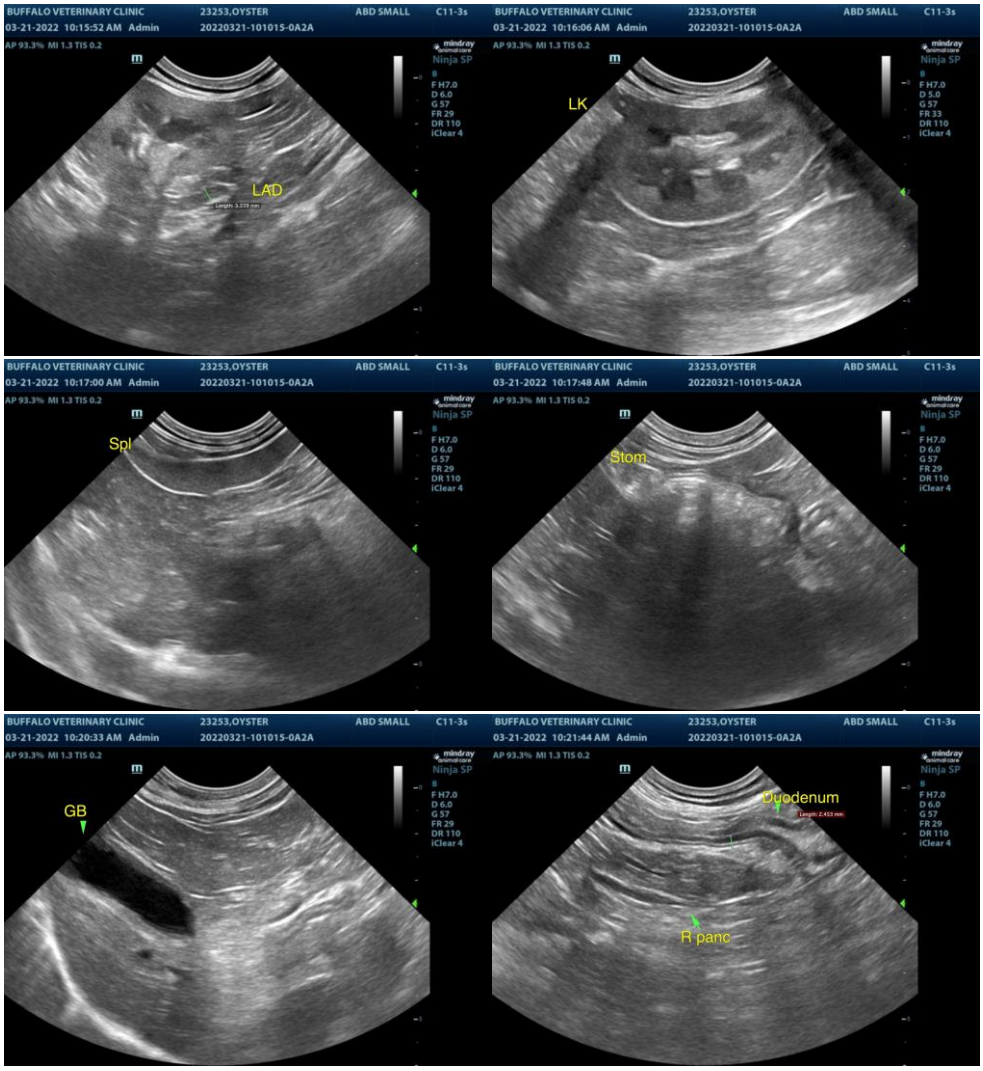

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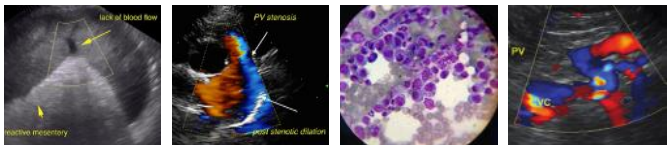

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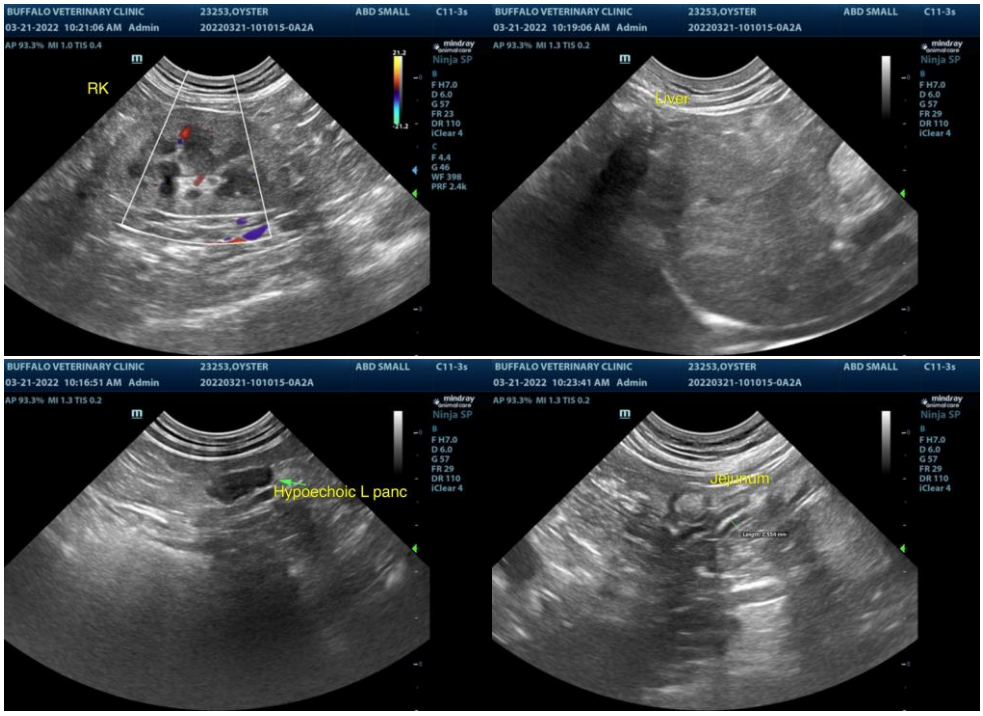

The information and recommendations provided are based on the images presented by the referring veterinarian/sonographer. No evaluation can be communicated regarding pathology that was not visible in the image/video clips provided.

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.

R. McKenzie Daniel, DVM, DABVP (Canine / Feline Practice)

info@SonoPath.com