SonoPath
Clinical Sonography & Telecytology
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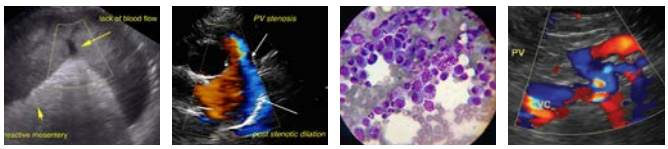

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1-800-838-4268 info@sonopath.com

PATIENT
Scout Taylor

SPECIES
Canine

BREED
Belgian Malinois

SEX
Neutered Male

AGE
18 Months

WEIGHT
27.8 kg

INTERPRETED BY
Eric Lindquist, DMV
DABVP, Cert. IVUSS

IMAGING PERFORMED BY
Donna Markland, DVM

HOSPITAL NAME
Island Mobile Paws

REFERRING VET
Central Island Vet Emergency Hospital

INVOICE
39317

DATE
7/8/22

PRESENTING CLINICAL SIGNS

Presented on July 7 for incoordination and pu/pd. Had severe hypernatremia and was hemoconcetrated with moderate azotemia on presentation. After 24 hours of fluid therapy, the electrolytes are normalized, but the azotemia is worse. PU/PD while on IV fluids. History of thorough workup for diarrhea and weight loss by board-certified internist in February. Workup included normal abdominal ultrasound and normal ACTH tests. Diarrhea mostly resolved now with occasional loose stool. Raw fed. No known toxins.
Abnormal PE/Chem/CBC/UA Results: July 7: HCT=65.6% SDMA=35 (0-14) Creatinine= 171 (44-159) Urea=9.3 (2;5-9.6) Na=175 (144-160) Cl=133 (109-122) TP=86 Glob=47 July 8 HCT=35.2 Neuts=12.86 (2.95-11.64) Platelets=122 (148-484) SDMA=34 Creat=389 Urea=13.5 Na=154 Cl=113 TP=56

ULTRASONOGRAPHIC EXAMINATION OF THE ABDOMEN

Urinary System

The **urinary bladder** presented a moderate amount of debris. A small calculus measured 4.0 mm. The patient may have passed a calculus recently.

The **prostate** was slightly heterogeneous and mildly irregular, measuring 1.58 cm.

The **kidneys** presented non-specific hyperechoic medullary rim sign with trace pyelectasia, some loss of corticomedullary definition, and increased cortical echogenicity. The left kidney measured 8.8 cm. The right kidney measured 8.8 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.

Spleen

The **spleen** was uniformly enlarged with relatively uniform parenchyma without evidence of masses. The capsule was mildly swollen. The spleen was folded upon itself and was mildly irregular. This is most consistent with hypersplenism and reactive hyperplasia deriving from splenic white or red pulp. However, early infiltrative disease, such as lymphoma or mast cell neoplasia can, at times, present in this manner but not suspected. 25g US-guided FNA would be best in order to ensure only reactive hyperplasia is present. If clinical signs fit with potential neoplasia or mast cell disease, then Benadryl injection (1 mg/pound IM) 15 minutes prior to FNA would be recommended.

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

The **stomach** was overdistended with fluid and stasis, consistent with ileus and/or uremic gastritis. The small intestine and colon were unremarkable.

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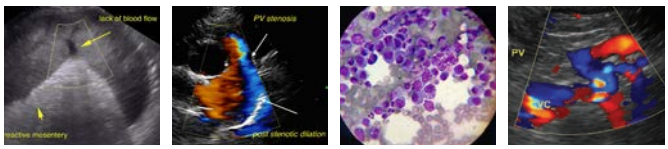

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Pancreas

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.

Free Abdomen

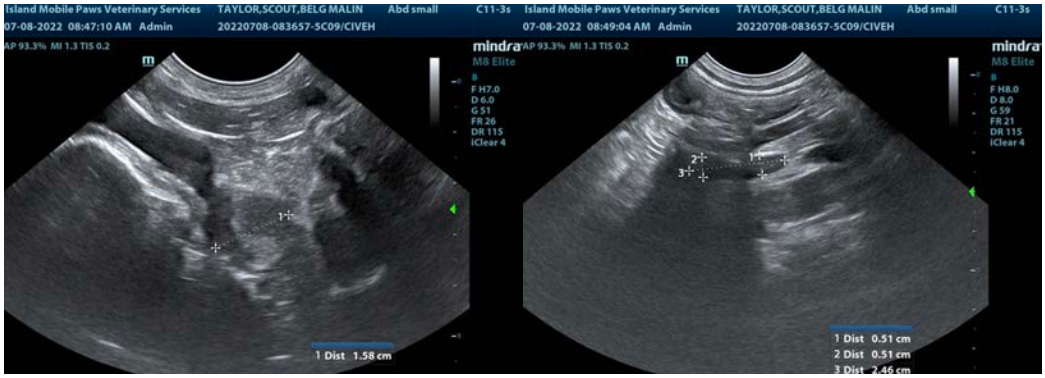
Sublumbar lymph nodes were slightly enlarged, reactive, measuring 1.5 cm x 0.5 cm.

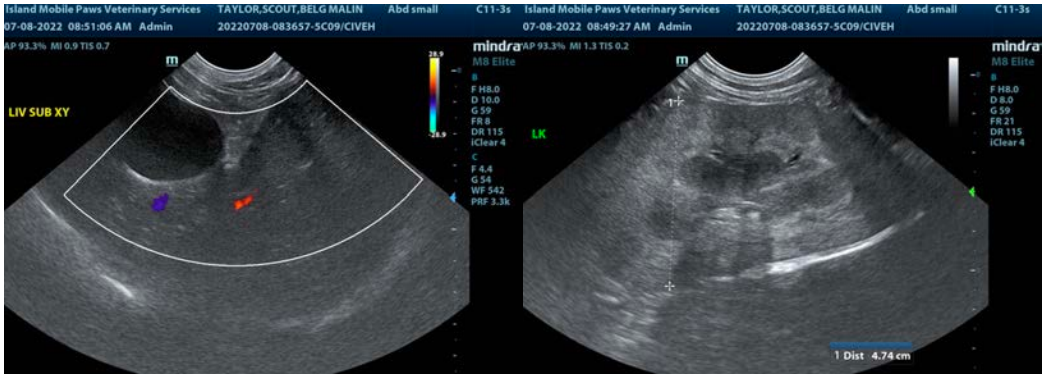
PRIMARY FINDINGS

- Acute on chronic renal changes with idiopathic medullary rim sign
- Urinary bladder debris
- Irregular prostate
- Enlarged, irregular spleen
- Gastritis pattern
- Reactive sublumbar lymph nodes

INTERPRETATION OF THE FINDINGS & FURTHER RECOMMENDATIONS

I'm concerned for toxin or infectious exposure to the kidneys with tubular damage, given the medullary rim sign. Recommend full urinary workup, culture and sensitivity, blood pressure, 72-hour IV fluid protocol, GI protectants, Ampicillin as suggested antibiotic. Renal biopsy would be ideal. Leptospirosis titers indicated. Assessment of patient history for toxin exposure indicated. Prognosis is very guarded. Recheck sonogram in 48-72 hours after therapy.





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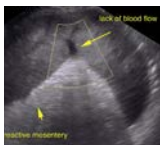
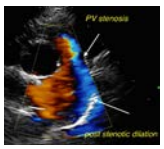
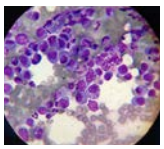
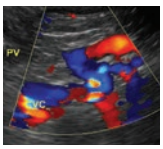

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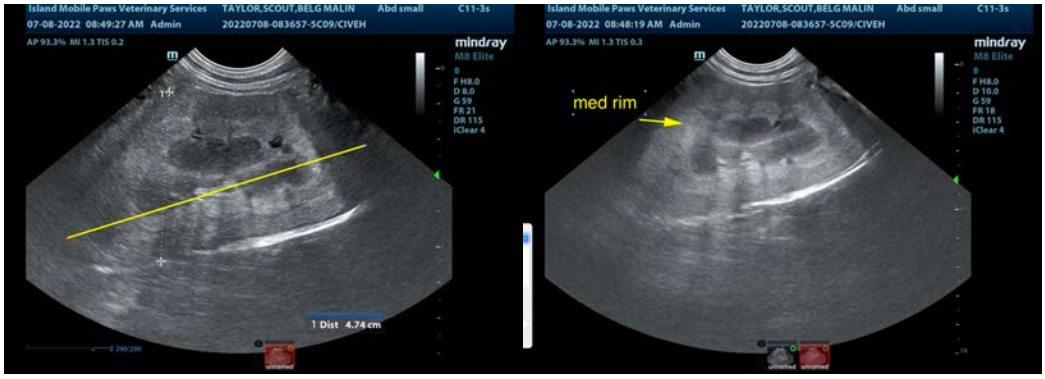
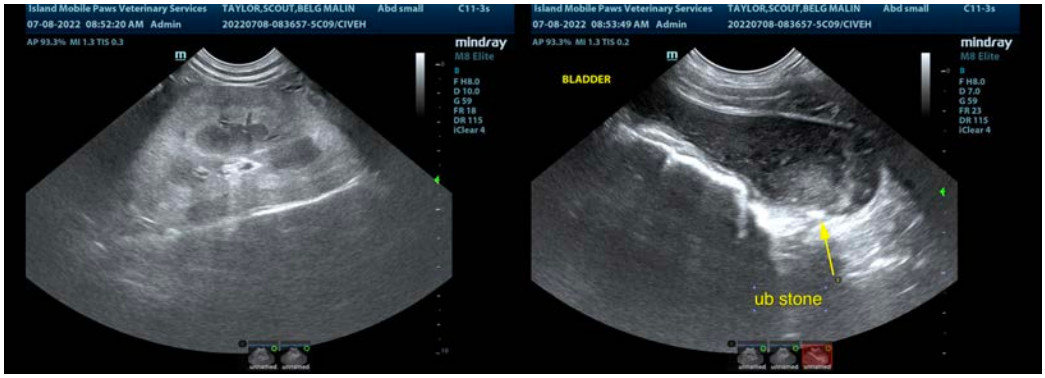


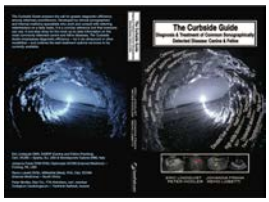


The information and recommendations provided are based on the images presented by the referring veterinarian/sonographer. No evaluation can be communicated regarding pathology that was not visible in the image/video clips provided.

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, Cert. IVUSS, CEO of SonoPath.com

info@SonoPath.com



The following is an applicable excerpt from the *Curbside Guide to Diagnosis & Treatment of Sonographic Disease* offered by SonoPath.com Lindquist, Frank, Lobetti, and Modler.

An essential quick guide for every general practitioner and sonographer.

https://sonopath.com/products/curbside-guide-editing-due-release-12012015

Acute Renal Failure

http://www.sonopath.com/ARF

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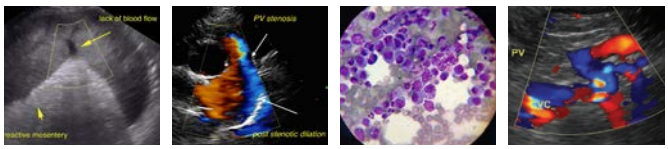

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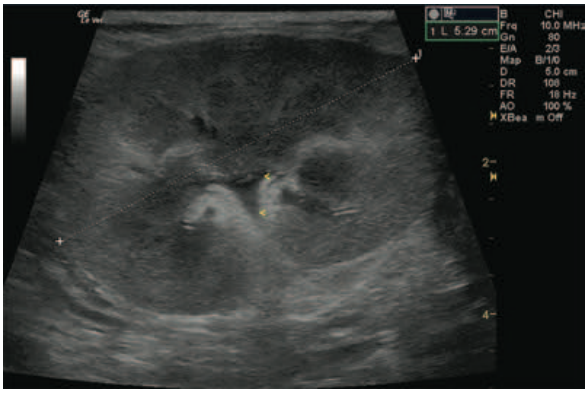
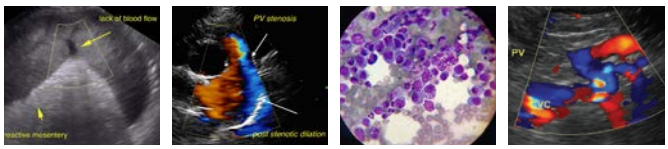


Long axis of the kidney in a cat with a renal transplant and acute renal failure. Note the generalized swelling of the kidney with loss of corticomedullary definition. The renal pelvis shows mild dilation with anechoic content (arrow heads). The renal crest and sinus are hyperechoic.

Description: Acute renal failure (ARF)—also referred to as acute kidney injury—is defined as a rapid deterioration in renal function that results in the accumulation of metabolic waste in the body. It is characterized by an impaired regulation of water and solute balances, and may be due to prerenal, postrenal, and/or primary renal causes. Prerenal azotemia reflects a reduced glomerular filtration rate (GFR), which is a consequence of renal hypoperfusion; it is not the result of structural renal damage. Immediate restoration of renal blood flow will reverse the azotemia over a period of time; however, if the hypoperfusion is severe or prolonged, or if there is prior renal dysfunction, acute primary renal failure due to ischemic acute tubular necrosis will be induced. Postrenal azotemia occurs when urine flow is obstructed or the excretory pathway is ruptured and there is subsequent urine resorption. Persistent urinary obstruction may cause irreversible renal damage. Early detection of postrenal azotemia will result in complete restoration of renal function. Acute tubular necrosis accounts for the majority of acute primary renal failure cases and is characterized by an abrupt and sustained reduction in GFR due to an ischemic or toxic renal insult. The conditions that incite ischemia are the same as those for prerenal azotemia; however, the duration of the ischemia is important. Nephrotoxins are a frequent cause of tubular necrosis. The high rates of blood flow and metabolic activity in the kidneys as well as their excretory function predispose dogs and cats to the toxic effects of drugs as well as endogenous or exogenous toxins.

Clinical Signs: The clinical course in acute tubular necrosis can be divided into three phases: an initiating phase, a maintenance phase, and a recovery phase. The initiating phase, which is marked by the onset of renal injury, is the period in which there is the greatest potential for preventing or reversing tubular damage and the progression to overt renal failure because it is during this period that renal cell damage develops. The challenge, however, is that the initiating phase may only become evident in retrospect as it generally lacks characteristic signs. The maintenance phase is characterized by the onset of oliguria (i.e., urine production is less than 1ml/kg/hour). The onset of this phase typically occurs during the first 24 hours, but may be delayed for up to 1 week. The duration of this phase is highly variable, but usually persists for up to 2 weeks. It is characterized by: fluid and electrolyte imbalances, including an alteration in hydration; hyponatremia; hyperkalemia; high anion gap metabolic acidosis; hypocalcemia; hyperphosphatemia; and azotemia. Clinical signs include gastrointestinal, hematological, and neurological manifestations of renal failure. The recovery phase commences when the GFR increases, which consequently slows down and reverses the azotemia. There is a progressive increase in urine volume, and although the tubular function begins to improve, it nevertheless remains impaired. Diuresis persists because of the diminished ability of the tubules to reabsorb sodium and respond to vasopressin. Clinical manifestations observed in the maintenance phase persist into the recovery phase. In some patients, infections and/or gastrointestinal bleeding may occur. Sites of infection include the respiratory



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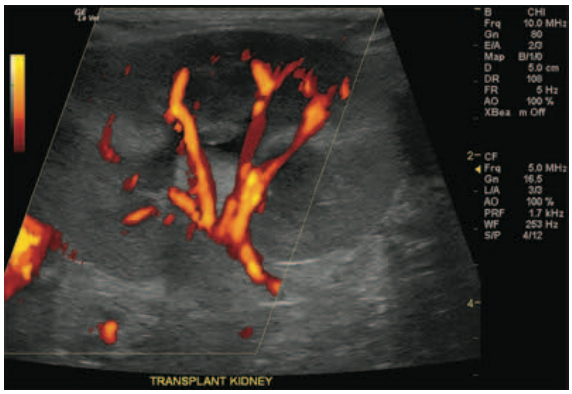
tract, operative sites, and the urinary tract. Septicemia may also occur and is sometimes the result of intravenous and urinary indwelling catheters.

Diagnostics: Extraurinary disorders that produce prerenal azotemia are associated with concentrated, hypersthenuric urine, which contains a relatively low concentration of sodium and high concentration of creatinine. ARF is typically characterized by enlarged or swollen kidneys, elevated hematocrit, and azotemia. Urine is isosthenuric or minimally concentrated, and contains high concentrations of creatinine. Proteinuria or glycosuria may also accompany this condition. The sediment will show casts and RTE cells. Complete anuria is usually associated with postrenal azotemia. Features that are typical for acute tubular necrosis include: anuria in the absence of a urinary tract obstruction or rupture; severe proteinuria; significant hematuria with red cell casts; and prolonged oliguria. In these cases, a diagnostic renal biopsy is indicated.

Treatment: Most patients with ARF are volume depleted. Fluid therapy is indicated to correct dehydration, which will restore adequate renal perfusion and may prevent further renal damage. If the etiology was prerenal in origin, then urine volume will increase. In the maintenance phase, fluid therapy should be directed toward maintaining fluid balance and preventing both overhydration and dehydration. In cases of renal disease it is important that only maintenance needs and ongoing losses are attended to as overhydration can develop if there is reduced renal function. Insensible losses are calculated at 20 ml/kg/day. Aggressive fluid therapy during the recovery phase may perpetuate polyuria. As the urine volume stabilizes, the volume of fluid administered should be reduced correspondingly. Because dehydration may occur during this phase, one should monitor body weight and clinically assess the hydration status as fluid therapy is being reduced. Oliguric patients who are unresponsive to fluid volume replacement can be treated with mannitol, furosemide, and/or dopamine in an attempt to increase GFR and urine volume. Hyperkalemia is commonly associated with the maintenance phase of ARF. Concentrations greater than 6 mmol/l may require treatment with sodium bicarbonate, dextrose, insulin and/or calcium gluconate. Hemodialysis should be considered in patients with severe, persistent uremia, acidosis, or hyperkalemia. It may also be used to treat overhydration and hasten the elimination of nephrotoxins.

Conclusion: Because ARF is frequently iatrogenic and associated with nephrotoxic drugs or inadequate fluid therapy, prevention is the best therapy.



Long axis of the kidney of the same cat as in the previous title image. Note the non-uniform power Doppler signal distribution with significant hypovascularity of the cranial pole compatible with regional infarction and transplant failure.

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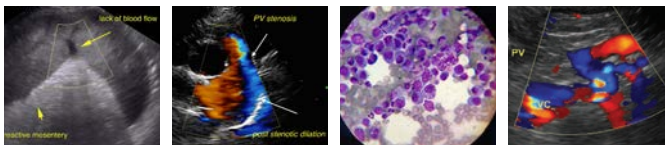

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Acierno MJ, Maeckelbergh V. Continuous renal replacement therapy. *Compend Contin Educ Vet* 2008;30:264-72.

Grauer GF. Early detection of renal damage and disease in dogs and cats. *Vet Clin North Am Small Anim Pract* 2005;35:581-96.

Labato MA. Strategies for management of acute renal failure. *Vet Clin North Am Small Anim Pract* 2001;31:1265-87.

Ross L. Acute kidney injury in dogs and cats. *Vet Clin North Am Small Anim Pract* 2011;41:1-14.