



PATIENT

Oliver Lewis

SPECIES

Canine

BREED

Collie Cross

SEX

Neutered Male

AGE

8 Months

WEIGHT

43

INTERPRETED BY

Eric Lindquist, DMV
DABVP, Cert. IVUSS

IMAGING PERFORMED BY

Maggiulli

HOSPITAL NAME

Willamette VH

REFERRING VET

Maggiulli

INVOICE

23834

DATE

8/7/23

PRESENTING CLINICAL SIGNS

Presented 8/4 weakness, lethargy, collapse. Vomited 2x. Temp 104, febrile. hospitalized, became non ambulatory, NG tube placed, u cath placed, p producing copious amounts of hemorrhagic gastroenteritis, worsening mentation, azotemia worsening

Abnormal PE/Chem/CBC/UA Results: Forelimb rads- These radiographic changes are consistent with metaphyseal osteopathy, formally known as hypertrophic osteodystrophy. 4DX Tick snap = negative Parvo Negative CBC = WBCs 19.3k, Nuets 16.08k, Monos 1.45k, platelets 92k smear slide review = multiple platelet clumps seen chem = BUN 35, ALP 226 EPOC = K 3.4, iCa 2.0, BUN 32 UA = USG 1.018, quiet sediment 8/6- EPOC: cA 1.79, cL 134, Crea 3.49, K 5.3, Na 161, pH 7.302, po2 64.3, BUN 83, so2 90.1 PT/PTT: 14.0 sec (wnl)/PTT 199 (elevated) 8/7- CBC- HCT 38.7%, MONO 2.16, EOS 0.04, PLT 19, PTT-199 CHEM 10- Cre 3.3, BUN 83, ALB 1.8, ALT 277, ALP 1348 EPOC - HCT iCA 1.72, CL 136, CRE 3.3, NA 165, PH 7.347, BUN 79, PCV 34%/6g/dl

ULTRASONOGRAPHIC EXAMINATION OF THE ABDOMEN

Urinary System

The **urinary bladder** revealed a minor amount of sand, nonobstructive, measuring approximately 7.0 mm. Bladder wall was unremarkable. Foley catheter was in proper position.

Both **kidneys** were swollen with ill-defined hyperechoic cortical changes and loss of corticomedullary definition. No pyelectasia was noted. This is a nonspecific presentation, suspicious for toxin exposure or infectious agents. No evidence of primary disease. The left kidney measured 7.2 cm. The right kidney measured 5.9 cm.

Adrenal Glands

The **adrenal glands** were not visualized.

Spleen

The **spleen** presented a smooth homogeneous parenchyma hyperechoic to liver and renal cortical parenchyma. The capsule was smooth without noticeable expansion or deviation from within the spleen or adjacent pathology. The splenic vasculature demonstrated normal volume without signs of congestion or thrombosis. No sonographic evidence of acute or chronic inflammatory, neoplastic, or infarctual changes were noted.

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. The hepatic veins were slightly dilated.

Gastrointestinal

The upper **gastrointestinal tract** in this patient revealed minor, edematous wall. There was no evidence of foreign bodies. Minor areas of fluctuant fluid accumulation were noted within the lumen with hyperperistalsis. This pattern continued to the ileocecal valve. The colon revealed a fluid filled



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lumen. This presentation is most consistent with gastrointestinal irritation/inflammation without obstruction.

Pancreas

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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.

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Other

The **transdiaphragmatic view** revealed comet tail lung pattern, consistent with alveolar disease. Chest radiographs +/- echocardiogram are indicated.

SEX

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A rapid view of the **heart** revealed poor contractility and arrhythmogenic activity. Assessment for shock is indicated.

ULTRASONOGRAPHIC FINDINGS

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- Suspect acute renal insult
- Urinary bladder sand
- Comet tail lung pattern
- Concurrent gastroenteritis
- Poor contractility and arrhythmogenic activity in the heart- assessment for shock is indicated.

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INTERPRETATION OF THE FINDINGS & FURTHER RECOMMENDATIONS

Screening for Addisons is warranted yet toxin or infectious exposure should be considered. Leptospirosis titers is indicated. Prognosis is very guarded depending upon response to therapy. Bladder sand is odd for this age patient. Ethylene glycol toxicity should be considered as a potential.

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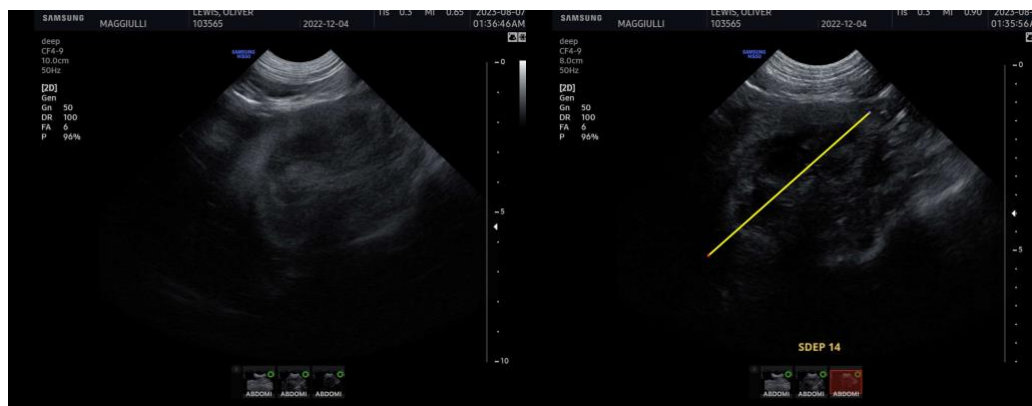
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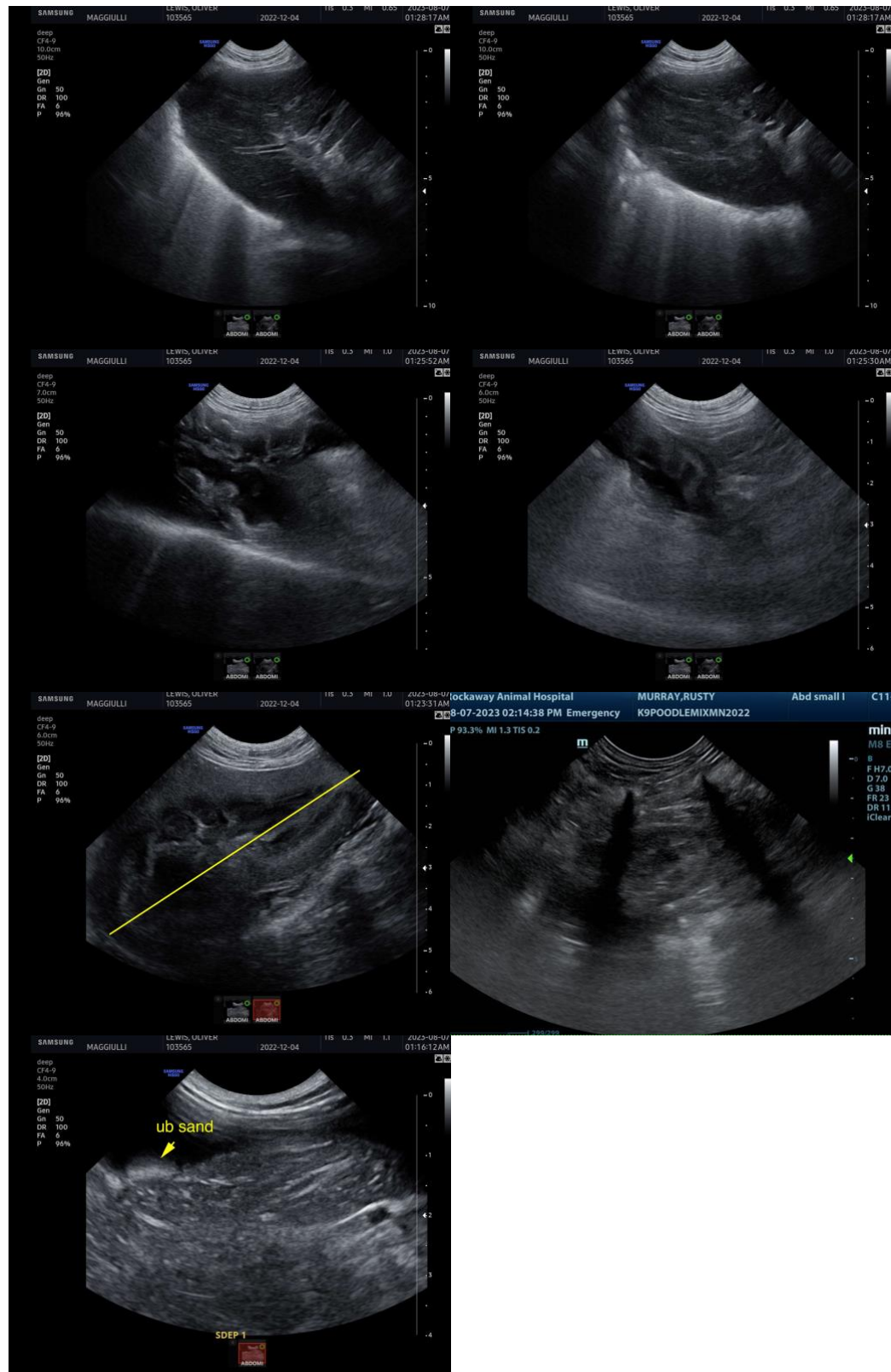
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The information and recommendations provided are based on the images presented by the



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referring veterinarian. No evaluation can be communicated regarding pathology that was not visible in the image/video clips provided.

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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.

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

BREED

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Acute Renal Failure

SEX

Neutered Male

<http://www.sonopath.com/ARF>

AGE

8 Months

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.

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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

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

Diagnosics: Extraordinary 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.

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

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