



PATIENT

Frank Ward

SPECIES

Canine

BREED

Mastif

SEX

Male

AGE

5 Years

WEIGHT

136 Pounds

INTERPRETED BY

Eric Lindquist, DMV
DABVP, Cert. IVUSS

IMAGING PERFORMED BY

Griffin

HOSPITAL NAME

Northside VC

REFERRING VET

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8/16/22

PRESENTING CLINICAL SIGNS

History: Patient presented down unable to walk. Temp 104.5 F

Abnormal PE/Chem/CBC/UA Results: 4DX: Negative CBC: RBC 10.33, HCT 66%, HGB 23, WBC 32%, Neu 23.88, sus bands, Mono 3.98, PLT 116 CHEM: Glu 177, SDMA 46, Creat 3.0, BUN 92, PHOS 14.2, TP 8.4, Glob 5.8, ALKP 382, Chol 432, Na 162 U/A: Cystocentesis USG 1.050, pH 7.0, 5 /HPF RBC >50 /HPF BACr * Suspect presence BACc * Suspect presence sqEPI None detected nsEPI 3 - 5 /HPF HYA None detected nhCST Suspect presence CRY <1 /HPF CaOxDi

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 were noted. Ureteral papillae were normal. The pelvic urethra was imaged 2.0 cm beyond the cystourethral junction. The prostate was uniform, measuring 4.3 cm- no evidence of pathology.

The **kidneys** revealed normal size and structure, corticomedullary definition and ratio for this age. The cortices presented largely uniform texture with normal echogenic relationship to liver and spleen. Medullary structure differed distinctly from the cortex and no evidence of pelvic dilation was present. The capsules were acceptably uniform without significant irregularities. The left kidney measured 7.0 cm. The right kidney measured 6.0 cm.

Adrenal Glands

The **right adrenal gland** was slightly irregular in contour. The right adrenal gland measured 1.0 cm in width.

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** revealed increased portal markings and mild heterogeneous parenchymal changes. The gallbladder and common bile duct were unremarkable.

Gastrointestinal

The **stomach** revealed concentric gastric thickening with echogenic mucosal remodeling. No overt loss of mural detail noted. The small intestine and colon were unremarkable.

Pancreas

The **pancreas** was nebulous with mild heterogeneous parenchymal changes. Some minor inflammation is likely.

Free Abdomen



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The mesenteric **lymph nodes** were mildly enlarged with a reactive pattern, measuring up to 3.0 cm x 1.5 cm. FNA, cytology and culture are indicated. Reactive mesentery was noted around the enlarged lymph nodes.

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ULTRASONOGRAPHIC FINDINGS

- Mesenteric lymphadenopathy with reactive mesentery
- Unremarkable kidneys- acute insult is suspected
- Mild hepatic remodeling
- Gastric thickening with echogenic mucosal remodeling

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INTERPRETATION OF THE FINDINGS & FURTHER RECOMMENDATIONS

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Acute renal failure, Leptospirosis or toxin exposure possible. Occult neoplasia is possible yet less likely. Leptospirosis titers is indicated. FNA of the enlarged lymph nodes is indicated. Urine culture and sensitivity and blood pressure is indicated.

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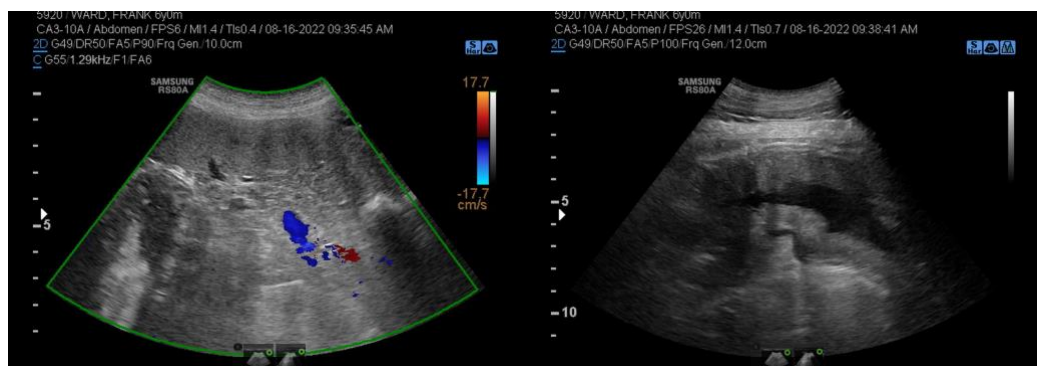


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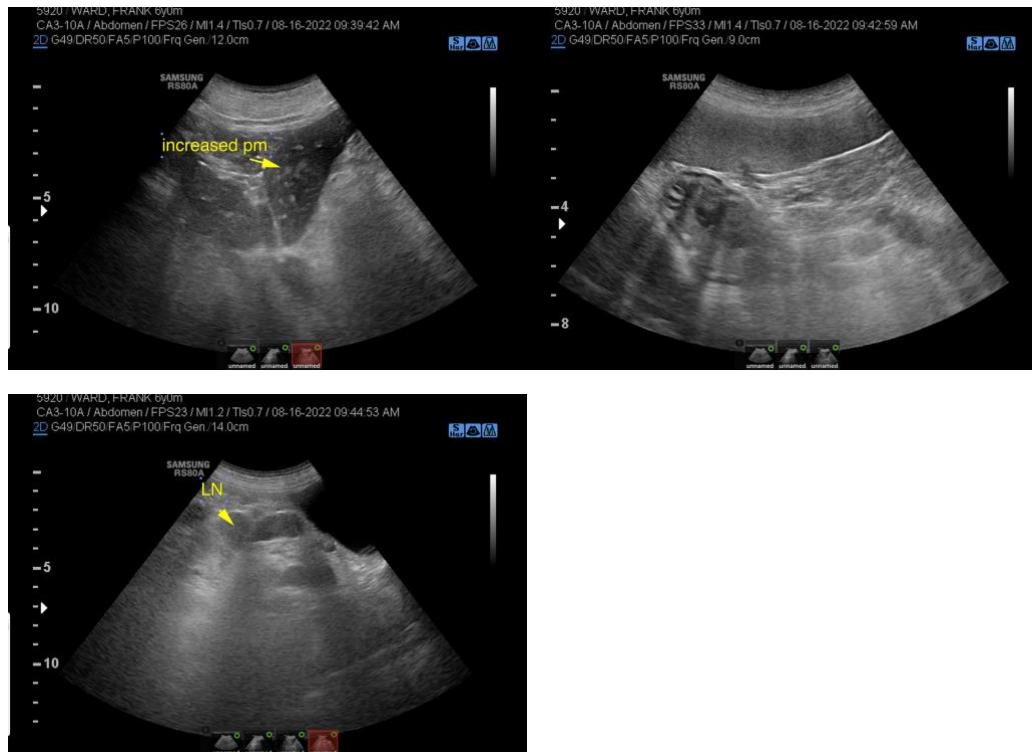
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The information and recommendations provided are based on the images presented by the referring veterinarian. No evaluation can be communicated regarding pathology that was not visible in the image/video clips provided.

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Fever of Unknown Origin

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

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Description: The definition of a fever of unknown origin (FUO) has not been clearly defined for animals. Currently, it is either understood to be a fever that does not resolve within the period one would expect for a “self-limiting infection” being treated with appropriate antimicrobial therapy, or that for which an underlying diagnosis has not been determined despite considerable diagnostic effort. The common causes of FUO were summarized concisely in a presentation at the American

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College of Veterinary Internal Medicine 2004 Forum. The presenters synthesized information from three veterinary papers on the subject, which suggested the following:

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Final Diagnosis	Bennett (dogs & cats)	Dunn and Dunn (dogs only)	Lunn (dogs & one cat)	Total
Infection	21	16	10	47
Immune	18	22	6	46
Bone marrow disease	4	22	2	28
Neoplasia (outside marrow)	0	10	2	12
Miscellaneous	2	12	2	16
No diagnosis	0	19	2	21
TOTALS	45	101	24	170

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The types of infection diagnosed in this case series were varied, ranging from discospondylitis (8 cases), blastomycosis (6), and bacterial endocarditis (4), to leishmaniasis (1), prostatitis (1), and *Ehrlichia canis* infection (1); a multitude of other infectious causes also fell within the spectrum. Of the cases in which immune-mediated disease was found, 44% had immune-mediated polyarthritis. Bone marrow diseases included myeloproliferative disease, myelodysplasia (8), lymphocytic leukemia (8), myeloma (3), chronic granulocytic leukemia (3), lymphoblastic leukemia, and malignant histiocytosis. The types of neoplasia located outside the bone marrow included lymphoma (6), metastatic disease (2), and neoplasms of the lung, spleen, and stomach. Finally, miscellaneous diseases included hypertrophic osteodystrophy (6), meningitis (3), portosystemic shunt (3), lymphadenitis (2), panosteitis, and intervertebral disc disease. Overall, the most common causes across all cases were polyarthritis (44), lymphoid neoplasia (15), discospondylitis (8), myelodysplasia (8), hypertrophic osteodystrophy (6), and blastomycosis (6).

Clinical Signs: Animals usually present with either persistent or waxing and waning fevers ranging from 103°F to 106°F. Other clinical signs depend on the underlying cause of the fever. Careful and thorough physical examination is required to assess potential causes.

Diagnostics: FOU etiologies are partly related to geography, and thus locale or travel history should factor into a practitioner's diagnostic approach. A patient's lifestyle may also provide clues regarding exposure to certain etiologic agents. Therefore, conducting a thorough history can unveil important pieces of the diagnostic puzzle. Physical examination is especially important and should include an inspection of all accessible lymph nodes, palpation and movement of the joints, a fundic



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examination, a neurological evaluation, spinal and limb palpation and range of motion tests, and a rectal examination.

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A minimum database should include a CBC reviewed by a clinical pathologist, as well as a biochemical profile and urinalysis. Retroviral testing should also be considered in cats. In areas where tick-borne disease is prevalent, in-house testing should be performed early. Advanced laboratory work can include: urine culture, blood culture, and infectious disease panels (PCR and/or serology). In dogs, one may screen for the following infectious agents: *Ehrlichia* spp., *Borrelia burgdorferi*, Rock Mountain Spotted Fever, *Bartonella* spp. (culture and PCR), and *Leptospira* spp. in cases of hepatic or renal involvement. In cats, one should evaluate for FeLV, FIV, feline infectious peritonitis (FIP) virus, toxoplasmosis, *Hemoplasma* spp. (*Mycoplasma*), and *Bartonella* spp. (culture and PCR). Testing for *Ehrlichia* spp., *Rickettsia* spp., and *Anaplasma phagocytophilum* can also be considered. A fungal assay is indicated if the patient lives in or has had exposure to a region with a higher incidence of fungal disease. Other infectious disease tests may be performed depending on the geographical location of the pet. Screening for *Brucella* should be done in breeding dogs. Immune-mediated disease screening can include a Coomb's test, a slide agglutination test (if the patient is anemic), and an antinuclear antibody (ANA) test. Immune disease is often a diagnosis of exclusion.

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Imaging should include thoracic radiographs, abdominal ultrasound, and/or abdominal radiographs. Ultrasound can be very useful for assessing evidence of cholangiohepatitis, pyelonephritis, chronic urinary tract infection, abscess formation, peritonitis, and neoplasia; it also permits an examination of the intra-abdominal lymph nodes. An echocardiogram can offer assessment for vegetative endocarditis, whereas spinal radiographs offer assessment for discospondylitis. In cases where all other testing has proven negative and the patient has not responded to broad-spectrum antibiotics and supportive care, arthrocentesis should be considered to evaluate for septic joint disease, immune-mediated polyarthritis, and infectious disease. Finally, one can consider assessing the cerebrospinal fluid for meningoencephalitis, GME, and meningitis/arteritis. A bone marrow exam should be performed if blood dyscrasias are noted on the CBC.

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Treatment: Treatment of the fever depends entirely on the underlying cause. Ideally, a thorough diagnostic plan will yield a diagnosis that will guide the appropriate therapeutic course. However, if an exhaustive approach has not produced a definitive diagnosis and there is no response to broad-spectrum antibiotics, trial therapy with immunosuppressive agents such as prednisolone can be considered to treat presumed immune-mediated diseases. Given the potential for negative sequelae should an underlying infection be present, one must be certain that the investigation is thorough and monitor the patient's response carefully.

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Conclusion: If a documented fever has not responded to antibiotics, antipyretics, or general nursing care, it is important to obtain a diagnosis to guide more specific treatment. A systematic physical examination and thorough history-taking will help inform further diagnostics in addition to what is revealed by the minimum database.

References:

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Flood J. The diagnostic approach to fever of unknown origin in dogs. *Compend Contin Educ Vet* 2009;31(1):14-21.

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Male

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Lunn KF. Fever of unknown origin: a systematic approach to diagnosis. *Compend Contin Educ Vet* 2001;23(11):976-92.

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Acute Renal Failure

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

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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



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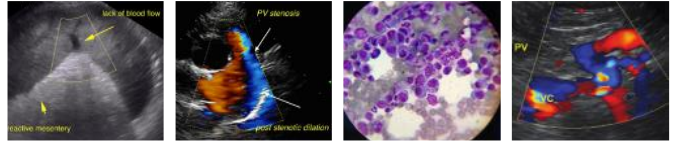
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and the progression to overt renal failure because it is during this period that renal cell damage develops. The challenge, however, is that the initiating phase may only become evident in retrospect as it generally lacks characteristic signs. The 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 tract, operative sites, and the urinary tract. Septicemia may also occur and is sometimes the result of intravenous and urinary indwelling catheters.

Diagnostics: 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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