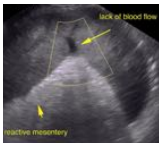

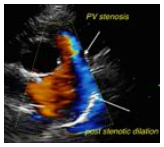

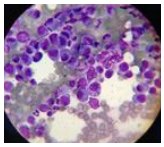
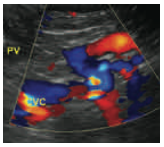

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1-800-838-4268 info@sonopath.com

PATIENT

Nugget Lehey

SPECIES

Canine

BREED

Mix

SEX

Spayed Female

AGE

13 years

INTERPRETED BY

Eric Lindquist, DMV DABVP, Cert. IVUSS

IMAGING PERFORMED BY

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Allendale

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94785

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12/21/21

PRESENTING CLINICAL SIGNS

Elevated ALKP 455 was 254 on 10/15/21

History of PLN.

Creatinine was 2.1 and is now 2.5

UP was 1.6 and is now 3.6

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** presented subjectively moderate degenerative changes with increased cortical echogenicity. Non-obstructive, minor dystrophic mineralization was noted. Subjectively the kidneys were near end stage and approximately 50-60% compromised. The left kidney measured 5.91 cm. The right kidney measured 6.62 cm.

Adrenal Glands

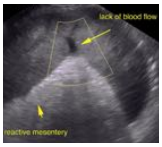
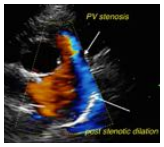
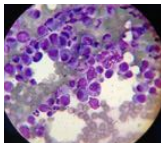
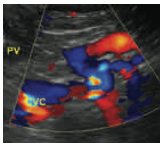
Both **adrenal glands** were visualized and recognized as having normal shape, size, position and echogenicity for this breed. The phrenic vasculature, glandular echogenicity and detail were unremarkable. Capsule, cortex, and medullary definition were normal for this age patient. The left adrenal gland measured 2.65 x 0.57 cm. The right adrenal gland measured 3.12 x 1.44 cm at the cranial pole and 0.89 cm at the caudal pole.

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 was noted.

Liver

The **liver** images from right and left intercostal as well as subcostal views revealed subjectively normal liver size, contour, and structure. Some age-related parenchymal remodeling was noted but likely not clinically significant at this time. Vascular and biliary tracts were of normal volume and no evidence of congestion was noted. The gallbladder presented some dependent debris with essentially normal contour. The cystic and common bile ducts were normal. No overt evidence of active inflammatory,

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infiltrative or regenerative pathology was noted but should be paired with current or past LE elevations regarding any clinical significance to this presentation. The hepatic lymph nodes were unremarkable.

Gastrointestinal

Examination of the **gastrointestinal tract** revealed a stomach and intestine free of stasis, of normal wall thickness, acceptable curvilinear mural detail, and peristaltic activity. Small and large intestine demonstrated normal luminal chyme and stool consistency respectively. No obstructive or overt infiltrative disease was noted. No associated abnormal lymphatic activity was noted.

Pancreas

The base and limbs of the **pancreas** were observed to be largely isoechoic to surrounding omental fat. Some parenchymal remodeling, however, with mild deviation from curvilinear normalcy was observed. Pancreatic duct and capsular irregularities were present consistent with age related changes. If pain upon imaging (+ Murphy sign) was present or if the patient is focally painful in subxiphoid palpation then low-grade smoldering chronic pancreatitis should be suspected.

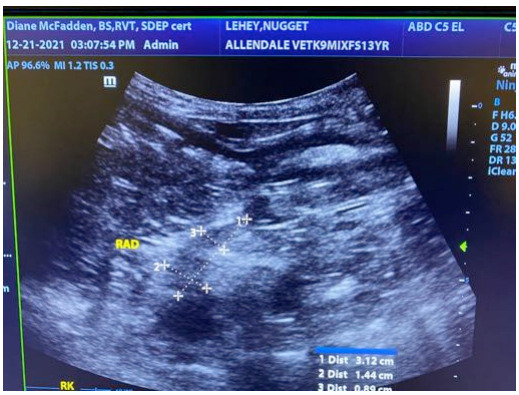
ULTRASONOGRAPHIC FINDINGS

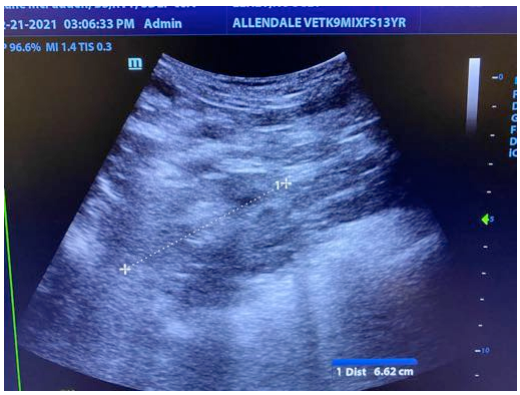
Chronic renal disease, non-specific with secondary mineralization.

Structurally normal adrenal glands and geriatric abdominal changes.

INTERPRETATION OF THE FINDINGS & FURTHER RECOMMENDATIONS

Treatment is recommended to support chronic renal failure. Blood pressure measurements +/- urine culture and sensitivity is recommended if any inflammatory sediment develops in the urine. Ace inhibitor therapy and nutraceuticals are indicated.





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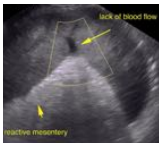
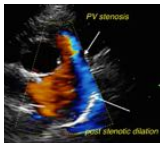
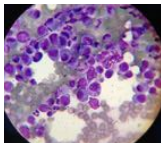
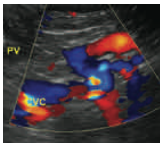

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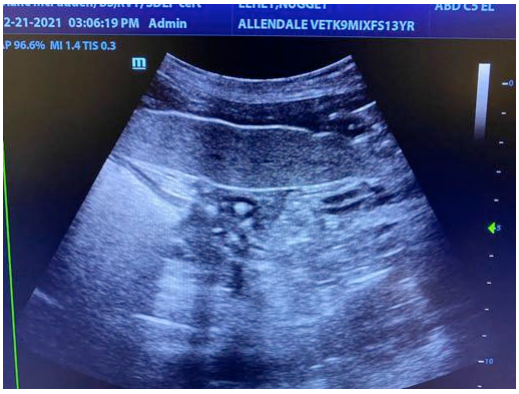

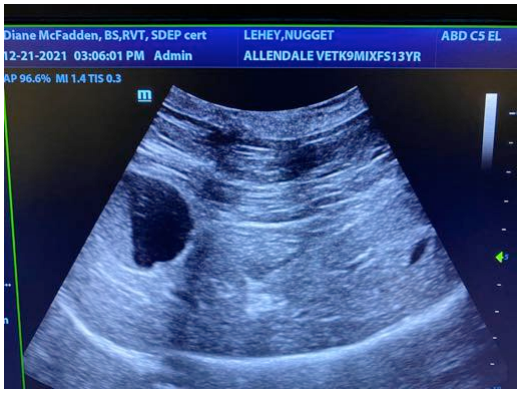

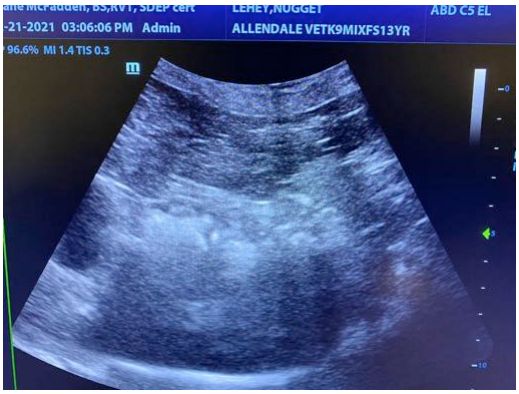

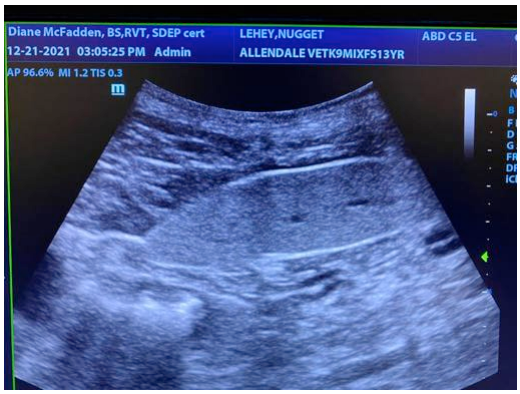

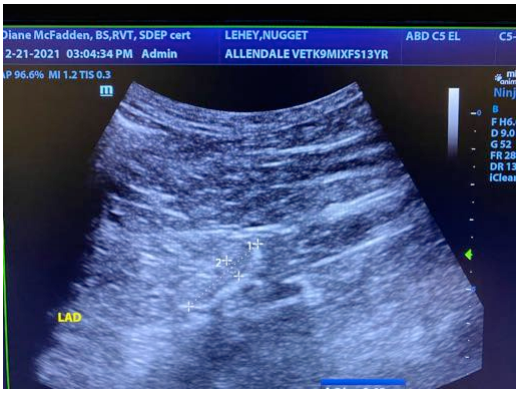

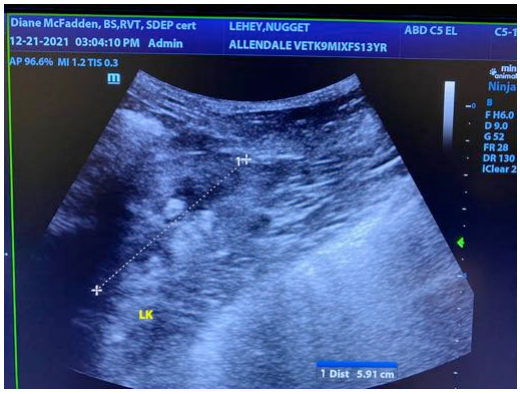

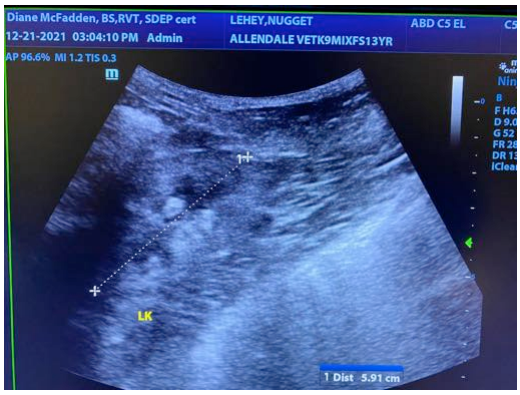

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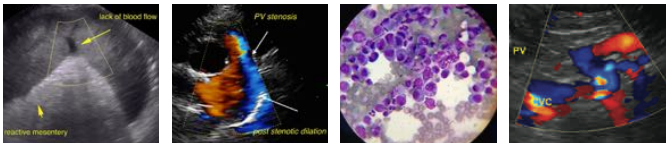

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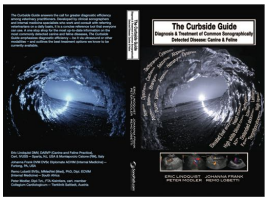
The information and recommendations provided are based on the images presented by the referring veterinarian. No evaluation can be communicated regarding pathology that was not visible in the image/video clips provided.

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



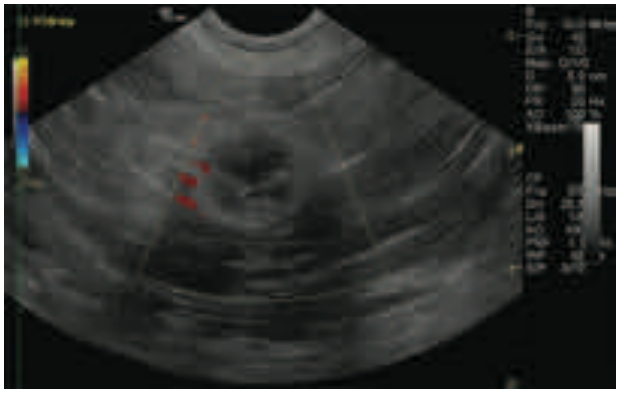
The following is an applicable excerpt from the *Curbside Guide to Diagnosis & Treatment of Sonographic Disease* offered by SonoPath.com Lindquist, Frank, Lobetti, and Modler.

An essential quick guide for every general practitioner and sonographer.

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

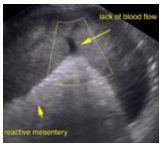
Chronic Kidney Disease

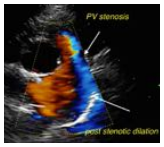
http://www.sonopath.com/CKD

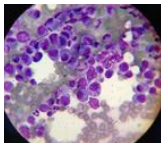
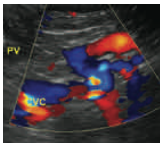


Long axis of the left kidney in a cat with chronic nephritis and end stage kidney disease. Note the significant decrease in size, irregular shape and loss of echoarchitecture. The renal cortex is hyperechoic, the corticomedullary definition is reduced.

Description: Chronic kidney disease (CKD) is generally considered a progressive and irreversible disease. It is estimated that 1 in 3 elderly cats and 1 in 5 elderly dogs in the USA have CKD. Although CKD may progress as the result of an unresolved underlying cause, it appears that there is a point at which the process becomes self-perpetuating. CKD can be congenital, familial, or acquired. Many animals develop CKD after incomplete recovery from an episode of acute kidney disease for which the cause of the initial insult was known. Although most of the primary initiating causes of CKD are well defined, it is often the case that owners have no knowledge of the primary insult that has caused their pet's condition. Current hypotheses suggest that an initiating insult inaugurates the process of CKD, but that nephron loss continues long after the original insult has disappeared. Animals do not show clinical





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signs associated with CKD until the glomerular filtration rate (GFR) is reduced to 25-30% of normal. Thus, the disease only becomes apparent after a prolonged period and after continued loss of a vast amount of functional renal tissue. During the early, clinically silent phase of CKD, the kidneys undergo a series of functional and anatomical adaptations. The histological appearance of the renal tissue sometimes indicates glomerular, tubular, or interstitial changes; however, this does not usually permit identification of a primary cause. In cases of advanced CKD, regardless of the primary initiating cause or disease process, there are no differentiating histological appearances as the kidney only responds to injury and reduced functional renal mass with stereotypical structural and functional changes.

Classification: The International Renal Interest Society (IRIS) has proposed a classification system for CKD that facilitates a staged approach to the disease in dogs and cats. The classification scheme is based on the use of serum creatinine to estimate the degree of decline of GFR caused by kidney disease. Moreover, it recognizes that the degree of azotemia in cats and dogs is not always the same. The classification system for CKD can be divided into 4 stages:

Stage I: Non-azotemic CKD (Creatinine < 1.4 mg/dl dogs, < 1.6 mg/dl cats)
In the initial stage of CKD, the animal is not azotemic, but is isothenuric and generally has no observable clinical signs.

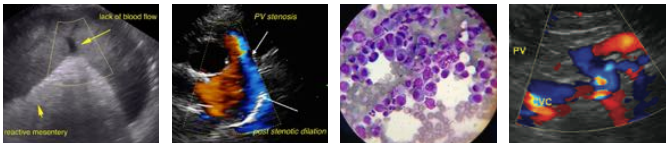
Stage II: Mild renal azotemia (Creatinine 1.4-2.0 mg/dl dogs, 1.6-2.8 mg/dl cats)
This stage of CKD occurs when there is sufficient loss of renal tissue such that azotemia is present; however, there are usually no clinical signs. During this stage, it is important to slow the progression of the disease. Generally, the rate of progression in cats is slow (months to years), but erratic and more rapid in dogs (weeks to months).

Stage III: Moderate renal azotemia (Creatinine 2.1-5.0 mg/dl dogs, 2.9-5.0 mg/dl cats)
This stage reflects the effects of further declines in GFR and the increased likelihood of clinical signs associated with advanced CKD. Both progression and uremia are concerns at this stage.

Stage IV: Severe renal azotemia (Creatinine ≥ 5.0 mg/dl dogs or cats)
Animals in this stage have uremic syndrome. Identifying and alleviating complications is critical at this stage.

Diagnostics: Arriving at a diagnosis of CKD is relatively straightforward; it is based upon findings of elevated BUN and creatinine in the face of isothenuric urine. The diagnosis of early renal disease, in the absence of azotemia, can be more challenging. Renal function can be assessed using the gold standard technique—nuclear scintigraphy to assess the GFR; however, the iohexal clearance test is in fact more readily available and can be administered directly in a clinical setting. This test is especially useful in the workup of polyuria and polydipsia (PU/PD), in the absence of azotemia, to differentiate early renal disease from other possible causes of PU/PD. Iohexal is administered (300 mg/kg IV) as a slow bolus over 2 minutes. Premedication with diphenhydramine (1 mg/kg IM) is advised due to the infrequent but possible occurrence of an anaphylactoid reaction. Serum samples are collected at 2, 3, and 4 hours, and must be collected at exact time points; the time of collection is noted on the tubes. The serum is then frozen and submitted for GFR analysis.

Additional diagnostic tests include a systemic blood pressure test to assess for concurrent systemic hypertension, a urine culture and sensitivity, and a urine protein-creatinine ratio using a urine sample that contains inactive sediment.



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Treatment:

1. Diet: Dietary therapy is probably the most commonly prescribed therapy for animals with CKD. Yet, because renal patients frequently have selective appetites, clinicians are often faced with the challenging decision of whether to recommend switching the animal to a renal diet or continuing the current diet with the view that eating any food is better than risking reduced food intake by attempting a potentially unwanted diet change. When animals with stage III renal disease were fed a therapeutic renal diet, they showed a better quality of life and lived substantially longer. In one study, dogs on a therapeutic renal diet remained free of uremic signs almost two-and-a-half times longer and had a median survival time over three times longer than dogs on a normal maintenance diet. Renal-related death was the primary cause for the higher rate of premature mortality among dogs fed the normal maintenance diet. It appeared that longer survival times were due to the fact that renal function declined more slowly in dogs on the therapeutic renal diet.

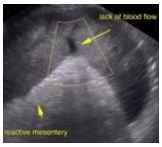
2. Dietary phosphorus restriction and phosphate-binding agents: Phosphorus is retained in CKD, which eventually results in hyperphosphatemia and in turn promotes renal secondary hyperparathyroidism. In many species, both phosphate retention and hyperparathyroidism are the major causes of the progression of kidney disease. In a model of induced CKD in dogs, dietary phosphate restriction, when combined with protein restriction, was shown to slow the progression of kidney disease and improve survival. There have been no clinical or experimental studies establishing the value of adding phosphate-binding agents to dietary phosphate restriction in dogs.

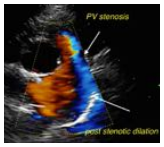
3. Calcitriol: The kidneys are responsible for converting 25-hydroxycholecalciferol to its most active metabolite, 1,25-dihydroxycholecalciferol, or calcitriol. Because CKD impairs calcitriol production, calcitriol deficiency may be one factor that contributes to secondary hyperparathyroidism. Calcitriol supplementation has been advocated as a means of normalizing hyperparathyroidism. In a randomized, controlled clinical trial that examined the effects of calcitriol therapy on CKD, calcitriol was effective in reducing renal mortality, but did not appear to influence appetite, activity, or quality of life. One potential adverse effect of calcitriol supplementation is the development hypercalcemia. Although hypercalcemia reportedly occurs in 30-57% of humans treated with calcitriol, hypercalcemia appears to be an uncommon side effect in dogs.

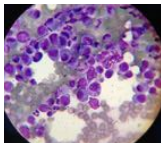
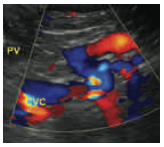
4. Anti-hypertensive therapy: Hypertension is a well-recognized complication of CKD. The most profound clinical effect of hypertension seems to be hypertensive retinopathy accompanied by retinal detachment, hemorrhage, and blindness. Hypertension-related CNS disorders, such as seizures and loss of balance, have also been observed. Systemic hypertension is a risk factor for rapid progression of renal failure and decreased survival time in animals with spontaneous CKD; it is also associated with a greater magnitude of proteinuria. Systolic blood pressures greater than 160 mm Hg in dogs and 180 mm Hg in cats are considered hypertensive. Currently, angiotensin-converting enzyme (ACE) inhibitors (e.g. enalapril, benazepril) appear to be the drugs of choice for managing hypertension in dogs, whereas amlodipine (a calcium channel antagonist) is preferred for managing hypertension in cats. ACE inhibitors and beta blockers do not appear as effective in lowering feline blood pressure.

5. ACE inhibitor therapy: ACE inhibitors appear to be of value in limiting the progression of kidney disease in humans suffering from proteinuric renal diseases. Studies have shown that dogs with glomerulonephritis that were treated with enalapril displayed reduced proteinuria; the disease progression was also stabilized. Although ACE inhibitors are commonly prescribed for humans with CKD, it is less apparent whether they should be recommended for non-proteinuric animals. ACE inhibitors reduce intraglomerular blood pressure, which has been shown to reduce the progression of CKD.

6. Metabolic acidosis: The normal response of the kidney to an acid load is to excrete strongly acidic, bicarbonate-free urine. In normal animals, the total capacity of renal tubular cells to excrete hydrogen ions is only partially utilized, which suggests a secretory acid excretion reserve exists; however, in





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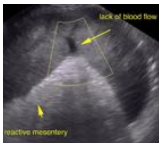
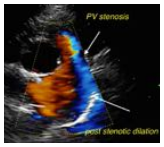
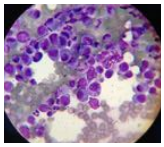
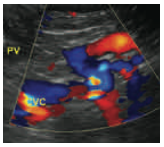
animals with CKD, this reserve is saturated and can result in systemic metabolic acidosis. Although metabolic acidosis has the potential to cause detrimental effects in animals with CKD and is very easy to treat, it is often overlooked. Progressive loss of lean body mass and bone disease is not uncommon in animals with CKD. Although the pathophysiology of these changes is complex, chronic metabolic acidosis plays a pivotal role in the pathophysiology of protein catabolism and renal osteodystrophy. Acidosis can result from a failure to reabsorb filtered bicarbonate, excrete the daily load of acid, or acidify the urine. The acidification process is an integrated function that occurs at several nephron segments. A large portion of excreted acid is carried by ammonia, which is synthesized by the kidney. Protein catabolism provides a source of glutamine, which is a substance for renal ammoniagenesis. As renal disease progresses, the amount of ammonia produced per nephron is increased; however, because the number of surviving nephrons is decreased, the total amount of ammonia produced is reduced. This decreases the net amount of excreted acid. The progressive decrease in renal ammoniagenesis in CKD is one of the main causes of metabolic acidosis. Metabolic acidosis can be easily corrected by administering either sodium bicarbonate or potassium citrate. In cats with CKD, it may be preferable to use potassium citrate because of the apparent association between metabolic acidosis and negative potassium balances; however, it is important to monitor serum potassium levels as well as serum bicarbonate or total carbon dioxide concentrations.

7. Uremic gastritis: This condition is characterized by glandular atrophy, edema of the lamina propria, mast cell infiltration, fibroplasia, mineralization, and submucosal arteritis. Clinically, as the severity of azotemia worsens, uremic signs of vomiting, nausea, and anorexia develop. Although some of these signs may be the result of uremic toxins on the medullary emetic chemoreceptor trigger zone, uremic gastritis may also contribute to these problems.

Cats with CKD have been shown to have an increase in serum gastrin concentrations, which contributes to the pathogenesis of uremic gastritis. Although vomiting is a frequent yet inconsistent finding in uremic dogs, many cats with uremic gastritis may show only partial to complete anorexia instead of vomiting. Besides anorexia and vomiting, uremic gastritis may also result in gastrointestinal bleeding. Unfortunately, uremic gastritis is often not addressed until a dog is vomiting or a cat is anorexic. It is recommended that practitioners be more proactive in treating this problem to lessen the likelihood of animals developing clinical signs. One general recommendation is to give animals with stage III CKD an H_2 receptor antagonist (e.g. cimetidine, ranitidine, or famotidine) prior to the onset of any clinical signs.

8. Anemia: Anemia occurs in many animals with CKD. The severity and progression of anemia often correlates with the degree of CKD; however, concurrent diseases and iatrogenic blood loss associated with repeated sampling of blood for diagnostic tests and monitoring might exacerbate the anemia. Anemia associated with CKD is usually non-regenerative and normocytic normochromic. Some animals with CKD may have concurrent iron deficiency, resulting in a microcytic hypochromic anemia. Although the principal cause of anemia in most animals with CKD is an erythropoietin deficiency, the following may also be contributing factors: nutritional deficiencies; erythropoietic inhibitor substances in uremic plasma; shortened red blood cell life span; myelofibrosis; and blood loss via gastric ulceration secondary to uremic gastritis. Erythropoietin deficiency in CKD results from an insufficient capacity for new hormone synthesis in response to hypoxia due to decreased renal mass. Erythropoietin is a hormone secreted on demand by the kidneys in response to intrarenal tissue hypoxia resulting from either a decreased oxygen-carrying capacity associated with anemia or decreased oxygen content. It enhances erythropoiesis by stimulating the formation of proerythroblasts and promotes hemoglobin synthesis and the release of reticulocytes from bone marrow into the circulation.

An important but often overlooked consideration for minimizing anemia is iatrogenic blood loss. This is particularly a problem in hospitalized cats and small dogs when repeated samples of blood are taken from already anemic animals for diagnostic tests and monitoring. Clinicians often attribute anemia to other causes and fail to consider the impact repeated blood sampling can have on small, anemic cats and dogs. Another important consideration for anemia in animals with CKD is gastrointestinal blood loss. If gastrointestinal blood loss is suspected, a trial course of an H_2-receptor antagonist, omeprazole, or sucralfate should be considered. Improvements in hematocrit would indicate a positive response to therapy. Nutritional causes, such as iron deficiency and loss of B vitamins, should also be addressed.

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9. Erythropoietin therapy: Erythropoietin therapy is the treatment of choice for non-life threatening anemia in dogs and cats with CKD when hematocrit values fall below 20% and clinical signs are attributable to the anemia. Although recombinant canine and feline erythropoietin have been developed, the only readily commercially available form is recombinant human erythropoietin (e.g. Eprex®, Epogen®, Recormon®).

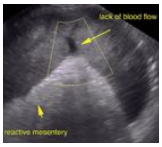
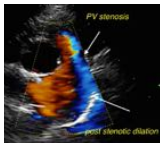
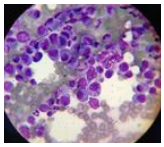
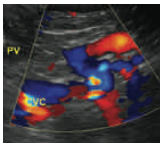
The initial dosage of erythropoietin is 50-100 units/kg subcutaneously three times a week, with a target hematocrit in dogs of 37-45% and 30-40% in cats. The hematocrit should be monitored weekly until the lower end of the target range is reached. At that time, the dosage interval of erythropoietin can be decreased to once or twice a week. The hematocrit should be monitored weekly until it has stabilized in the target range for at least 4 weeks. At this time, the hematocrit can be monitored monthly as long as it remains stable.

Generally, the target hematocrit is attained within the first 8-12 weeks of starting erythropoietin therapy. A poor response to initial therapy should prompt an evaluation for an underlying cause, such as iron deficiency, gastro-intestinal blood loss, and concurrent infectious, inflammatory, or neoplastic disease. Furthermore, if an animal suddenly becomes unresponsive to erythropoietin therapy, the possibility of anti-erythropoietin antibody formation, in addition to the above causes, should be considered.

Iron supplementation is recommended for all animals receiving erythropoietin therapy, even if the animal does not have an iron deficiency. The demand for iron associated with stimulated erythropoiesis is high, and even animals without preexisting iron deficiencies may be unable to keep up with the demand for iron.
The most commonly recognized side effect of erythropoietin administration in animals is the development of refractory anemia associated with the formation of neutralizing anti-erythropoietin antibodies. Antibody formation is estimated to occur in approximately 50% of dogs and cats that receive erythropoietin therapy. If antibodies are going to form, they tend to occur within 1-6 months after the therapeutic regime has been commenced. Discontinuing erythropoietin therapy appears to result in the cessation of antibody formation. The rate of recovery from the resulting anemia will depend on the antibody titer and its clearance from the body, as well as the ability of the animal to secrete endogenous erythropoietin. The relatively high prevalence of anti-erythropoietin antibody production raises the question of when to initiate erythropoietin therapy in animals with CKD. If therapy for anemia is started prior to the animal showing any clinical signs associated with anemia, anti-erythropoietin antibodies may develop, depriving the animal of the clinical benefits of this therapy. Most animals will not show significant clinical signs of chronic anemia until the hematocrit falls below 20%.

Erythropoietin therapy should be discontinued if any one of the following develops: anti-erythropoietin antibody formation; systemic hypertension unresponsive to antihypertensive therapy; polycythemia; or any local or systemic hypersensitivity reactions.

Note: Darbopoeitin, a recombinant analog of human erythropoietin, is an alternative to erythropoietin. It is reported to have less of an effect on antibody formation in cats, thereby lessening the occurrence of pure red cell aplasia.

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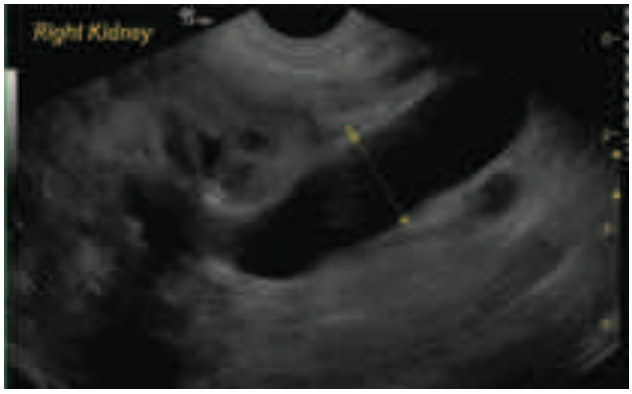
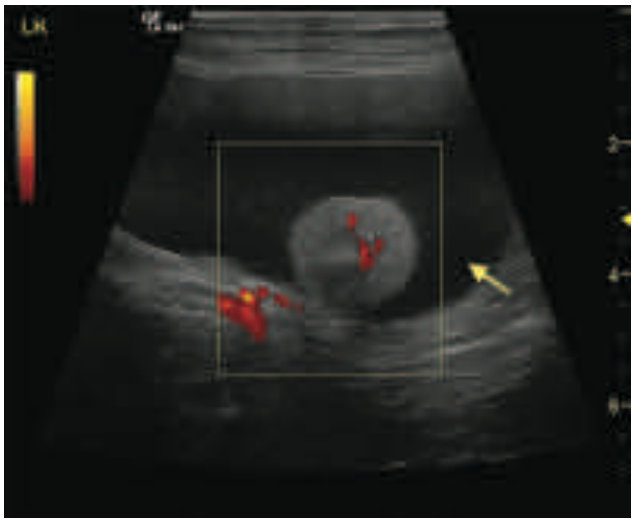
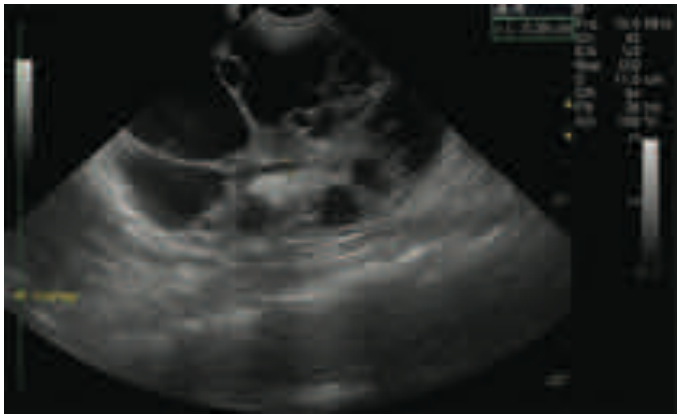


Long axis of the right kidney in a dog with chronic renal failure and hydroureter. Note the small size, increased cortical echogenicity and surface irregularity of the kidney. Also note the moderate dilation of the renal pelvis and the generalized dilation of the amotile ureter seen as a tubular structure with anechoic content (between calipers).



Long axis of the left kidney in a cat with chronic nephritis and perirenal pseudocyst formation. The kidney is small, hypovascular and shows severe echoarchitectural changes with increase in echogenicity. The pseudocyst is seen as a large fluid filled cavity surrounding the kidney within the retroperitoneal space (arrow).



Long axis of the right kidney in a cat with polycystic kidney disease. The renal parenchyma has been replaced by multiple cysts of varying size almost entirely. Note the mild pyelectasia (between calipers).

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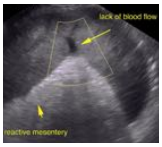
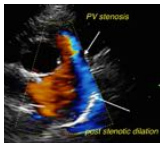
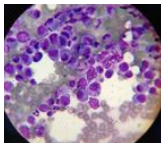
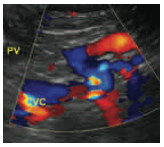
Brown S: Evaluation of chronic renal disease: a staged approach. *Compend Contin Educ Pract Vet* 1999;21:752-63.

Chalhoub S, Langston C, Eatroff A. Anemia of renal disease: what it is, what to do and what's new.

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