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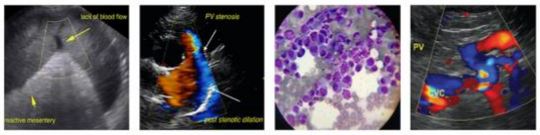

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PATIENT

Juneau Sullivan

SPECIES

Canine

BREED

AK Klee Kai

SEX

Neutered Male

AGE

13 years

WEIGHT

16.6 Pounds

INTERPRETED BY

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

IMAGING PERFORMED BY

Rebekah Jakum, CVT ARDMS/RVT

HOSPITAL NAME

Littlestown VH

REFERRING VET

Dr. Jennings

INVOICE

13198

DATE

9.20.2021

PRESENTING CLINICAL SIGNS

History: Chronic intermittent pancreatitis with diarrhea and vomiting

Medication: RC Selected Protein, Hill's i/d LF, Metronidazole, Cerenia, Apoquel

Labs: Canine chronic enteropathy/IBD panel- all values elevated. Precision PSL 741, WBC 18.6 k with neutrophilia, Albumin level 3.8

ULTRASONOGRAPHIC EXAMINATION OF THE ABDOMEN

Urinary System

The urinary bladder, trigone, cystourethral junction, and visible pelvic urethra to a depth of 4.0 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. Aortic trifurcation was normal.

The residual prostate was symmetrically normal in size with uniform parenchyma and slight coarse echotexture measuring 0.77 cm in width.

Normal size and margination were present in the kidneys. A normal 1:3 cortex / medulla ratio was maintained. The medulla and cortices were uniform in texture with some increased echogenicity and loss of corticomedullary symmetry and definition expected for the age of the patient. No evidence of pelvic dilation was present. Pinpoint medullary mineralization and small cortical cysts noted in both kidneys. The left kidney measured 3.8 cm in length. The right kidney measured 3.3 cm in length.

Adrenal Glands

The left adrenal gland was uniform in size and contour with a uniformly hypoechoic parenchyma. The left adrenal gland measured 1.7 cm length x 0.58 cm width at the caudal pole. The right adrenal gland was uniform in size and contour with a uniformly hypoechoic parenchyma. The right adrenal gland measured 1.7 cm length x 0.56 cm width at the caudal pole.

Spleen

The spleen exhibited primarily finely textured and homogenous parenchyma which was hyperechoic to the liver and renal cortical parenchyma. Multifocal, well-defined, symmetrical, echogenic nodules were present throughout the cranial to caudal 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 or neoplastic changes were not noted. The echogenic nodules tend to trend benign and are most consistent with benign hyperplasia or myelolipomas.

Liver

The liver was mildly enlarged in size, structure, and contour. The liver parenchyma was mildly nonuniform and hypoechoic to the spleen with a moderate coarse echotexture and subjective mild to benign parenchymal remodeling. The hepatic and portal vasculature were normal in appearance without signs of congestion. The gallbladder was non distended in size with mild inspissated uniformly echogenic gallbladder debris. The cystic duct and common bile ducts were normal without evidence of dilation.

Gastrointestinal

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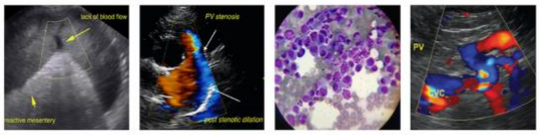

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The visualized gastric walls were sonographically unremarkable with intact wall layering and without evidence of mural pathology. The stomach was mildly gas distended. The gastric body wall measured 0.34 cm.

The small intestine presented intact wall layering with subjective propensity for generalized prominent mucosa layer. The jejunum wall measured 0.42 cm.

The colon was normal with subjective semi-formed to soft feces present.

Pancreas

The pancreas was normal in size and contour with isoechoic to heterogeneous parenchyma compared to adjacent omentum. No signs of active inflammation or neoplasia.

Free Abdomen

No overt lymphadenopathy or peritoneal effusion was present.

ULTRASONOGRAPHIC FINDINGS

Primary Findings

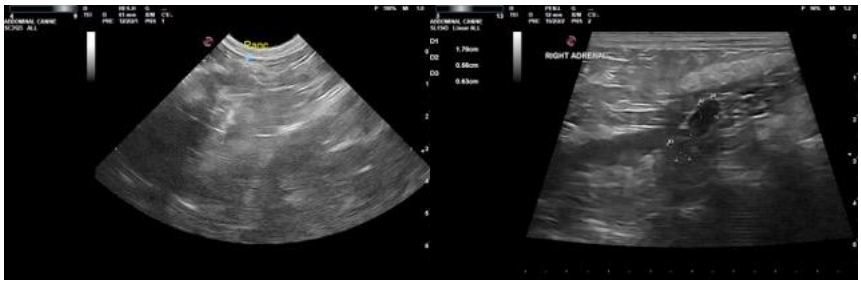
- Mild chronic renal changes with pinpoint medullary mineral and cortical cysts
- Benign splenic nodules – consistent with probable myelolipomas
- Intact small bowel wall layering with propensity for prominent generalized mucosa – consistent with chronic enteropathy, likely IBD
- Sonographically unremarkable colon with subjective semi formed to soft feces
- Heterogeneous pancreas – age-related or patient variant. Minor parenchymal remodeling owing to previous inflammation or low grade to chronic pancreatitis which may present sonographically unremarkable possible

Secondary Findings

- Mild inspissated gallbladder debris (non-mucocele), likely incidental

INTERPRETATION OF THE FINDINGS & FURTHER RECOMMENDATIONS

Endoscopic intestinal biopsies likely ideal for further clarification and definitive diagnosis. Empirically, continued presumptive IBD therapy would be appropriate.



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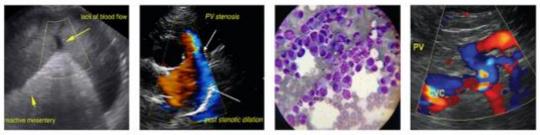

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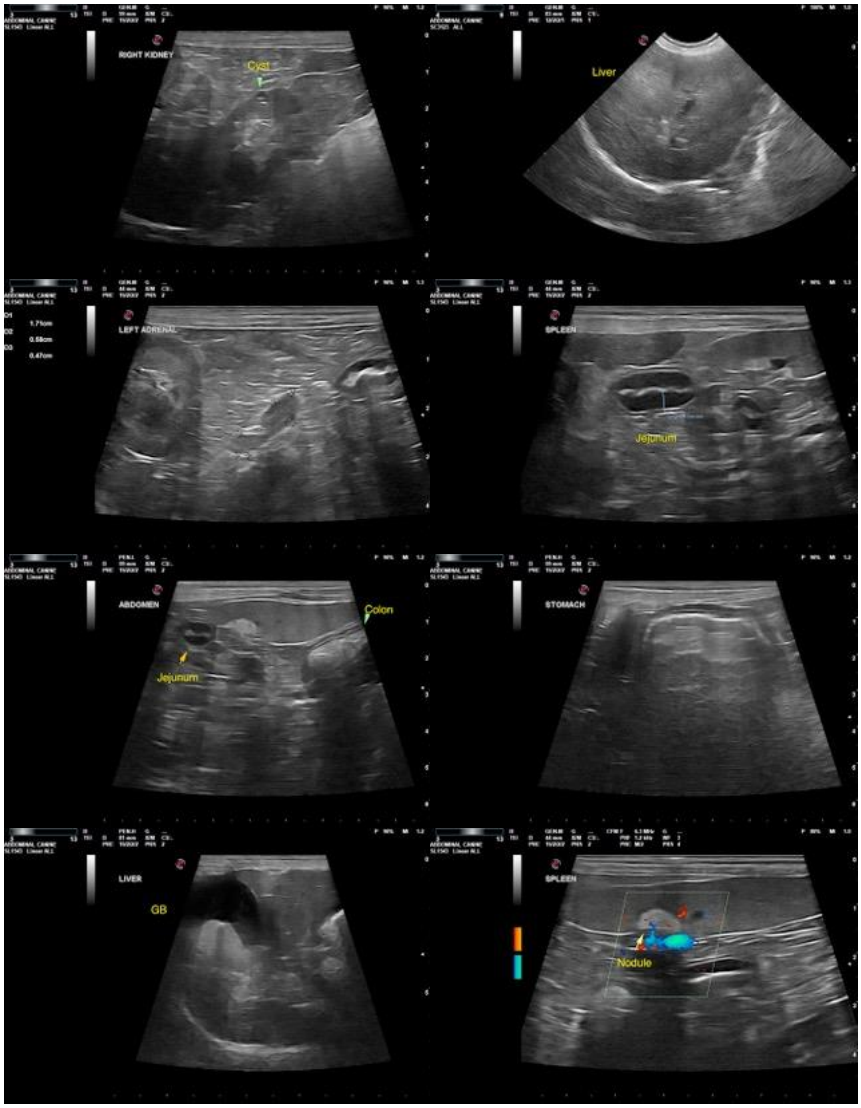

The information and recommendations provided are based on the images presented by the referring veterinarian. No evaluation can be communicated regarding pathology that was not visible in the image/video clips provided.

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.

R. McKenzie Daniel, DVM, DABVP (Canine/Feline Practice)
mac.daniel@sonopath.com

Canine Inflammatory Bowel Disease (IBD)

http://www.sonopath.com/K9IBD

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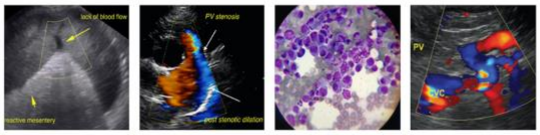

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Description: Inflammatory bowel disease (IBD) occurs when bowel inflammation results either from an aberrant immune response or an appropriate immune response to a normal luminal resident pathogen. It is thought that the immune response, once initiated, becomes self-perpetuating. Currently, this disease is classified as idiopathic. Antibiotic responsive diarrhea (ARD) and food responsive diarrhea (FRD) are similar yet separately classified enteropathies.

Clinical Signs: The most common form of canine IBD is lymphoplasmacytic enteritis (LPE); it is typically seen in middle-aged to older dogs. The most common clinical signs include diarrhea, vomiting, anorexia, and weight loss. Diarrhea can be identified as originating from the small intestine (indicated by weight loss and large volume watery stools), large bowel (indicated by tenesmus, hematochezia, increased frequency of small volumes of diarrhea, and mucoid feces), or both.

Diagnostics: In order to differentiate IBD from underlying lymphoma or other conditions, it is important to acquire tissue samples. Tissue samples enable the practitioner to classify the cell type, grade the degree of cellular infiltration, evaluate for the presence of fibrosis, and ultimately gauge how aggressive the therapy must be in order to adequately treat the disease process and prevent irreversible damage. At minimum, endoscopic biopsies of the duodenum and ileum are recommended; jejunal biopsies may also be necessary. Biopsy samples must contain full villus structures (not just the friable tips of villi), ideally down to the level of the muscularis. Presentations of IBD associated with only the ileum are becoming increasingly common. Although the ileum can be a difficult area to access endoscopically, it can be reached via the colon. If indicated, we therefore suggest that an experienced endoscopist be used to obtain ileal biopsies to improve the chances of acquiring accurate results. In general, 6-8 samples obtained from various regions by an experienced practitioner should suffice; however, it is important to remember that the disease may not be diffuse, and that abnormalities may be found only in a portion of the samples. Six to seven adequate samples or 10-15 marginal samples are needed for dogs, whereas fewer samples are typically required to obtain a diagnosis for cats. Crypt lesions within the duodenum are more difficult to diagnose and may require a larger number of samples (13 adequate or 28 marginal). Thus, it is critical that the pathologist has an appropriate number of samples at his or her disposal.

Grading of IBD is based on architectural disruption of the invaded tissues. The World Small Animal Veterinary Association (WSAVA) International Gastrointestinal (GI) Standardization Group developed specific criteria to diagnose and treat canine IBD, which include guidelines on obtaining and interpreting endoscopic samples. Mild forms lack mucosal or glandular disruption, and there is no fibrosis of the lamina propria. Severe IBD presents as architectural disruption with ulceration, necrosis, villous atrophy, glandular loss/hypoplasia, and fibrosis of the lamina propria; however, clinical signs do not always correlate with the severity of the histological changes.

Ultrasonographic examination of the bowel wall imparts some correlative information for delineating the differences among various intestinal pathologies. Mucosal appearance is key in developing a working diagnosis. To date, there are four primary presentations that can be correlated with particular ultrasonographic appearances; however, biopsies are necessary to confirm the diagnoses:

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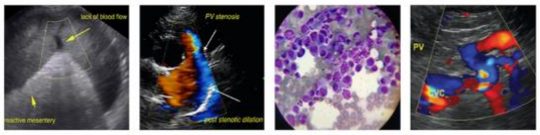

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1. Food intolerance typically presents as a prominent hypoechoic mucosa. Patients experiencing this particular change uniquely may benefit from the use of an alternative protein or hypoallergenic diet trial as a primary treatment option.

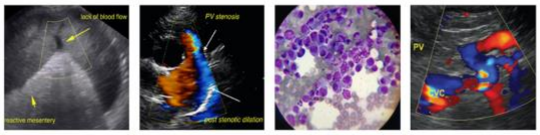
2. IBD can present as a hypoechoic mucosa with echogenic stippling visible throughout the mucosal layer. Biopsies should be performed to confirm as IBD responds best to combination therapies, including immunosuppressants. However, mucosal stippling/speckling is a non-specific finding; it is not directly associated with a finding of IBD and can be seen in dogs that do not present clinical signs of GI disease. Changes in mucosal echogenicity may also reflect a finding of IBD.

3. Patients suffering from protein-losing enteropathy (PLE), including lymphangiectasia, display prominent hyperechoic mucosal striations. Again, biopsy is indicated, and multifaceted therapy should be directed towards immune-suppression and reestablishing a positive protein balance.

4. Although the thickening of the muscularis layer and disruption of the submucosal layers are characteristic of lymphoma or other neoplasia, severe IBD may also present sonographically as such. Concurrent mesenteric lymphadenopathy is also suggestive of lymphoma. Note: Bowel tissue that appears to be normal on ultrasound does not preclude the possibility of lymphoma or IBD.

In addition to assessing ultrasound results, one must evaluate serum cobalamin and folate levels. Low or low-normal levels of cobalamin indicate severe jejunal dysfunction; cobalamin may also be low in cases of exocrine pancreatic insufficiency (EPI). With small intestinal bacterial overgrowth (SIBO), folate levels are typically high due to folate production by microbes; however, folate levels may also be low despite SIBO when severe jejunal and ileal mucosal dysfunction occurs. SIBO is not the same as antibiotic-responsive diarrhea (ARD). Definitive diagnosis depends on small intestinal culture results (via endoscopy or laparoscopy) or an assessment of serum unconjugated bile acids (SIBO case values are typically 10-20X normal). Histiocytic ulcerative colitis is most frequent in Boxer dogs, but has been reported in other breeds as well, and is largely responsive to antibiotics, such as enrofloxacin, metronidazole, and amoxicillin.

EPI is a frequent counterpart and differential for IBD. Voluminous pale-colored feces, voracious appetite, weight loss, and diarrheic feces are typical but not exclusive to EPI. Moreover, these signs are not always present given the long, compensating nature of the gastrointestinal tract. Testing is therefore essential if other causes of weight loss have been ruled out. Malabsorption and parasitism can contribute to mucosal disease and may be concurrent pathologies. Flatulence, borborygmus, and poor hair coat are evidence of a maldigestive/malabsorptive state. A low serum trypsin-like-immunoreactivity test (TLI) is diagnostic for EPI and hypocholesterolemia is typical.



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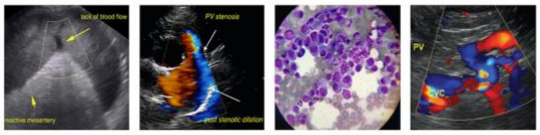
Defining the presence, via biopsy, of concurrent intestinal lymphangiectasia (IL) (dilated mucosal/submucosal lymph vessels) and PLE also helps determine the prognosis and refine treatment protocols. PLE is suspected in hypoalbuminemic patients when hepatic function is normal (based on a normal bile acid profile) and the urine protein-creatinine (UPC) ratio reveals no significant protein loss (or some secondary loss in cases of associated glomerulonephritis from GI immune complex disease). A fecal α-1 protease inhibitor test can be used to detect protein loss in feces and requires three fecal samples taken on different days. This test is most useful in cases where the patient has concurrent nephropathy and/or hepatic disease, and one is trying to determine if there is concurrent fecal protein loss and whether GI biopsies are necessary. In cases of lymphangiectasia, cholesterol levels and blood lymphocyte numbers are often decreased. Furthermore, ascites secondary to severe hypoalbuminemia (albumin less than 1.5 g/dl) can result in concurrent loss of anti-thrombin III (AT III) (50,000 Dalton MW similar to albumin). AT III deficiency can lead to a prothrombotic state, which can result in thromboembolic disease; anti-thrombotic medications may be necessary (see below). Clinically, practitioners should monitor for signs of embolic disease and monitor coagulation parameters, such as FDP, D-dimer, PT, APTT, and fibrinogen.

Eosinophilic IBD is less common and can be associated with a food allergy or parasitism. Thus, it may respond well to strict elimination diets and/or anti-parasiticides. Certain dog breeds, such as Boxers, Dobermans, German Shepherds, and Rottweilers, appear to be more predisposed to eosinophilic IBD. Severe eosinophilic enteritis can cause significant PLE in some patients. Ruling out concurrent parasitic and protozoal infection is critical in all patients, and appropriate diagnostic tests include standard fecal floatation, assessment for *Giardia* with a zinc sulfate concentration technique, fecal ELISA, or a direct florescent antibody test. A fecal PCR test can screen for various infections, such as *Campylobacter coli*, *Campylobacter jejuni*, canine distemper virus (CDV), canine enteric coronavirus (CECoV), canine parvovirus 2 (CPV-2), *Clostridium perfringens* enterotoxin A (CPEA) gene, *Cryptosporidium* spp., *Giardia* spp., and *Salmonella* spp. Colonic scrapings can be done to evaluate for evidence of fungal disease in geographically affected regions. Empirical deworming is reasonable in the management of these patients.

Treatment: The first line of therapy for IBD is dietary; a hypoallergenic food trial should be assayed for a minimum of 12 weeks. Diets that include hydrolyzed proteins diets are preferable. If a dog responds favorably, then a novel single-source protein diet can be tried using protein sources such as whitefish, salmon, venison, or kangaroo. In addition, omega-3 fatty acids can be supplemented as natural anti-inflammatory agents; however, most therapeutic diets already contain enhanced levels of omega-3 fatty acids. Supplemental, fermentable fiber sources stimulate beneficial microbial fermentation, thereby releasing volatile fatty acids for colonocyte nutrition and enhancing overall colonic health. Better colonic health has been documented to have many upstream effects, such as bolstered immunity and improved peristalsis and neuroendocrine function. In the face of PLE and lymphangiectasia, a fat-restricted diet is preferred given the loss of cholesterol and fats through the dilated intestinal lacteals. However, supplementation with medium chain triglycerides (MCT) is beneficial as these are mostly absorbed directly into the portal system, bypassing the lymphatics. Their use, in combination with a fat-restricted diet, may enhance caloric intake and management. Probiotic supplementation should also be considered to help normalize the GI flora (i.e., Purina Veterinary Diets® FortiFlora®).



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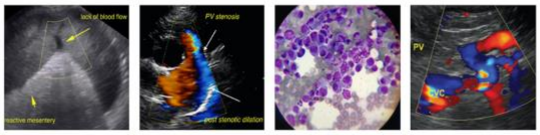
The management of SIBO includes metronidazole (10-20 mg/kg PO BID), tylosin (10-20 mg/kg PO BID), oxytetracycline (20 mg/kg PO BID-TID), or amoxicillin (10-20 mg/kg PO BID). *Helicobacter* and *Campylobacter* species are often found upon GI biopsy and may also play a role in the development of clinical signs. Their significance at this point remains unclear; however, *Helicobacter* is associated with gastric ulcerative disease in humans. Definitive diagnosis depends on gastric biopsy or the less invasive urea breath test, which is highly sensitive and specific for urease-containing bacteria. Coverage for 14-21 days with combinations of amoxicillin, metronidazole, bismuth subsalicylate, and an antacid, such as famotidine or omeprazole, are standard, but eradication rates are variable. Please refer to the chapter on "Canine Erosive Gastritis and Duodenitis" for additional information.

The pharmacological treatment of IBD should proceed as follows: for cases of colitis (with no upper GI signs), sulfasalazine (SASA) can be administered at a standard dose of 20-30 mg/kg PO TID, but can be dosed up to 50 mg/kg TID or a maximum dose of 1 gram TID in refractory patients. One suggested protocol entails administering 12.5mg/kg PO TID of SASA for 4 weeks before modifying the dosage. A gradual weaning of the effective dose by 25% every 2 weeks can be attempted once the 12-week food trial has allowed for elimination of allergen accumulation. Although not common, keratoconjunctivitis sicca (KCS) can occur as an untoward side effect of SASA. Owners should be made aware of this possibility, and patients should be monitored for any development of ocular signs. In dogs that cannot tolerate SASA, oral 5-ASA preparations, such as olsalazine (10-20 mg/kg PO TID) or mesalamine, can be utilized; however, we still have little information about the effects of these medications in dogs and KCS remains a possible negative sequelae. After signs have been in remission for 2-3 months, gradual reduction and withdrawal of immunosuppressive therapy may be possible.

Ideally, prednisone or prednisolone should only be administered once histopathological results confirm IBD and rule out lymphoma. The administration of prednisone prior to biopsy might mask the lymphoma and make it more difficult to achieve a definitive diagnosis. The use of prednisone in advance of a chemotherapeutic protocol can result in multi-drug resistance, thereby lessening the effectiveness of the chemotherapy. In clinical practice, trial therapies are commonly initiated in patients with typical clinical signs of IBD, in cases where infectious causes have been excluded, and when biopsies are unfeasible due to financial constraints or a condition such as severe hypoalbuminemia. Ultimately, if prednisone is being used empirically, then chemotherapy will not be as effective in this population of patients.

Prednisone should be administered at 2 mg/kg/day for a 2-4 week induction period. Subsequently, the patient should be weaned slowly to 1 mg/kg/day, and eventually dosed every other day. In large and giant breed dogs, dosing per body surface area is recommended to avoid overdosing and the precipitation of severe side effects; the recommended dose is 30-40mg/m^2 for large breed dogs. The concurrent administration of metronidazole, azathioprine, and SASA (in cases of concomitant colitis) may allow for a reduced dose of prednisone. Azathioprine can be dosed at 2mg/kg PO Q24hr for approximately 10 days, then 1 mg/kg PO Q24hr, and eventually every other day on alternate days to the prednisone. It should be noted that azathioprine could cause significant bone marrow suppression. Thus, it is recommended that practitioners evaluate a CBC 7 days after the onset of therapy and then on a weekly basis for the first month. Subsequently, CBCs should be performed biweekly for another 1-2 months, and then monthly. Liver enzymes should also be monitored since hepatic necrosis can occur as an idiosyncratic effect. In the



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long term, azathioprine should be administered on alternating days. Cyclosporine is an alternative immunosuppressant option; however, it can be quite expensive, especially for large breed dogs, and should be dosed at 3-5mg/kg PO Q12-24hr to start. Blood cyclosporine levels should be evaluated 7 days after initiating treatment, and then one can adjust the dosage at that point if need be. Concomitant use of ketoconazole (2.5-5 mg/kg PO BID) inhibits some metabolism of cyclosporine, leading to higher blood concentrations of the latter without increasing the overall dose or cost to the owner. Typically, the dose of cyclosporine can be cut in half when dosed in conjunction with ketoconazole.

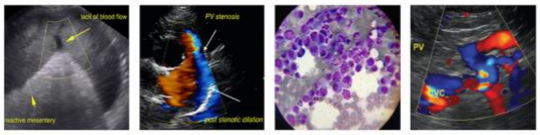
Dogs with histiocytic ulcerative colitis are managed with enrofloxacin 5 mg/kg PO Q24hr, which can also be combined with amoxicillin (20 mg/kg PO BID) and metronidazole (10-15 mg/kg PO BID).

Because dogs with severe hypoalbuminemia are predisposed to thromboembolic disease, it is important to administer anti-thrombotic medications, which include low dose aspirin (1 mg/kg PO Q24hr) or clodiprogel (Plavix) at 2 mg/kg PO Q24hr. A one-time loading dose of clodiprogel at 10 mg/kg can be given in the face of a thromboembolic episode. Hypoalbuminemic cases can be temporarily stabilized with colloid therapy, which entails administering hetastarch (5-15 ml/kg IV bolus or 20 ml/kg CRI in maintenance LRS) or, if available, fresh frozen plasma (10-20 mg/kg). The latter is not, however, usually a viable option for regulating serum albumin levels, as very large volumes of plasma are required to effectively correct albumin levels. Human albumin is a concentrated form of albumin, which can be used to improve oncotic pressure when critically necessary. Repeat exposure can result in anaphylaxis but has been used safely in many patients. Raising serum albumin levels is important, especially if surgical biopsies are to be obtained, as severe hypoalbuminemia could cause delays in the healing process and incite dehiscence. Providing oncotic support may also be necessary while instituting a more definitive treatment.

Pancreatic enzyme supplementation is indicated for cases with confirmed concurrent EPI. Low or low-normal B_{12} (cobalamin) levels should be treated as well. Recent research suggests that the current veterinary reference range for cobalamin may be inaccurate at the lower limit. That information, coupled with emerging knowledge that many patients suffer from ileal disease, which affects absorption, suggests that we may not be treating these patients adequately. Cobalamin injections (50 µg/kg SC weekly for 6 weeks, then every other week for 6 weeks, then monthly) are recommended; folate and vitamin B can also be given orally. B_{12} can be reevaluated after the initial 12-week induction schedule. It is currently recommended to continue monthly treatments in patients displaying any continued IBD signs. Moreover, some research suggests that discontinuing B_{12} supplementation may be linked to the recrudescence of signs.

Conclusion: IBD cases can be very challenging to diagnose and treat. Educating the owner on the importance of securing an accurate diagnosis and maintaining a long-term course of treatment in order to see improvements is recommended for better compliance and success with patients.

References:



PATIENT

Juneau Sullivan

SPECIES

Canine

BREED

AK Klee Kai

SEX

Neutered Male

AGE

13 years

WEIGHT

16.6 Pounds

INTERPRETED BY

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

IMAGING PERFORMED BY

Rebekah Jakum, CVT ARDMS/RVT

HOSPITAL NAME

Littlestown VH

REFERRING VET

Dr. Jennings

INVOICE

13198

DATE

9.20.2021

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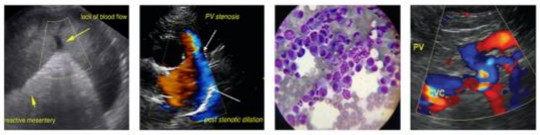

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