

What's Your Diagnosis?

Clare Reynolds, Class of 2017

Signalment: 8 mo MC DSH

Presenting Complaint: Patient was presented for a 2 week history of fever, hyporexia, and weight loss

History: Patient was adopted from a shelter, and had a history of being underweight for his age. He was initially presented to his primary care veterinarian for upper respiratory infection, extreme lethargy, and bilaterally elevated nictitating membranes. His temperature upon presentation was 104.2° F. His rDVM performed a CBC, which revealed a non-regenerative anemia with a hematocrit of 20.6%. Patient was then tested for FIV and FeLV, which he was negative for. The initial therapy plan included dexamethasone and various courses of antibiotics, but was ineffective. Four days later, patient was hospitalized at his primary care clinic for monitoring. Two days later, patient was referred due to inability to control fever.

PE Findings: Upon presentation, patient was bright, alert, and responsive, but had an unkempt hair coat. His weight was 2.24 kg and his BCS was 2/5. Heart rate was 140, respiratory rate was 36 breaths/min, temperature was 105.4° F, and patient had strong pulses. His mucous membranes were pale and tacky, and he had moderate skin tenting and sunken eyes, but had a capillary refill time of under 2 seconds; he was estimated to be 7% dehydrated based upon these findings.

Upon cardiac auscultation, a grade II/VI parasternal murmur was detected. Abdominal palpation revealed thickened loops of bowel.

An ocular exam revealed bilateral aqueous flare, keratic precipitates, rubeosis iridis, and chorioretinitis. The intraocular pressure was 6 in the right eye (OD) and 3 in the left eye (OS). Based on these findings, bilateral uveitis was diagnosed.

Diagnostic Plan:

- CBC
 - Leukocytosis with neutrophilia
 - Non-regenerative anemia
- Blood Cytology: Leukocyte concentration mildly increased consisting mainly of segmented neutrophils with moderate toxic change including increased Dohle bodies and foamy basophilic cytoplasm. Moderate anemia present with no evidence of regeneration. Chronic inflammatory leukogram present with possibly an acute component, and likely mild to moderate thrombocytopenia. No organisms seen.
- Imaging
 - Thoracic Radiographs 3-view

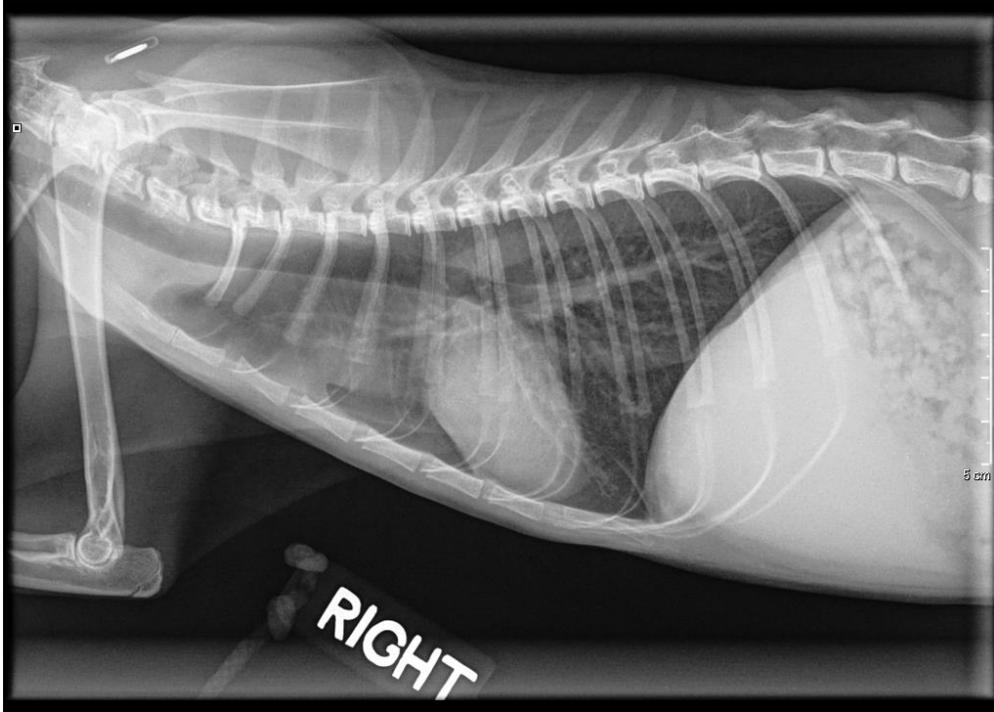


Fig 1. Right lateral thoracic radiograph



Fig 2. Left lateral thoracic radiograph



Fig 3. Ventrrodorsal thoracic radiograph

- Findings:
 - Multiple irregularly marginated, linear, gas opacities caudal to both scapulas and dorsal to and overlying the dorsal spinous processes of the thoracic vertebrae from T3-T11
 - Presence of thin linear metal opacity dorsal to left scapulo-humeral joint, consistent with microchip
 - Skeletal structures WNL; open growth plate at olecranon tuberosity, consistent with age (8 months old)
 - Portion of cranial abdomen viewed reveals markedly enlarged liver, with caudal liver lobes extending well

beyond rib cage, displacing the gastric axis caudally and to the left

- Stomach is mildly distended and contains heterogeneous, nodular, soft tissue opacity ingesta mixed with gas opacity
- Cranial mediastinum has normal width on VD view; an ill-defined, poorly marginated soft tissue opacity roughly spanning T1-T5 vertebrae present lying just dorsal to sternum overlying the cranioventral lung field on lateral views but not visualized on ventrodorsal view
- A mild, diffuse unstructured interstitial lung pattern is present, most notable in caudodorsal lung field

▪ Conclusions:

- Gas opacities within extrathoracic soft tissues consistent with subcutaneous fluid administration or trauma
- Hepatomegaly has differentials of diffuse primary or secondary lymphoma, hepatic lipidosis, feline infectious peritonitis, and toxoplasmosis, with lesser considerations to nodular hyperplasia and other types of neoplasia
- Soft tissue opacity in cranioventral lung field has differentials of thymus and enlarged sternal lymph nodes, with likely causes due to an infectious agent or neoplasia such as lymphoma, with lesser consideration given to other types of neoplasia
- Mild, diffuse, unstructured interstitial lung pattern has differentials of early or resolving interstitial edema, diffuse neoplastic infiltration such as lymphoma, or viral or protozoal pneumonia
- Recommendations include abdominal radiographs and abdominal ultrasound for further evaluation

○ Abdominal Ultrasound



Fig 4. Left kidney



Fig 5. Spleen

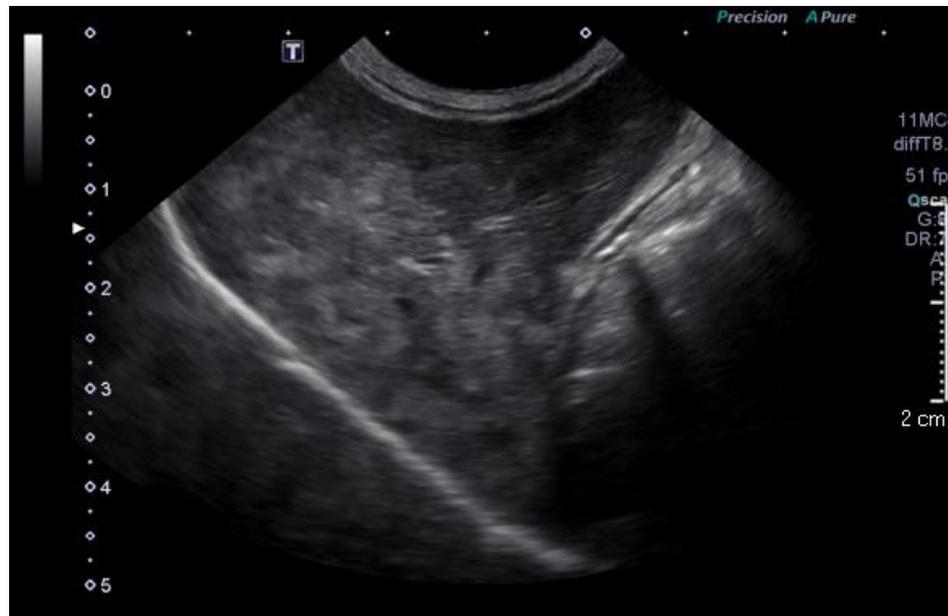


Fig 6. Liver



Fig 7. Abdominal lymph node

- Findings:
 - Enlarged left kidney (5.1 cm length) with 1.5 mm thick hypoechoic rim and thin hyperechoic medullary rim (Fig 4); normal length is 3.0-4.3 cm
 - Reportedly enlarged right kidney (4.6 cm length) with similar characteristics as left kidney (no image provides adequate visualization to confirm this finding)
 - Coarse splenic echotexture with ill-defined margination on picture (Fig 5)

- Multifocal to coalescing nodules in liver parenchyma (Fig 6)
- Liver reported to be subjectively enlarged (Fig 6 not adequate to visualize this finding)
- Enlarged, heterogeneous jejunal lymph nodes (1.9 cm thickness) (Fig 7); up to 7.2 mm in diameter is normal
- Reported scant abdominal and retroperitoneal effusion (no image provides adequate visualization to confirm this finding)
- Conclusions:
 - Findings are consistent with feline infectious peritonitis due to findings of bilaterally enlarged kidneys with a hypoechoic cortex and hyperechoic medullary rim, multifocal to coalescing hepatic nodules with hepatomegaly, and abdominal lymph node enlargement, when considered with patient's history.
 - Lesser considerations include lymphoma; while many findings coincide with those found with lymphoma, kidneys did not have the hyperechoic cortex typical of renal lymphoma.
 - Recommendations include an FNA of lymph nodes or biopsy for a definitive diagnosis.
- Ultrasound-guided FNA of abdominal lymph nodes: Consistent with macrophagic inflammation with possible mild neutrophilic inflammation. No organisms seen.
- Ultrasound-guided FNA of liver: Consistent with macrophagic inflammation with possible neutrophilic inflammation. No organisms seen.

Discussion: FIP, or Feline Infectious Peritonitis, is a systemic, immune-mediated, fatal disease of felids that is caused by a mutation of feline coronavirus (FCoV), characterized by insidious onset of lethargy, inappetence, persistent fever that is non-responsive to antibiotics, pyogranulomatous tissue reactions, body cavity effusions, neurologic signs, and/or uveitis, typically in young cats between 3 months and 2 years old.

Normal (benign) FCoV, transmitted via the fecal-oral route, replicates in enterocytes only, causing gastrointestinal upset; however, a mutation of the spike protein causes the virus to lose the ability to replicate in the enterocytes. This mutation can occur in both FCoV-1, which is the most common type, as well as FCoV-2. Antiviral antibodies are produced, and the virus is then phagocytized by macrophages, which then distributes the virus systemically. A second mutation is suspected to allow the virus to replicate within the macrophages; perivascular viral replication then ensues, causing vasculitis and granulomatous or pyogranulomatous lesions in target organs such as the CNS, eyes, liver, kidneys, intestines, abdominal lymph nodes, and lungs.

Classic history includes lethargy, gradual weight loss, decreased appetite, stunted growth in kittens, and fluctuating persistent antibiotic-resistant fever. Owners may notice a gradual increase in abdominal contour if ascites has occurred. Classic presentation includes poor body condition with dull, roughened hair coat, and thickened intestines. Additional presenting features include icterus, dyspnea and muffled auscultation due to thoracic effusion, abdominal distention with a possible fluid wave due to abdominal effusion, organomegaly felt upon palpation, signs of ataxia, personality changes, nystagmus, and/or seizures due to encephalitis, and ocular changes consistent with uveitis, such as aqueous flare, keratic precipitates, vitreous clouding, vascular cuffing, color changes to the iris, and hyphema. There is some debate as to whether a distinct “wet form” and “dry form” exist, or if these are just different stages or manifestations of the same disease, as cats with effusion have microgranulomas, and cats with the “dry form” may develop effusion later in the disease process. Treatment and prognosis are similar, regardless of whether effusion is present or not.

There is no definitive test for FIP; however, a clinical picture can be developed by combining a variety of diagnostic tests, as FIP is typically a diagnosis of exclusion. The most reliable diagnostic test is detection of the FCoV antigen in macrophages by immunofluorescence or immunohistochemistry of effusions or affected tissues; in cats without effusion, organ biopsies are necessary to do this testing. FCoV antibody test is minimally invasive, but a negative result doesn't rule out FIP, and a positive result only indicates that the patient has been exposed to FCoV in its lifetime. Conventional blood PCR cannot differentiate between FCoV and the mutated version. A new PCR has been designed to detect the specific mutation at the spike protein, but extensive field testing has not been conducted yet. CBC has limited value as it typically reveals a stress leukogram with a mild nonregenerative anemia of chronic inflammation. Chemistry Panel can be normal, or have hyperglobulinemia and hyperbilirubinemia. The Rivalta test may be performed on cats with effusion to assess ability of the effusion to coagulate; sensitivity is 91%. Fluid analysis typically reveals a clear, viscous, straw-colored exudate with >3.5 mg/dL protein and mildly elevated total cell count, with possible flecks of white fibrin. Thoracic radiographs may reveal pleural effusion and granulomatous nodules in cats with FIP presenting for dyspnea. Abdominal radiographs may reveal peritoneal effusion with loss of serosal detail, enlarged abdominal lymph nodes, seen as a localized patchy soft tissue opacity in the center of the abdomen, and hepatomegaly. Additionally, kidneys are frequently markedly enlarged and have smooth or irregular margins bilaterally. With cats that have a large amount of peritoneal effusion, a pendulous abdominal contour will be evident. Ultrasound may reveal granulomatous lesions in the target organs (liver, kidney, intestines) abdominal lymph node enlargement, kidney enlargement with a hyperechoic medullary rim parallel to the corticomedullary junction and smooth or irregular margins, intestinal thickening due to associated enteritis, and peritoneal effusion. CT or MRI may reveal granulomatous lesions, cavitory effusions, and changes in size and margination of organs, with the addition of CNS abnormalities such as hydrocephalus and ependymitis in cats with a neurological manifestation; ependymitis is seen on MRI as a marked contrast enhancement of the lining of the ventricles.

Treatment is limited for patients with FIP, as it is incurable. Palliative therapy includes fluids as needed, coaxing the patient to eat whatever it is willing to eat, corticosteroids such as prednisolone to suppress the immunologic response to the viral presence. Patients with effusion will benefit from thoracocentesis or abdominocentesis as indicated. Prednisolone acetate may help ocular involvement. Antiviral medications have not proven to be efficacious in clinical trials. Vaccination against FIP is not recommended by the American Association of Feline Practitioners, as it will produce an antibody-positive results and confound further testing, and has low efficacy. Prognosis is grave, as FIP causes 100% mortality, with a median survival time after diagnosis is 8 days. Some cats can live longer with immunosuppressive therapy, but ultimately succumb to the disease.

References:

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This paper is dedicated to the memory of French Fry, a very sweet, loveable young kitty who provided this learning opportunity for students of KSU CVM.