Mittens' Mysterious Marrow

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Introduction

Feline leukemia virus (FeLV) and feline immunodeficiency virus (FIV) are two of the most common infectious retroviral diseases affecting cats across the globe[1,2]. Retroviruses have a large degree of genetic variation, with FIV having greater genetic variation than FeLV[2]. The seroprevalence of FeLV is dependent on geography and risk factors[1,3]. In a cross-sectional study from 2010, it was determined that the seroprevalence of FeLV in the United States and Canada was 3.1% [3]. Cats with FeLV have three different outcomes once infected with the virus. They can develop either an abortive, regressive, or progressive infection with focal infections being reported mostly in research settings[4,5,6]. Each outcome is determined by the amount of viral load or "infection pressure" that is present as well as their immune system's response to the virus[4]. If the viremia lasts longer than 12 weeks, that normally results in a lifelong persistent viremia where the proviral DNA is incorporated into the host cell's genome[5,6].

Diagnosis of FeLV can be challenging and frustrating to many practitioners. This is due to the nature of the virus interacting with the cat's immune system which can result in fluid test results over time[4,5,7]. In this case, diagnosis was difficult due to conflicting or discordant test results in the patient. Therefore, not all diagnostic tests can be reliable, and this is when it is important to consider all the patient's clinical signs that indicate a high index of suspicion. The patient discussed below will highlight such a case.

History and Presentation

Mittens, an approximately 10-month-old, female spayed, domestic shorthair feline, presented to MSU-CVM Small Animal Emergency Services on July 18, 2020 for anemia, lethargy, and inappetence. Mittens originally presented to her primary veterinarian on July 7th,

with a one to two-week history of weight loss, pale gums, and not acting herself. Upon presentation to her primary veterinarian, a complete blood count (CBC), chemistry panel, and a FeLV/FIV SNAP test were performed. The IDEXX-SNAP test results were negative, and the CBC revealed a hematocrit of 5%. She was given injections of dexamethasone SP, vitamin K, and Cerenia. Two view thoracic radiographs were taken which did not reveal any reported abnormalities. A blood transfusion with feline type A blood was given and she was monitored overnight. On July 8th, a repeat CBC revealed a hematocrit of 8.9%. She was dewormed and discharged with Lixotinic (a vitamin and mineral supplement), doxycycline, prednisolone, and vitamin K. A recheck CBC was taken on July 10th and her hematocrit was 9.2%. Mittens improved for a few days but then stopped eating. Her owners noted that she ingested cat litter and was given a lactulose enema at her primary veterinarian. Abdominal radiographs revealed no abnormalities. Additionally, Mittens was noted to have a history of an upper respiratory tract infection with conjunctivitis in October of 2019 which was treated with Clavamox drops and triple antibiotic eye ointment by her primary veterinarian. Mittens was up to date on vaccinations but had not previously been tested for FeLV or FIV. She was rescued by her owners in September 2019 as a stray kitten and she lives at home with two other cats from the same litter who are both healthy.

Upon presentation to MSU-CVM Emergency services, Mittens was quiet, alert, and responsive. She weighed 3 kg and had a body condition score of a 3/9 (ideal being 4-5 out of 9) indicating that she was underweight. She had tacky and pale mucous membranes, indicating she was 5% dehydrated. An accurate capillary refill time could not be obtained. She was mildly tachypneic with a respiratory rate of 40 breaths per minute. Her rectal temperature was low at 99.8ºF. On pulmonary auscultation, normal bronchovesicular sounds were heard, and no crackles

or wheezes were appreciated. On cardiac auscultation, a grade 2/6 hemic heart murmur with intermittent gallop rhythm was appreciated, but her femoral pulses were strong and synchronous. The remainder of the triage exam was unremarkable.

A FIV/FeLV SNAP test was performed which was negative for the FIV antibody and negative for the FeLV antigen. A CBC revealed a marked microcytic, hypochromic anemia (packed cell volume of 3.5%), a mild thrombocytosis (696 10^3/ul), and a moderate lymphopenia (772 10^3/ul). A chemistry panel revealed a mild hyperglycemia (181 mg/dl), mild to moderately increased alanine transaminase (233 U/L), mildly increased alkaline phosphatase (48 U/L), mild hypoglobulinemia (3.6 g/dl), mild hypermagnesemia (2.8 mg/dl), and a mildly increased creatine kinase (292 U/L). Additionally, an absolute reticulocyte count revealed 4,260 /µl reticulocytes, which was indicative of a non-regenerative anemia and dysfunction at the level of the bone marrow. An appropriate regenerative response in a cat is >60,000 reticulocytes u/l. On blood smear, there was no evidence of identifiable polychromatophilic cells or nucleated red blood cells that would have indicated a regenerative anemia. Based on Mittens' pale mucous membranes, hemic murmur, and marked non-regenerative anemia she was a candidate for a transfusion of red blood cells, so she was blood typed and cross-matched. Mittens was blood typed as type A, and her plasma was compatible with the donor's red blood cells after a major crossmatch was performed.

Mittens was given a whole blood transfusion over 5 hours and was monitored closely for possible transfusion reactions. After the 4th hour of her transfusion, Mittens spiked a fever of 103ºF and the transfusion was paused. She appeared brighter but her temperature remained elevated, so the transfusion was discontinued after she received a total of 35 mL. Mittens appeared to have a febrile non-hemolytic transfusion reaction. Her packed cell volume was 17%

post-transfusion. Mittens' anorexia and lethargy improved after the transfusion, but she was seen ingesting cat litter which is also known as pica.

Diagnostic Approach

On July 19th, Mittens' PCV was 12% in the afternoon and 14% during evening treatments. A saline agglutination was negative for both the macro and micro agglutination. Based on Mittens' signalment, history, and bloodwork, dysfunction at the level of the bone marrow was suspected so a bone marrow aspirate and core biopsy were recommended and performed. Differential diagnoses under consideration included FeLV, precursor immune mediated anemia (PIMA), erythroleukemia, iron deficiency, myelophthesis, *Mycoplasma hemofelis,* and *Cytauxzoon felis*. Although Mittens' FeLV/FIV SNAP test was negative for soluble p27 antigen in the peripheral blood, feline leukemia virus was considered most likely for Mittens' case.

On July 20th, Mittens was anesthetized for a bone marrow biopsy and aspirate. Prior to anesthesia Mittens' PCV was 11% and she was febrile with a temperature of 104.3ºF. The craniomedial aspect of the left humeral head was clipped and aseptically prepped and a local lidocaine block was given subcutaneously and over the periosteum. A small stab incision was made over the skin and an intraosseous Jamshidi needle was advanced through the incision and into the cortex of the humerus. Both a bone marrow aspirate and biopsy were collected. The bone marrow sample was noted to have a grossly abnormal gelatinous consistency. A sample of the marrow was sent off for an FeLV IFA (immunofluorescent antibody) test which detects intracellular FeLV p27 antigen in the bone marrow. Throughout the procedure, Mittens was responsive to stimulation and she received 3 doses of intra-op alfaxalone (0.7mg/kg), one dose of dexmedetomidine (6 mcg/kg) and one dose of ketamine (1mg/kg). She recovered from

anesthesia uneventfully and while she was recovering, she was seen ingesting litter, so her litter was switched to shredded paper. Buprenorphine (0.01mg/kg) IV or buccally q8 was added to her treatments for post-procedural pain management for several days following the biopsy.

That afternoon, Mittens received another blood transfusion because she began showing clinical signs of anemia, tachypnea and tachycardia. She did not have any evidence of a transfusion reaction but her respiratory rate increased after the first hour but then returned to normal for subsequent readings thereafter. She remained febrile throughout the transfusion. Her bone marrow cytology revealed a left shift of the erythroid lineage with proliferation of prorubricytes and rubriblasts. There were also macrophages that were noted to be phagocytizing erythrocytes and erythroid precursors. These results were consistent with a precursor targeted immune mediated anemia (PIMA), which has also been previously referred to as pure red cell aplasia (PRCA) or non-regenerative IMHA (NRIMHA) in several papers[8,9,10].

In a paper that studied bone marrow findings in dogs with suspected PIMA, the criteria that classified a dog with PIMA included "a persistent nonregenerative anemia, ineffective erythropoiesis, dysplastic features in no more than rare cells of any lineage, and clear selective phagocytosis of intact erythroid precursors by macrophages in the bone marrow or a concurrent splenic aspirate"[8]. These dogs also did not demonstrate erythrocyte agglutination that is seen with typical IMHA and their presenting clinical signs included lethargy, anorexia/hyporexia, weight loss, collapse, exercise intolerance, vomiting, seizures, and pica[8]. They also found that the detection of rubriphagocytosis is rarely present in dogs with no evidence of PIMA and its presence increases confidence in a clinical diagnosis of PIMA[8]. In this study, they classified early PIMA as early maturation arrest with predominance of rubriblasts while they classified PRCA as absence or paucity of erythroid precursors without a left shift or rubriphagocytosis[8].

The various terms encompass a similar spectrum of the same disease process, which is immune mediated destruction of different stages of erythrocyte cell maturation[8,9,10]. Typically, treatment of this disease is with immunosuppressive drugs such as prednisolone alone, or in conjunction with another immunosuppressive therapy[8,9,10].

Mittens was started temporarily on dexamethasone SP (0.4mg/kg) IV q24 for her diagnosis of PIMA. The following day, prednisolone at 2mg/kg PO q24 was initiated for long term management. The results of the bone marrow core biopsy revealed a diffuse, severe, erythroid hypoplasia with a profound decrease in erythroid precursor populations shifting the M:E ratio to 5:1. Not only was there a production problem of red blood cell progenitors, there was also destruction of the bone marrow. The results of the IFA test were positive for FeLV p27 antigen in the bone marrow. An IFA test is a confirmatory test for feline leukemia virus and not a screening test because it is less sensitive than ELISA (enzyme-linked immunosorbent assay) and false positive and negative results can occur[5,6,7]. In some cases, cats can be negative for circulating antigen using ELISA but have bone marrow cells that test positive with IFA[5]. These results confirmed our suspicions and a final diagnosis of progressive FeLV was made.

Diagnosis

The diagnosis of retroviral infections in cats are done with screening tests and confirmatory tests. These tests include ELISA on blood or serum, ELISA on tears/saliva, IFA, PCR, and virus isolation[4,5,7]. The AAFP recommends screening all cats for infection at the time they are acquired, prior to initial vaccination against FeLV and FIV and following potential exposure of infected cats[7]. In a study from 2017, they compared the diagnostic performance of four different FeLV antigen/FIV antibody combination test kits used in practices around the world. They found that the ELISA IDEXX SNAP had a 100% sensitivity and 100% specificity

and had the best performance out of all the screening tests[11]. The study also found that many of the point-of-care test devices would report false positives based on a seroprevalence between 1-5%[11]. Screening tests should be highly sensitive to identify the animals that truly have the disease[6]. When tests lack sensitivity, infected cats may not be detected and remain at risk for infecting other cats[7]. Because a positive screening test has potential important clinical consequences, additional confirmatory tests are recommended, especially in a population where prevalence is low[7]. For example, in a single-cat household where the cat is indoors only.

The ELISA blood test requires that whole blood be used[6,7]. A positive test means that cell-free p27 antigen is found in the blood and the cat is actively shedding the virus. After a cat is infected with FeLV, this test will detect p27 antigen by 4 weeks of initial infection[6,7]. The limitations of the test are that it does not detect if the virus has invaded the bone marrow and it won't identify cats with regressive/latent infections[4,5,7]. The other ELISA test detects virus in saliva and tears. The last stage of FeLV infection is when the virus replicates within the epithelial tissues of the salivary glands[5,6]. This test is not recommended to be used as a screening test because it is insensitive with many false positive and negative results[7,11].

There is no universal gold standard test for retroviral infections in cats[7]. Even with all the options for diagnosis, it can still sometimes be difficult to identify positive cats because the test status in some cats is very fluid. Virus isolation can be used as a confirmatory test, but it is mostly used in research settings and is not practical for routine diagnosis because of long turnaround times[4,5,7]. Confirmatory tests are not always necessary, but they are recommended if the positive predictive value is low and if a healthy cat will be euthanized based on a single ELISA[6,7]. Another situation where a confirmatory test is recommended is if an animal is showing clinical signs but has a negative ELISA test, which was the case with Mittens. According to the

AAFP, cats with discordant test results should be considered potential sources of infection for other cats until their status is clarified[7]. The IFA test can be done on whole blood or bone marrow and it detects marrow-origin p27 antigen in cells[5,7]. This test identifies cats that have progressive infection but cats with early viremia will test IFA-negative and ELISA positive[5,7]. That is why IFA is less sensitive than the ELISA test. IFA is also prone to false positive results if laboratory personnel are not trained to identify immunofluorescence properly[5,6,7,12]. Lastly, DNA PCR is another confirmatory test that will detect positive cats within 2 weeks of exposure[6,7]. This test detects proviral DNA in the cat's genome, but it does not equate with viral replication. It can detect cats that are either regressive or progressive. There is also a new IDEXX quantitative PCR test that stages cats by determining the proviral load and can group cats into the regressive or progressive stage[4,6,7].

Pathophysiology

Feline leukemia virus is an enveloped, single-stranded RNA virus that belongs to the genus *Gammaretrovirus* of the family *Retroviridae*. It belongs to the subfamily *Oncornavirinae* which means "oncogenic RNA virus"[5,6,12]. FeLV and FIV are both in the family *Retroviridae* and share similar structures except that the capsid shape of FeLV is icosahedral and FIV is cone shaped[5]. Feline leukemia virus produces the enzyme reverse transcriptase which catalyzes the single stranded viral RNA into a double-stranded viral DNA copy, called the provirus[2,5,12]. The provirus is inserted into the host cell and integrates itself into the host cell genome by viral integrase protein[5,12]. There are two distinct groups of structural proteins within FeLV, which include core proteins and envelope proteins. The core protein is composed of the capsid protein p27, which is the basis for commercial ELISA and IFA testing. The p27 antigen is present in the cytoplasm of infected cells, peripheral blood, saliva, and tears of infected cats[5]. The two

envelope proteins consist of p15e and gp70; p15e induces immunosuppression while glycoprotein 70 (gp70), facilitates viral attachment and invasion of the host cell as well as stimulates production of protective antibody[5,6]. Glycoprotein 70 also contains the subtype antigens A, B, C, and T[5,6]. These subgroups are associated with the infectivity, virulence, and clinical syndrome caused by individual strains of the virus[5,6,12].

There are three main subtypes of FeLV: FeLV-A, FeLV-B, and FeLV-C[5,12]. The fourth subtype, FeLV-T has been associated with immunosuppression and has a tropism for T-lymphocytes[2,5]. Each subtype uses a different receptor to enter cells and only FeLV-A is transmitted between animals[5]. FeLV-B arises through recombination of FeLV-A proviral DNA and FeLV-C arises from accumulation of mutations or insertions in the *env* gene of FeLV-A[5]. All cats infected with FeLV-B and FeLV-C are co-infected with FeLV-A but FeLV-B and FeLV-C are more pathogenic than FeLV-A[5]. Feline leukemia virus subtype A is transmitted primarily via close contact with salivary secretions including mutual grooming and sharing of food/water dishes[5]. Other routes of transmission include biting, blood transfusion, litterbox, blood contaminated instruments, and endotracheal tubes[5,6]. Vertical transmission from queens to kittens is also an important mode of transmission because this can lead to thymic atrophy and "fading kitten syndrome"[5,6]. This syndrome causes depletion of T-lymphocytes and 80% of kittens die while the remaining 20% will often be persistently viremic[5,12]. FeLV-B may accelerate the development of lymphoma or enhance neuropathogenicity and subtype C is associated with non-regenerative anemia[5].

Once a cat is exposed to FeLV via oronasal exposure, the virus replicates in the oral lymphoid tissue[5,6,12]. It then infects a small number of circulating monocytes and lymphocytes within the peripheral blood[5]. The virus proliferates within the spleen, lymph nodes, and gut-

associated lymphoid tissues[5]. Finally, a small number of infected lymphocytes reach the bone marrow, and the virus infects rapidly dividing precursor cells, including marrow-origin neutrophils and platelets and establishes a peripheral viremia[5,6]. This occurs 28 days post exposure and the final step of viral replication includes widespread epithelial infection where the virus is shed in the saliva and urine[5,12]. There are four potential outcomes of FeLV infection which include abortive, focal, regressive, and progressive infections[5,6].

Cats with abortive infections do not develop viremia because of low-dose exposure to FeLV[5]. The virus infects and replicates minimally in the oropharyngeal lymphoid tissues[5]. After exposure to the virus, infected cats will have high levels of neutralizing antibody but will test negative on antigen and PCR tests[5,6]. These cats are not contagious to other cats[5,6]. Focal infections, while rare, are characterized by virus that is localized to an organ or tissue and is not found in the blood or bone marrow[5]. These cats may shed virus intermittently which could lead to discordant test results[5,7]. In regressive infections, proviral DNA is inside host cell genomes but production and shedding of the virus no longer occurs[5]. The viremia can occur for three weeks and then resolve, or it can become a more durable viremia that can last up to 12 weeks[5,6]. During this viremia they are actively shedding virus and are infectious to other cats[5,6]. Eventually, neutralizing antibodies can clear the viremia but are unable to eliminate the provirus inside the host cell genome[5,6]. When this occurs, the cat is considered to have a latent infection which has the potential to reactivate with stress, illness, immunosuppression, or pregnancy[4,5,6]. There are a few clinical syndromes caused by regressive infection that occur without reactivation of the virus and those include bone marrow suppression and lymphoma[4].

The most clinically important form of infection is the progressive infections because it is associated with a variety of FeLV-related diseases[4,5]. Once the virus invades the bone marrow

this results in a lifelong or persistent viremia[5]. These cats rarely revert to ELISA or IFA negative status and they have either low or no levels of neutralizing antibody[5,6,7]. Some clinical outcomes of the disease include immune suppression leading to opportunistic infections, hematologic disorders, neoplasia, immune-mediated diseases, peripheral lymphadenopathy, neurologic disease, reproductive failure, gastrointestinal disease, panleukopenia like syndrome, oral inflammation (stomatitis), and cutaneous horns[4,5,6,12]. Age is an important host factor in a cat's susceptibility to infection with FeLV[4]. Adult cats have a natural age-related resistance while the likelihood of becoming progressively infected is higher in kittens[4,5]. Typically, younger cats experience more opportunistic infections like bacterial upper respiratory tract infections, urinary tract infections, hemoplasmosis, FIP, dermatophytosis, toxoplasmosis and cryptococcus[5,6,12]. This predisposition towards secondary infections has been attributed to the immunosuppressive properties of the viral envelope peptide, p15e which inhibits T and B cell function and impairs cytokine production[5,12]. In a study that determined the association between oral health status and retroviral seropositivity, they found that cats with oral inflammatory disease such as stomatitis, gingivitis and periodontitis were significantly more likely to be seropositive for FeLV and FIV than healthy cats[13].

Cats are more likely to present to their veterinarian for anemia or immunosuppression than they are for leukemia[5]. The most common hematologic disorders caused by FeLV include anemia and cytopenias due to bone marrow suppression[5]. Approximately 90% of FeLV associated anemias are nonregenerative and cause mild to severe anemia[5]. The mechanisms of anemia in cats infected with FeLV are decreased erythrocyte production, loss, and destruction. Infection with FeLV subtype C results in pure red cell aplasia and non-regenerative anemia causing macrocytosis[5,12]. The mechanism occurs through FeLV-C binding and interfering with

heme exporter protein and causing heme toxicosis inside erythrocytes[5]. The virus causes erythrocytes to be released from the marrow that are large, immature, and nucleated but there is an absence of reticulocytosis[5]. So, the anemia may appear regenerative, but it is actually non-regenerative[5]. FeLV can also lead to thrombocytopenia or thrombocytopathy, neutropenia or neutrophil dysfunction, and aplastic anemia[5,12]. Myelophthsis secondary to leukemia, myelofibrosis and myeloid and/or erythroid dysplasia can also cause anemia and bone marrow dysfunction[5]. Also, opportunistic infections or neoplasia causing inflammation can lead to anemia of inflammatory disease[5]. Lastly, FeLV can cause red blood cell destruction because of immune-mediated hemolytic anemia or co-infection with *Mycoplasma hemofelis*[5,12].

Lymphoid malignancies, like lymphoma and lymphoid leukemia are the most common neoplasms seen with FeLV infection[5,6]. Lymphoma is typically T- cell in origin and the most common types are mediastinal, multicentric, spinal, renal, or ocular[5,6]. The virus does not have oncogene but can be oncogenic by two mechanisms that include insertional mutagenesis and recombinant virus formation[5,6,12]. Insertional mutagenesis is more common and occurs when the FeLV provirus inserts itself into the host genome and activates proto-oncogenes or disrupts tumor suppressor genes[5,6]. Recombinant virus formation involves incorporating host cell oncogenes sequences that can be rearranged and activated to induce malignancy[5,6].

Treatment and Prevention

There is no proven treatment that will clear an FeLV infection. Several medications have been researched for treatment of FeLV and those include Zidovudine also known as azidothymidine (AZT), feline recombinant interferon omega, and human recombinant interferon alpha. AZT is a nucleoside analog that blocks reverse transcriptase of retroviruses[14]. In one study, it was shown to reduce antigenemia, improve oral cavity inflammation and prolong

lifespan in both naturally and experimentally infected cats[5]. AZT is more beneficial in the treatment of FIV than FeLV[14]. Human interferon (IFN) has immunomodulatory effects and has shown efficacy in vitro by inhibiting FeLV replication. In studies involving naturally infected cats, however, they became refractory to treatment after a few weeks due to development of anti-human-IFN antibodies[5,6,14]. Feline recombinant interferon has anti-viral, anti-tumor and immune modulating properties[14]. This treatment is available in Europe for treatment of cats with FeLV and FIV. One study showed that feline interferon had significant therapeutic effects on clinical signs and increased survival time as well as quality of life in cats[14,15].

Cats with opportunistic infections may require longer periods of treatment compared to cats without FeLV[5,14]. Lymphoma and leukemia are treated with the CHOP and COP chemotherapy protocols[5,14]. Unfortunately, many studies consider FeLV infection to be a negative prognostic indicator in cats with lymphoma [5,16]. One paper found that FeLV-positive cats tend to have significantly shorter remission and survival times with available chemotherapeutic protocols[16]. In anemic cats, periodic blood transfusions may be needed especially if the anemia is severe[6].

In cases of FeLV that cause immune-mediated cytopenias and IMHA, these respond well to treatment with glucocorticoids and other immunosuppressive therapies[6]. Prednisolone alone or in conjunction with other immunosuppressive drugs is used to treat PRCA and PIMA/NRIMHA[8,9,10]. Typically, prednisolone is started at an immunosuppressive dose of 2mg/kg/day PO[17]. Remission can take weeks to months and it is important to recheck hematocrit or perform serial CBCs every 2 weeks until the anemia is in remission[8,17]. Once the PCV normalizes and remains stable, the dosage of glucocorticoids can be tapered by 20-25% every 4-6 weeks[17]. Side effects of long-term glucocorticoid administration in cats include diabetes

mellitus[17]. Glucocorticoids also cause fluid retention so they should be used cautiously in patients with concurrent heart disease[17].

The best way to prevent retroviral infections is through testing, vaccination, owner education, and environmental management of infected cats[7]. There is a recombinant non-adjuvanted vaccine and an inactivated adjuvanted or killed vaccine available[7]. Vaccines usually provide immunity that lasts 1 year[5,7]. The AAFP recommends FeLV vaccination in kittens and it should be part of their core vaccine protocol[7]. After cats are one year of age, vaccination can be discontinued if there is no further risk based on lifestyle, environment, and overall health status[7]. The commercial vaccines provide protection against progressive infections but vaccination will not always prevent proviral DNA integration after FeLV exposure[5,7,]. In one study, they found that cats that were not vaccinated against FeLV were 7 times more likely to become infected with FeLV than cats that had been previously vaccinated[18]. The FeLV vaccine has been associated with injection site sarcomas (ISS), so vaccination should be considered in cats that are likely to be exposed[5,7]. Vaccines should be given below the stifle and elbow in case an ISS forms and amputation of a limb is required[7]. Another way of preventing FeLV include indoor housing of cats to decrease exposure to infected cats[7]. One study demonstrated that the FeLV vaccine could be given in the tail which is an easier site for amputation and may facilitate earlier detection of ISS[19]. Cats should be tested and separated from antigen-positive cats and screened prior to donating blood[5,7]. Food and water bowls as well as litterboxes should not be shared between antigen-positive and antigen-negative cats[5,7].

Case Outcome

Mittens did well in the hospital after she was started on prednisolone. She appeared brighter and her appetite improved. She was discharged on July 23rd, with instructions to continue the prednisolone and follow up with her primary veterinarian on July 27th to recheck her anemia with a CBC and reticulocyte count. Her owners were told about the potential side effects of steroids and its ability to induce diabetes mellitus. Her poor long-term prognosis due to her progressive FeLV infection was discussed with her family. While cats with progressive infection have a shorter life expectancy, they can be clinically healthy for many years but their chance of getting lymphoma increases by 60-fold compared to uninfected cats[5]. It was unknown how long Mittens would be shedding FeLV unless her owners retested her in the future. It was recommended to Mittens' family that they temporarily separate Mittens from the other cats in the house and separate food and water bowls as well as litter boxes. Isolation of retrovirus infected cats can be difficult for owners. It is important to counsel owners about best practices to decrease disease transmission for example, making sure environmental needs of all cats in the home are met to reduce conflict and stress[7]. It is possible that Mittens' littermates may already have the virus but to decrease their chances of becoming progressively infected, it was also recommended that they get re-vaccinated for FeLV.

A complete blood count on August 18th revealed a hematocrit of 31%. Once her hematocrit reached 35%, her prednisolone dose was decreased by 25%. Overall, Mittens continues to do well at home with no signs of anemia and she has been temporarily separated from her littermates.

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