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# CHAPTER 96
# VIRAL INFECTIONS

Jane E. Sykes, BVSc(Hons), PhD, DACVIM

**KEY POINTS**

- A number of viral infections may be associated with acute and severe illness, leading to presentation of affected dogs and cats to emergency and critical care veterinarians.
- Treatment of viral infections is generally supportive and includes intravenous fluid therapy, early nutrition, antiemetic therapy, supplemental oxygen therapy, and antibiotics for secondary bacterial infections. Hospitalization in isolation may be required.
- The feline leukemia virus and feline immunodeficiency virus status of all cats should be known.
- The use of antiviral medications is limited, and few controlled studies evaluate their effectiveness in dogs and cats. Famciclovir can be effective for treatment of severe infections by feline herpesvirus 1.
- The range of diagnostic tests for viral infections in dogs and cats has increased with the availability of nucleic acid–based assays, such as the polymerase chain reaction (PCR). Quality control for PCR assays can be problematic. The use of laboratories that perform real-time (fluorogenic) PCR and that include a quality assurance program can lessen the chance of false-positive results.

A large number of viruses can cause acute and severe illness in dogs and cats (Table 96-1). The most common or important viral infections that may come to the attention of emergency and critical care veterinarians are canine parvovirus (CPV), canine distemper virus (CDV), canine influenza virus (CIV), feline panleukopenia virus, feline herpesvirus 1 (FHV-1), feline calicivirus (FCV), feline infectious peritonitis virus (FIPV), feline immunodeficiency virus (FIV), feline leukemia virus (FeLV), and rabies virus infection. The FIV and FeLV status of all cats should be determined on arrival by questioning the owner or testing using in-house enzyme-linked immunosorbent assays for FeLV antigen and FIV antibody. Because cats may be infected subclinically by these viruses and because some cats subsequently undergo regressive FeLV infections, positive test results alone are not reason for euthanasia. CPV infection is covered in the following chapter. Other viral diseases that may present to emergency and critical care veterinarians include enteric viral infections such as rotavirus and coronavirus infections, feline paramyxovirus infection, pseudorabies virus infection, vector-borne viral infections such as West Nile virus infection, infectious canine viral hepatitis, and canine herpesvirus infection.

An extensive discussion of the etiology, clinical signs, diagnosis, treatment, and prevention of every one of these infections is beyond the scope of this chapter. Instead, the purpose of this chapter is to provide the reader with an update on selected common and important viral infections in dogs and cats that may be evaluated by emergency and critical care veterinarians. Treatment of viral infections is largely supportive and usually includes intravenous fluid therapy, early enteral or parenteral nutrition, antiemetics, analgesia, and oxygen therapy when pulmonary disease is present. Blood products may be needed for cats with retroviral infections. Antibiotics may be needed for secondary bacterial infections. Attempts to culture secondary bacterial invaders and determine sensitivity to antimicrobial agents should be considered before commencing antimicrobial therapy. Use of antiviral medications is still limited in dogs and cats, but famciclovir can be effective for treatment of severe infections with feline herpesvirus 1.

## CANINE DISTEMPER VIRUS INFECTION

CDV infection is a contagious disease of dogs that may involve the gastrointestinal (GI), respiratory, or neurologic systems. Distemper still occurs sporadically, even in vaccinated dog populations. Disease most commonly occurs in dogs 3 to 6 months of age, when maternal antibody level is declining, but can occur in older dogs that have been vaccinated infrequently or improperly, especially after stress, immunosuppression, or contact with other affected dogs.[1]

CDV is an enveloped ribonucleic acid (RNA) virus that belongs to the family Paramyxoviridae. The virus survives for about 3 hours at room temperature and is highly susceptible to routine hospital disinfectants such as quaternary ammonium compounds. Several strains of CDV exist and vary in pathogenicity. Some, such as the Snyder Hill strain, are more likely to produce neurologic disease than others. A study has documented the existence of CDV strains that differ from vaccine strains and from those previously documented in the United States.[2]

**Table 96-1 Viral Infections to Be Included on the List of Differential Diagnosis in Dogs and Cats with Respiratory, Gastrointestinal, or Neurologic Symptoms**

| | Affected Body System | | |
|---|---|---|---|
| **Species** | **Respiratory** | **Gastrointestinal** | **Neurologic** |
| Dog | Canine distemper<br>Influenza viruses<br>Canine parainfluenza<br>Canine adenovirus<br>Canine herpesvirus<br>Canine respiratory coronavirus<br>Possibly other viruses such as canine pneumovirus | Canine distemper<br>Canine parvovirus<br>Canine enteric coronavirus<br>Rotaviruses, astroviruses, adenoviruses, caliciviruses, and several other novel viruses such as norovirus | Canine distemper<br>Rabies<br>Arthropod-borne infections (togaviruses, bunyaviruses, and flaviviruses)* |
| Cat | Feline calicivirus<br>Feline herpesvirus<br>Feline infectious peritonitis<br>Influenza viruses<br>Retroviruses† | Feline panleukopenia<br>Feline coronavirus<br>Rotavirus<br>Retroviruses[1] | Feline panleukopenia<br>Feline infectious peritonitis<br>Rabies<br>FIV<br>Retroviruses†<br>Paramyxoviruses |

*FIV,* Feline immunodeficiency virus.
*These also have the potential to cause disease in cats, but disease has been reported more often in dogs. Most animals are infected subclinically.
†Feline retrovirus infections also may be associated with these signs through induction of neoplastic disease or secondary infections resulting from immunosuppression.

CDV is shed in respiratory secretions for up to 90 days after infection. Initial replication of CDV is in lymphoid tissue, and viral destruction of lymphocytes results in lymphopenia and pyrexia. Approximately 1 week after infection the virus spreads to epithelial tissues (lungs, GI tract, kidney, bladder) and the central nervous system (CNS), and virus shedding begins. Poor cell-mediated immunity (CMI) is associated with spread of the virus to a variety of tissues, severe respiratory and GI signs with or without CNS involvement, and death. Dogs with an intermediate or delayed CMI response may develop persistent infection of the uvea, CNS, and footpad and nasal epithelium, leading to neurologic, cutaneous (hard pad), and ocular signs such as chorioretinitis. Infection with CDV is highly immunosuppressive, and secondary infections with opportunistic pathogens such as *Nocardia* and *Salmonella* spp. may occur.

Distemper should be high on the list of differential diagnoses for any dog with respiratory and/or CNS signs. Mild signs are common and resemble those of kennel cough. Severe, generalized distemper may begin with a serous to mucopurulent conjunctivitis and rhinitis and progress to include signs of lower respiratory disease, lethargy, anorexia, vomiting and diarrhea, severe dehydration, and death. Neurologic signs then occur in some dogs, either with systemic illness or after a several-week delay. Neurologic signs are frequently progressive despite treatment and are a poor prognostic sign. Myoclonus, an involuntary twitching of various muscle groups, can be most pronounced when affected dogs are at rest and is virtually pathognomonic for CDV infection. Ocular signs may consist of sudden blindness resulting from optic neuritis, chorioretinitis, or retinal detachment. Cutaneous signs may be useful for prognostication. Footpad and nasal hyperkeratosis often are accompanied by neurologic complications, whereas the presence of vesicular and pustular dermatitis implies a good CMI response and rarely is associated with neurologic complications.

Physical examination of dogs suspected to have distemper should include a fundic examination, careful inspection of the skin, including the nose and footpads, and careful thoracic auscultation. Any dog suspected to have distemper should be placed in isolation if possible. This may be complicated by a requirement for oxygen therapy.

The most commonly used diagnostic test for distemper is cytologic examination of conjunctival scrapings. Acutely, these may show cytoplasmic inclusions in epithelial cells when stained with Wright or Diff-Quik stain (Figure 96-1). The sensitivity of cytology is increased after application of immunofluorescent antibody to smears by regional diagnostic laboratories. Smears should be air dried and, if possible, fixed in acetone for 5 minutes before transport. Intracytoplasmic inclusions also may be seen in erythrocytes, lymphocytes, other white blood cells, and cells within the cerebrospinal fluid (CSF). Thoracic radiography may reveal an interstitial pattern or an alveolar pattern with secondary bacterial bronchopneumonia. Analysis of CSF may show increased protein and cell count, and measurement of anti-CDV antibody in the CSF also can be useful for diagnosis in dogs with neurologic signs. Other antemortem diagnostic tests for distemper include immunohistochemistry for CDV antigen on biopsies of nasal mucosa, footpad epithelium, and haired skin of the dorsal neck, and reverse transcriptase–polymerase chain reaction (RT-PCR) testing for viral nucleic acid. Specimens suitable for RT-PCR testing include buffy coat cells, whole blood, serum, CSF, and urine.[3] With any PCR assay, quality control can be problematic, and the use of laboratories that perform real-time (fluorogenic) PCR and that have a quality assurance program is recommended. Virus isolation is difficult and is not used widely for diagnosis.

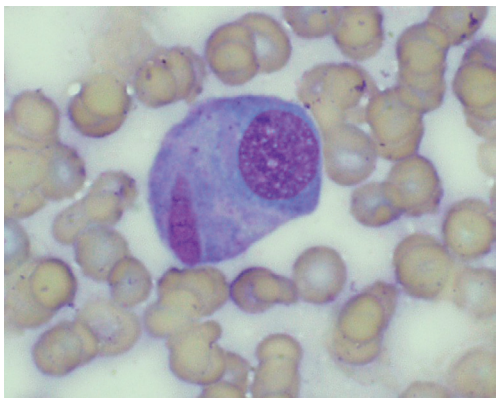

**FIGURE 96-1** Distemper virus inclusion within the cytoplasm of a conjunctival epithelial cell. Diff-Quik stain.

Attenuated live vaccines can prevent canine distemper and should provide at least partial protection even in the face of variant strains. The interested reader is referred to a comprehensive review of vaccination for CDV for further information on the topic.[1]

## CANINE INFLUENZA VIRUS INFECTION

Canine influenza first appeared in racing Greyhounds in Florida between 1999 and 2003.[4] At the time of writing, evidence of CIV infection has been detected in dogs in animal shelters, adoption groups, pet stores, boarding kennels, and veterinary clinics in at least 38 U.S. states. The most significant outbreaks of disease resulting from CIV have occurred in Florida, New England, Colorado, Wyoming, and Texas. Sequence analysis has indicated that the virus isolated from dogs shares more than 96% homology with equine influenza A. All the genes from the canine isolates are of equine influenza virus origin, providing evidence that the virus crossed the species barrier. Concern has been raised by scientists that this virus also may have the potential to cross the dog-human species barrier, as occurs with avian influenza viruses. Influenza viruses are enveloped viruses that are susceptible to routine hospital disinfection practices.

Clinical signs occur 2 to 5 days after exposure to the virus. As with distemper, canine influenza virus causes a syndrome that may mimic kennel cough, although fever may be more likely to occur with influenza virus than with parainfluenza virus, adenovirus, or *Bordetella bronchiseptica* infections. Nearly 80% of exposed dogs develop clinical signs, which consist of a cough that persists for 2 to 3 weeks despite therapy, serous to mucopurulent nasal discharge, and a low-grade fever. Some dogs develop more severe pneumonia with a high fever (104° to 106° F), tachypnea, and respiratory distress. The overall mortality has been less than 5%. Shedding of virus occurs for 7 to 10 days after the onset of clinical signs.

Dogs with these signs should be placed in isolation. Findings on thoracic radiography are the same as those described above for distemper. Antemortem diagnosis of CIV infection relies on serology using hemagglutination inhibition, RT-PCR, or virus isolation. To distinguish past exposure from recent infection, serology should be performed on samples collected at the time of presentation and 2 to 3 weeks later. Because most dogs have not yet been exposed positive results in a single specimen collected 7 days after onset of clinical signs may be suggestive of current infection.

Nucleic acid testing using RT-PCR is offered by a few laboratories and can be performed on pharyngeal swab specimens. Pharyngeal swabs should be kept refrigerated and transported as soon as possible on ice to the laboratory performing nucleic acid testing. Detection of virus appears to be difficult beyond 3 to 4 days after the onset of clinical signs; the same is true for virus isolation.[5] Virus isolation and RT-PCR also can be successful when performed on lung tissue from dogs that have died within 2 to 3 days of the onset of clinical signs. Swabs for virus isolation must be placed in virus transport medium.

Treatment of serious influenza virus infection in human patients has involved use of the neuraminidase inhibitor oseltamivir phosphate, which inhibits spread of the virus from cell to cell.[6] Anecdotal reports exist regarding treatment of dogs with this drug, but no published studies are available, and nothing is known regarding the optimal dosage in dogs to inhibit viral replication. Until the results of such studies become available, use of this drug to treat dogs that have been diagnosed definitively with CIV infection is not recommended.

## OTHER EMERGING RESPIRATORY VIRAL INFECTIONS OF DOGS

Other emerging respiratory viral pathogens of dogs include canine respiratory coronavirus (CRCoV), other influenza virus types, and canine pneumovirus. CRCoV was reported first in 2003 in a group of dogs with respiratory disease in a rehoming facility in England that had been vaccinated against canine adenovirus-2, CDV, and canine parainfluenza virus.[7] It is distinct from canine enteric coronavirus. Alone, CRCoV causes subclinical infections or mild respiratory disease, but like human respiratory coronaviruses, it may cause reversible damage to, or loss of, the respiratory epithelial cell cilia. As a result, infected dogs become predisposed to secondary infections. Serologic evidence of exposure to CRCoV appears to be widespread in dogs from North America, Great Britain and continental Europe, Japan, Korea and New Zealand, and the virus has been detected widely using PCR-based methods in dogs with respiratory disease from many of these countries. Canine pneumovirus is a parainfluenza virus that belongs to the genus *Pneumovirus*. It was isolated first from dogs with acute respiratory disease in shelters in the United States in 2010.[8] The extent to which this virus causes disease in dogs remains to be investigated.

## FELINE PANLEUKOPENIA

Feline panleukopenia is caused by a small, single-stranded deoxyribonucleic acid (DNA) virus closely related to CPV. Cats with feline panleukopenia also may be infected with CPV strains 2a, 2b, and 2c.[9] Although most cats shed virus for just a few days after infection, it may be shed for as long as 6 weeks, and viral persistence in the environment plays an important role in disease transmission. The virus can survive for a year at room temperature on fomites and survives disinfection with routine hospital disinfectants; inactivation generally requires a 1:30 dilution of household bleach, potassium peroxymonosulfate, or concentrated accelerated hydrogen peroxide solutions.

Feline panleukopenia should be suspected in poorly vaccinated kittens with acute illness including fever, lethargy, anorexia, vomiting and, less commonly, diarrhea. Oral ulceration and icterus may be noted in complicated infections. Death may result from severe dehydration, secondary bacterial infections, and disseminated intravascular coagulation. Cats between 3 and 5 months of age may be most susceptible to severe disease, which is exacerbated by concurrent gastrointestinal infections.

Cats suspected to have feline panleukopenia should be placed in isolation. Supportive treatment is similar to that recommended for CPV. Diagnosis is based on clinical signs along with the finding of leukopenia on a complete blood count. Leukopenia is not always present and may occur with other diseases such as salmonellosis. Severe panleukopenia may be associated with concurrent infection with FeLV.[10] In-house fecal enzyme-linked immunosorbent assays for CPV are suitable for diagnosis of feline panleukopenia, although false-negative results may occur, so a negative test result does not rule out feline panleukopenia. Sensitivity in one study ranged from 50% to 80% depending on the kit used, and specificity ranged from 94% to 100%. False-positive fecal antigen assay results after vaccination with attenuated live viral vaccines appear to be uncommon but vary with the test used.[11] PCR assays are also available for detection of viral DNA in fecal and tissue specimens from affected cats. Cats with panleukopenia that survive the first 5 days of treatment usually recover, although recovery is often more prolonged than it is for dogs with parvoviral enteritis. In 244 cats with feline panleukopenia from Europe, the survival rate was 51%.[12] Nonsurvivors had lower leukocyte and platelet counts than survivors, and cats with white cell counts below 1000/µl were almost twice as likely to die than those with white cell counts above 2500/µl. Only total leukopenia, and not lymphopenia, was correlated with mortality. Hypoalbuminemia and hypokalemia also were associated with an increased risk of mortality.

## FELINE RESPIRATORY VIRAL DISEASE

The most common causes of feline respiratory viral disease are FHV-1 and FCV. FHV-1 is an enveloped DNA virus. It survives a maximum of 1 day at room temperature and is susceptible to destruction by common disinfectants. FCV is a nonenveloped RNA virus, which survives up to 10 days at room temperature. Inactivation requires hypochlorite solutions, concentrated accelerated hydrogen peroxide solutions, or potassium peroxymonosulfate; quaternary ammonium compounds are not effective.[13]

FHV-1 and FCV infections may be acquired by contact with acutely infected cats, contact with organisms in the environment, or by contact with carrier cats. The chance of infection is increased when large numbers of cats are housed together. Both viruses replicate mainly in the tonsils and respiratory tissues. In addition to the nasal, conjunctival, and oral shedding common to both viruses, FCV also is shed in the feces and occasionally in the urine.

Almost all cats infected with FHV-1 develop latent infections, whereby the virus persists in tissues such as the trigeminal ganglia for the life of the animal. Reactivation of virus shedding occurs in roughly 50% of infected cats, with or without concurrent clinical signs. This may occur spontaneously or after stressful events. Shedding occurs 4 to 11 days after the stress and lasts 1 to 2 weeks. In contrast, shedding of FCV by persistently infected cats is continuous and not affected by stress. In some cats, shedding is lifelong; in others, it ceases after several weeks.

Acute disease caused by FCV and FHV-1 occurs after an incubation period of 2 to 10 days. The most severe signs tend to occur in very young and elderly debilitated cats. Concurrent immunosuppressive illness or infection with other respiratory pathogens and opportunistic bacteria can influence dramatically the severity of disease. Clinical signs common to both infections include conjunctivitis, serous or mucopurulent nasal discharge and sneezing and, less commonly, coughing and dyspnea. Lethargy, anorexia, hypersalivation, and pyrexia also may be present in acute infections. FHV-1, but not FCV, may be associated with corneal ulceration and keratitis. Ulcerative glossitis is more common and severe with FCV infection but may be associated with FHV-1 infection. A small proportion of FCV carriers develop chronic lymphoplasmacytic or chronic ulceroproliferative stomatitis, which is often refractory to therapy. Transient lameness and pyrexia have been reported in association with acute FCV infection and after FCV vaccination.

Highly virulent strains of FCV have been isolated from outbreaks of severe systemic febrile illness.[14,15] This condition is characterized by a high mortality, fever, anorexia, ulcerative facial dermatitis, and diffuse cutaneous edema (Figure 96-2). Coagulopathies also can develop, along with hypoproteinemia and mild hyperbilirubinemia. The suspected or confirmed outbreaks of infection reported shared several significant features: (1) in every outbreak in which a suspected index case was identified, a hospitalized shelter cat appeared to be the source of infection, (2) otherwise healthy, adult, vaccinated cats have been affected prominently, whereas kittens tended to show less severe signs, (3) spread occurred very readily, including via fomites to cats belonging to hospital employees and clients, (4) spread of disease was limited to the affected clinic(s) or shelter, with no spread within the community reported, and (5) the outbreak resolved within approximately 2 months.[14,15]

Attempts to make a diagnosis in cases of feline respiratory viral illness are encouraged especially in catteries because knowledge of the causative organism can assist with treatment strategies. Because of the communicability and high mortality associated with virulent FCV infection, microbiologic testing is essential for cats suspected to have the systemic febrile syndrome, and suspect cats should immediately be handled as if they were infected with the organism. Infection with FCV and FHV-1 can be diagnosed using virus isolation or PCR assays from nasal, conjunctival, or oropharyngeal swabs, although oropharyngeal swabs are most likely to yield a diagnosis. For virus isolation, swabs should be transported on ice in a viral transport medium containing antibiotics to prevent bacterial overgrowth; commercial swabs are available for this purpose. PCR assays may be more reliable for diagnosis of FHV-1 than of FCV infection. However, because apparently healthy cats commonly have positive results using sensitive PCR assays for FHV-1, it may not be possible to prove an association with a particular disease.[16] Results should be interpreted carefully in light of the clinical signs present.

**FIGURE 96-2** A kitten suffering from virulent systemic calicivirus disease (FCV-Kaos strain) showing characteristic signs of facial edema and crusting and alopecia of the face and pinnae. *(From August J: Consultations in feline internal medicine, vol 5, St Louis, 2006, Elsevier.)*

Cats with severe upper respiratory tract signs and suspected or confirmed FHV-1 infection may benefit from treatment with systemic or topical antiherpesviral drugs. The most effective and safe systemic antiherpesviral drug is famciclovir. Famciclovir is a prodrug that is converted to penciclovir. The latter is a guanosine analog that inhibits the viral DNA polymerase. Famciclovir is extremely well tolerated and has been used safely in kittens as young as 12 days of age.[17] The dosage of famciclovir is 40 to 90 mg/kg PO q8h. Cats with herpetic keratitis can be treated with topical ophthalmic antivirals such as trifluridine, idoxuridine, vidarabine, or a 0.5% solution of cidofovir. Although more expensive, cidofovir has the advantage of requiring only twice daily administration, whereas idoxuridine, trifluridine, and vidarabine must be administered 5 to 6 times a day. Trifluridine is irritating and may not be well tolerated by cats.

The outbreaks of systemic febrile caliciviral disease have demonstrated the importance of control measures to limit the spread of feline respiratory viruses because of the high mortality, poor efficacy of vaccines, and lack of specific treatments. Quick recognition and implementation of effective control measures, including disinfection, quarantine, and testing procedures, are critical to reduce the impact of this disease. These have been described in detail elsewhere.[18] An adjuvanted, inactivated vaccine for virulent systemic disease that contains a single hypervirulent strain was introduced in the United States in 2007. However, the degree to which this vaccine cross-protects against other hypervirulent strains is unknown, and in every outbreak, the strain involved has differed, and the outbreak has ceased when infection control measures were implemented. Because of this and the increased risk of sarcoma formation with adjuvanted vaccines, the usefulness of the vaccine has been questioned.

## FELINE INFECTIOUS PERITONITIS

FIPV infection is caused by feline coronavirus, an enveloped RNA virus. Feline coronaviruses mutate readily, and researchers hypothesize that a relatively nonpathogenic feline coronavirus that replicates within the gastrointestinal tract (FCoV) mutates within the host to form virulent FIPV. Mutation occurs soon after infection with FCoV, or years later. Spread of FIP from cat to cat does not occur, so affected cats do not have to be isolated.

The prevalence of antibodies to feline coronavirus in single-cat households is approximately 25%, whereas in some multicat households, all cats may have positive titers. In contrast, FIP affects 1 in 5000 cats in single-cat households and approximately 5% of cats in catteries. The incidence of FIP is related to levels of virus in the environment, immunosuppression resulting from overcrowding and other stressors, and genetic factors. Purebred cats are more susceptible, and affected cats are usually 3 months to 3 years of age. Occasionally geriatric cats are affected, perhaps because of waning immune function.

Feline coronavirus is highly infectious and is spread via the fecal-to-oral route. FCoV replicates in enterocytes and destroys the villus tips, sometimes resulting in mild gastrointestinal signs. Mutation to virulent FIPV is associated with the ability to replicate within macrophages and possibly loss of the ability to replicate in enterocytes. Cats with a poor CMI response develop pyogranulomatous vasculitis because of deposition of antigen-antibody complexes within the venular epithelium. Pleural and peritoneal effusions develop (effusive FIP). Cats with a partial CMI response are able to slow replication of the virus, with subsequent granuloma formation in a variety of tissues (noneffusive FIP). This may deteriorate to effusive FIP if the CMI response wanes.

Cats with FIP often are evaluated for fever, weight loss, anorexia, and lethargy. Other signs and physical examination abnormalities may include respiratory distress resulting from pleural effusion or pneumonia, abdominal distention because of ascites, abdominal masses, icterus, splenomegaly, irregular renomegaly, anterior uveitis, retinal detachments, multifocal neurologic signs, and GI signs relating to organ failure or obstructive intestinal masses.

FIP remains an antemortem diagnostic challenge. The presence of hyperglobulinemia on the complete blood count may increase suspicion for FIP, but it is not present in all cats and may occur with other diseases. The presence of high-protein (5 to 12 g/dl), low-cellularity (predominantly neutrophils) effusion fluid is also supportive of the diagnosis. However, tests such as the serum or effusion albumin-to-globulin ratio, effusion γ-globulin concentration, and the Rivalta test can be associated with false-positive and false-negative results, especially in populations in which the prevalence of FIP is low.[19] Serologic tests that detect anti-FCoV antibody are not FIP tests. Positive test results mean only exposure to a coronavirus, and many healthy cats have positive titers but never develop FIP. In one study, titers of 1 : 1600 or greater in cats that were suspected to have FIP had a 94% chance of truly having FIP, but cats that had any coronavirus antibody titer had a 44% chance of truly having FIP.[19] However, considerable interlaboratory variation in assay results occurs, so this may not be true for serology performed at all laboratories. The same study also showed that immunocytochemistry for feline coronavirus on macrophages in effusion fluid had a specificity of 100% for diagnosis of FIP, although the sensitivity was only 57%. The mutation that occurs when FCoV becomes virulent FIPV is not predictable, and there is no way to distinguish the viruses based on nucleotide sequence. Because FCoV may be found within tissues and body fluids, false-positive results may occur when testing tissues or fluids using RT-PCR. The standard for diagnosis of FIP is detection of pyogranulomatous vasculitis on histopathologic examination of biopsy specimens, with intralesional virus antigen as detected using immunostaining techniques.

Treatment of FIP remains a challenge, and controlled studies of antiviral drug use are few. Although treatment with feline recombinant interferon-ω (1 million U/kg SC q72h until remission, then weekly thereafter) and prednisolone (1 mg/kg PO q12h then tapered to q72h) showed promise in a preliminary study,[20] in which 4 of 11 cats with effusive disease survived as long as 2 years, a subsequent placebo-controlled clinical trial showed no benefit of the treatment. Currently the only medication that appears to slow the progression of the disease in cats is prednisolone. Although most cats typically live only a few months after diagnosis, occasionally survival times of up to 2 years have been documented when the disease has been detected early.

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