Feline Viral Upper Respiratory Diseases

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Infectious upper respiratory disease is the most common and probably the most devastating respiratory syndrome recognized in cats.[1] Three infectious diseases are responsible: feline viral rhinotracheitis (FVR), feline calicivirus infection, and feline pneumonitis (*Chlamydia psittaci*). Feline Viral Rhinotracheitis and caliciviral infection account for the majority of clinical cases and will be reviewed here. These two viral diseases comprise 80 to 90% of infectious upper respiratory disease in domestic cats and are isolated in approximately equal frequency. Both affect the feline respiratory tract and conjunctival membranes and cause a variety of clinical signs in infected cats. Both FVR and feline calicivirus have a widespread distribution throughout the world; serological studies have shown specific antibodies present in 80% or greater of adult cat populations.[2]

The Agents

The etiologic agent of FVR is feline herpesvirus, which causes characteristic intranuclear inclusion bodies in infected cells. The virus is fragile and is susceptible to heat, acid and common disinfectants (e.g. hypochlorite or quaternary ammonium compounds).[4] Feline herpesvirus appears to be highly species-specific for the domestic cat.

Feline calicivirus is composed of a number of serologic variants, or strains. It is slightly hardier than FVR, but is susceptible to the same disinfectants as FVR.

Pathogenesis and Lesions

The mucosal surfaces of the upper respiratory tract and conjunctiva are predilection sites for feline herpesvirus, and viral replication takes place in the mucosa of the nose, turbinates and conjunctiva. The principal lesions in FVR are necrosis of epithelium and associated purulent inflammation.[5] The subsequent exudates may cause dyspnea and paroxysmal coughing and sneezing. Lesions do not usually extend to the bronchi, bronchioles, or alveoli. Intranuclear inclusion bodies can be demonstrated in mucosal epithelium for up to 7 days after infection.

Feline calicivirus strains vary in their virulence. Viral replication occurs in the oral and respiratory mucosa and the conjunctiva. Formerly, it was thought that the virus affected mainly the upper respiratory tract, as in FVR, but it has been more recently demonstrated that while calicivirus does affect the conjunctiva and upper airways, the lung is also an important target for the more virulent strains.

Transmission

Transmission of both viruses is most commonly by direct contact with infectious oral, nasal, or ocular secretions, or by contact with contaminated fomites. Aerosol transmission is uncommon and appears to be much less important in the spread of the disease than earlier postulated.[6,7] Route of infection may be intranasal, oral, or conjunctival.

Clinical Syndromes

Cats of any age can be affected by viral respiratory disease, but the morbidity and mortality is highest in young cats and kittens. Anorexia, fever and depression are common with both viral infections. Feline viral rhinotracheitis produces a characteristic syndrome in susceptible cats. After a 2 to 8-day incubation period, early signs include hypersalivation, sneezing, and clear, serous, ocular and nasal discharges. As the disease progresses,

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conjunctivitis and coughing may develop. Ocular and nasal secretions may become mucopurulent, especially with secondary bacterial invasion. The ocular manifestations of feline herpesvirus primarily involve the conjunctiva, but ulcerative keratitis may develop.[8] A leukocytosis is generally present throughout the course of the disease. The majority of clinical signs have usually resolved within 10 to 20 days, but some animals may be left with chronic sequelae such as persistant conjunctivitis or rhinitis. The recurrent clinical signs seen in these chronically affected cats is most often due to secondary bacterial invasion of mucosal surfaces originally damaged by the virus.[4]

In general, the disease produced by feline calicivirus is similar to but milder than FVR, although the range of clinical signs varies greatly among the various strains of the virus. The majority of strains, after a 2 to 10-day incubation period, produce a mild upper respiratory disease with ocular and nasal discharge and ulceration of the tongue, hard palate, and nostrils. In some cases mouth ulcerations may be the only clinical sign of infection. The more virulent strains of calicivirus may produce a primary interstitial pneumonia with resulting dyspnea.

Involvement of systems other than the respiratory tract can occur with FVR. Feline viral rhinotracheitis has been associated with central nervous system dysfunction and liver disease, but only in very young, old, or immunosuppressed animals.[13] Feline herpesvirus has also been associated with ulcerations of the skin.[9]

The Carrier State

As stated previously, both feline respiratory viruses are relatively fragile, and thus they must rely for their continued survival on their ability to persist in the cat. In both FVR and feline calicivirus infections most of the clinical signs regress over a period of 10–12 days. After this time any animal which remains persistently infected with the virus is termed a "carrier." The carrier animal may be asymptomatic or show chronic symptoms of the disease. The carrier state of FVR is characterized by latent periods with only intermittent episodes of virus shedding. During such episodes the carrier is highly infectious to other cats.[4] Carriers may shed spontaneously or subsequent to stresses such as corticosteroid therapy, general anesthesia, cat shows, etc. An estimate of up to 80% of FVR-recovered cats remain carriers for a variable period of time.[4] Cats may spontaneously clear the virus after months or years, but some animals remain carriers for life.

Unlike FVR, carrier cats of feline calicivirus excrete virus more or less continuously from the orophrynx. Such animals are therefore a constant hazard to susceptible cats. In a survey of 1500 clinically healthy cats, sampled with a single oro-pharyngeal swab, feline calicivirus was found to be widespread; 8% of single household pets, 24% of cats attending cat shows and 41.5% of cats in institutional colonies were found to be excreting the virus.[12]

Diagnosis

Differentiation of FVR and feline calicivirus infection on the basis of clinical signs alone is difficult and generally not recommended. Clinical differentiation is possible based on the principle that while some manifestations (e.g. fever, depression, anorexia) are common to both diseases, others signs are more frequently associated with one infection or the other. Rhinitis and conjunctivitis with serous to mucopurulent discharges, keratitis, blepharospasm, sneezing, coughing, hypersalivation and oral respiration are signs most suggestive of FVR, while ulcers of the tongue, hard palate and nostrils, occasionally associated with signs of pneumonia are more consistent with feline calicivirus. Unfortunately, differentiation of the two viral infections is not always as simple as descriptions suggest, due to overlap and variation in clinical signs and severity of infection and complications with secondary infections. Therefore, definitive diagnosis is best made serologically or by virus isolation. However, because treatment of both viral diseases is symptomatic in the majority of clinical cases, etiologic diagnosis of upper respiratory viral disease in cats is generally not necessary or economically warranted.

Treatment

The intensity of treatment of viral respiratory disease is dependent on the severity of clinical signs. In some cases clinical manifestations are so mild and the cat so relatively unaffected that no treatment is required.[10] In the case of the more severely affected cat,

however, prompt and vigorous therapy is necessary. Therapy is for the most part symptomatic and supportive. Good nursing care is extremely imporant in the treatment of these cats, as respiratory arrest secondary to airway obstruction and/or pneumonia, dehydration, and malnutrition are the usual causes of death.[1]

Affected cats should be kept in a clean, warm, well-ventilated environment and discharges from the eyes and nose should be cleaned away frequently. Antibiotics should be given for a minimum of 7 days to help control secondary bacterial infection. Good choices include ampicillin, hetacin K, or chloramphenicol. In more severely affected animals in which dehydration becomes a problem, fluid therapy may be necessary. Subcutaneous lactated Ringer's solution or 2½% dextrose diluted 50:50 with saline is often adequate, but some cats may require intravenous fluids.

Cats should be encouraged to eat by offering strongly flavored odorous foods (e.g. sardines, tuna). Soft or semi-liquid foods, such as strained-meat baby food may be more palatable to those animals in which painful oral ulceration has developed. The placement of a pharyngostomy tube may become necessary in cats that have been anorectic for several days. This conveniently allows replacement of both calories and fluids with a minimum of stress to the cat and is far superior to repeated attempts at force-feeding or stomach tubing.

Warm humidification or nebulization is often beneficial in relieving airway obstruction. Acetylcysteine 20% diluted 1:8 with saline may be added to the nebulization formula as a mucolytic if secretions are viscous and inspissated. Additionally, 0.25% phenylephrine or 0.025% oxymethazoline will provide temporary relief of nasal congestion when used judiciously.[11]

Vitamin therapy including vitamins A (10,000 IU daily) and B-complex are indicated in the severely ill cat. Vitamin C may also be given intravenously at a rate of 1 gram daily.

Ocular therapy is critical to avoid corneal damage and panophthalmitis, especially in FVR. Broad spectrum antibiotic ophthalmic ointment should be started promptly, and corticosteroids should be avoided due to the potential for corneal ulceration to develop during the course of the infection.

The chronic rhinitis and/or sinusitis that is sometimes a sequelae to viral upper respiratory disease presents a therapeutic challenge for the veterinarian. The more chronic the condition becomes, the more unlikely it is that conservative therapy will help.[10] The majority of these animals are suffering from persistent or recurrent secondary bacterial infections, and prolonged (2–3 week) antibiotic therapy following culture and sensitivity may be beneficial. Unfortunately, the improvement seen is often temporary and ultimately surgical procedures, including trephining the frontal sinus, may be necessary. Some chronically affected cats show improvement when housed outside.[4]

Prevention and Control

The prevention and control of feline viral upper respiratory disease has two components: immunoprophylaxis via a vaccination program, and management practices aimed at preventing transmission of the viruses. The development of effective vaccines against FVR and feline calicivirus is potentially the most effective means of controlling the diseases.[10] Inactivated and MLV parenteral vaccines and MLV intranasal vaccines are available. The parenteral vaccines are also available in combination with feline panleukopenia MLV or inactivated vaccine, and have been extensively tested for safety and efficacy.[13,14] The parenteral vaccines evoke adequate serum antibodies. The intranasal vaccines also appear to be safe and efficacious, but do show an increased number of minor side reactions, particularly transient sneezing 1 to 16 days post-vaccination. Less common reactions include a raw nose, transient anorexia and lacrimation. Cats recover from these reactions uneventfully.[17] A single dose of intranasal vaccine produces significant protection for at least one year. Kittens may be vaccinated at 8 to 10 weeks of age with yearly revaccination.

Proper management practices can aid greatly in the prevention of transmission of feline viral respiratory disease. The greatest management challenge exists in veterinary hospitals and catteries. Within a veterinary facility adequate ventilation is probably the most effective means of disease prevention. Twelve air exchanges per hour are desirable.[10] Fomite transmission can be minimized by use

of disposable litter trays and feeding dishes and by thorough cleaning of cages with approved viricidal disinfectants. People handling potentially infectious cats should change outer smocks and wash their hands thoroughly afterwards.

In the cattery, in addition to vaccination, isolation becomes an important aspect of disease control.[10] Animals entering the colony following purchase, a cat show, or breeding should be quarantined for three weeks.[4] Animals in the colony can be screened virologically and serologically in an attempt to identify carriers, although because of the high prevalence of the diseases, elimination of carriers is seldom practical. Kittens may be weaned early (four to five weeks of age) before maternal antibody levels have declined and vaccinated immediately to maximize protection.

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