FELINE VIRAL RESPIRATORY DISEASE (URD)

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Respiratory disease has always been a major problem in feline medicine. The syndrome is not only troublesome for the individual pet, but perhaps more particularly for those animals which are brought together for breeding, boarding and showing.

Aetiology

The two major causes of respiratory disease in cats are two viruses; feline viral rhinotracheitis (FVR) virus - felid herpesvirus 1 - and feline calicivirus (FCV). Both viruses are widespread throughout the world and are probably of equal importance in causing the disease. Together they probably account for at least 80% of cases, (Kahn and Hoover, 1976). The feline strain of Chlamydia psittaci can also cause mild respiratory signs, but in general the major feature of C. psittaci infection is a marked persistent conjunctivitis, (see later). Feline reovirus has been shown to produce a mild, predominantly conjunctival disease experimentally (Scott et al., 1970), but it is probably not a very significant cause of URD in the field.

Bacteria, such as staphylococci, β-haemolytic streptococci, Pasteurella spp. and coliforms, and mycoplasmas, have also been implicated in the disease, though their main importance is probably as secondary invaders following primary viral damage. Nevertheless, a more primary role for mycoplasmas has been suggested by some (see later). The importance of Bordetella bronchiseptica, which has been detected in some laboratory colonies, has yet to be determined.

Clinical signs

These have been reviewed in more detail elsewhere (Kahn & Hoover, 1976; Gaskell & Wardley, 1978; Gaskell, 1985). FVR is generally a severe disease characterised by pyrexia, sneezing, hypersalivation, conjunctivitis, and marked ocular and nasal discharges. More rarely, ulcerative keratitis, lingual ulceration, lower respiratory tract involvement or virus generalisation may also occur.

Feline caliciviral disease (FCD) is typically milder than FVR. General malaise is less common, discharges are usually less copious, and ulceration of the tongue, hard palate or external nares is often a characteristic feature. Indeed, such ulceration may occur unaccompanied by upper respiratory or ocular signs. However, there are a large number of strains of feline calicivirus in comparison with the single strain of FVR, and these do vary slightly in pathogenicity. Strains have also been described that produce only a primary interstitial pneumonia (Kahn & Hoover, 1976); in some the characteristic signs are pyrexia, muscle pain and limping (Studdert et al., 1970; Pedersen et al., 1983); and in some the resulting infection is only subclinical.

Diagnosis

Many cases of URD can be diagnosed on the characteristic clinical signs, as described above. If a more definitive diagnosis is required,

perhaps because more specific advice on control measures is needed or because antiviral or chlamydial chemotherapy is being contemplated, then laboratory diagnosis may be attempted from oro-pharyngeal or conjunctival swabs sent in the appropriate transport medium.

In intractable cases where the problem is secondary bacterial infection, bacterial culture and sensitivity tests should be performed.

Treatment

This has been reviewed elsewhere (Gaskell & Gaskell, 1980; Gaskell, 1985; Gaskell & Wills, 1985). Briefly, no anti-viral drugs are at present in routine use for the treatment of viral respiratory disease in cats although 5-iododeoxyuridine has been used in cases of ulcerative keratitis in FVR. There are however anti-herpesvirus drugs such as acyclovir and other synthetic nucleosides that have been developed for use in man, and some of these may ultimately prove useful in treating herpesvirus infections in cats. In general, however, treatment for viral respiratory disease in cats is supportive, relying on nursing care, fluid therapy, and antibiotic treatment to control secondary bacterial infection. Antibiotics should be given for at least 7 days, and animals routinely re-examined after one week to ensure signs are resolving: adequate therapy in the acute stage against secondary bacterial infection reduces the possibility of chronic sequelae. In severe prolonged cases of anorexia, dehydration and debility, a pharyngostomy tube may be indicated (Lane, 1977). Where chlaymydial infection is present, topical or systemic tetracyclines are indicated, treatment being continued for at least two weeks after clinical signs have disappeared (Wills, 1983; Wills & Gaskell, 1985), (see later).

Epidemiology

Both feline calicivirus and FVR virus are highly successful pathogens, although infection is generally more common in colony cats compared to individual household pets. Thus the disease appears mainly in boarding catteries, breeding colonies, stray cat homes or other situations where a large number of cats have been brought together.

The feline respiratory viruses persist in such populations in three ways. Firstly, they persist by passing directly from acutely infected to susceptible cats: during the acute stage of the disease, infectious virus is usually present in secretions for 1-3 weeks. Secondly, they can persist in the environment so indirect transmission may occur via infected secretions on cages, feed bowls, and personnel. However the feline respiratory viruses are relatively fragile outside the cat (FVR virus survives for up to a day or so at the most, feline calicivirus up to 8-10 days) and so this is probably only of importance in the short term, within the close confines of a cattery. Aerosol transmission is not thought to be of major significance though sneezed macrodroplets in still air may travel over short distances. Finally, the viruses may persist in the cat itself in a carrier state, after it has recovered from the acute phase of the disease. Such carriers are widespread in the population and are undoubtedly of importance as a source of virus, although slightly longer and more intimate contact is probably necessary to achieve transmission than in the acute infection. There are no known reservoir or alternative hosts for the viruses.

Since carriers are an important source of virus in the cat population, the features of each virus carrier state will be described more fully.

The FVR carrier state (figure 1) is characterised by a latent phase with only intermittent episodes of virus shedding in oro-nasal and conjunctival secretions. At least 80% of FVR-recovered cats appear to be virus carriers, approximately half of which are likely to be epidemiologically important, that is, shed virus under natural conditions (Gaskell & Povey, 1977). Shedding is most likely to occur following a "stress", for example, corticosteroid treatment, or a change of housing (boarding kennels, going to stud, cat shows, etc.). There is a lag period before the onset of shedding of approximately one week. Animals then shed virus for up to two weeks. Therefore a carrier cat is likely to be infectious after a stress for about 3 weeks. During re-excretion episodes the shedding carrier may show mild clinical signs of URD.

Unlike FVR virus, FCV is excreted more or less continuously by carrier animals, and a carrier has been defined as a cat that excretes FCV more or less continuously for at least 30 days after the acute infection (figure 2). Although the FCV carrier state appears to be self-limiting in many cases, in others it may be life-long. FCV carriers may be arbitrarily divided into 3 groups: high, medium and low-level excretors, depending on the mean levels of virus shed over a long period (Wardley, 1976). High-level excretors are much easier to detect with a single oro-pharyngeal swab and are highly infectious to susceptible cats. A low-level excretor however, may need a series of swabs to be detected and is not so infectious. A survey of 1500 clinically healthy cats in the UK before vaccination showed that FCV carriers were widespread: at least 40% colony cats, 25% cats at shows and 8% of single household pets were found to be shedding the virus.

Thus in breeding or boarding catteries, infection is often introduced by the clinically normal carrier. Once endemic, the disease is generally seen in the acute form in young kittens at the stage at which they lose their passive immunity. In older cats its presence may be noted by the existence of chronically affected cats with persistent or recurrent rhinitis, sinusitis or conjunctivitis.

Prevention and control

The prevention and control of feline URD should be approached through a combination of management and vaccination. A number of vaccines (modified live and killed systemic, and modified live intranasal) are available for the protection of cats against the feline respiratory viruses (reviewed by Gaskell, 1981).

Generally the respiratory virus vaccines are relatively successful in preventing URD with few side effects in the majority of healthy, previously unexposed animals. Ideally vaccination of the whole cat population should be the aim. Nevertheless, because of the nature of the epidemiology of the disease, vaccine reactions and breakdowns may occur from time to time (see below) and management procedures are still necessary in many cases to help control the disease..

URD should be controlled in the individual animal by annual vaccination and by protecting the cat as far as possible from contact with

the virus: household pets are more likely to meet the virus in a cattery than in neighbouring household cats since cats are mainly territorial animals and the duration of contact between them is relatively short. Extra, booster vaccinations should be given if social contact and stress situations (e.g. boarding catteries, veterinary hospitals, or going to stud) are unavoidable. Boarding cattery owners should insist on recent vaccination but should not rely solely on this for URT disease control, for virus will inevitably be present in the cattery either from the occasional animal incubating the disease, or more likely, from carriers. Thus measures should still be taken to prevent any possible cross-infection and reduce the concentration of virus in the environment. These procedures are outlined in table 1: they will also generally inhibit the spread of other pathogens.

In stray cat homes in general the same measures apply, but it is often impossible to separate animals to the same extent. Nevertheless incoming cats should be batched and quarantined as far as possible; and those with clinical signs segregated. Unless animals can be isolated on arrival for four weeks, the systemic vaccines will not have time to become effective and in these circumstances it may be advisable to use the intranasal route, where protection has been shown to develop within four days (Cocker et al. 1984).

In virus-free breeding colonies, all cats should be vaccinated routinely if there is any contact at all with other stock. Care should be taken in administration, if a modified live vaccine is used. Great care should also be taken to avoid buying in carriers, remembering that vaccinated animals may still be a source of infection. New breeding stock should be from a respiratory virus free colony, and all incoming stock should be quarantined for three weeks and ideally screened virologically and, if unvaccinated, serologically, in an attempt to detect possible carriers.

In breeding colonies where the disease is already endemic, breeding queens should be regularly vaccinated with additional boosters before mating, or during pregnancy if with a killed vaccine. Queens should be moved into isolation to kitten at least three weeks before term, to keep the kittens away from any carrier animals in the colony. With particular queens with a history of disease in their kittens, it may be advisable to early wean (ideally at 4 - 5 weeks) her kittens into isolation, or alternatively avoid the use of that queen for breeding. All kittens should be vaccinated as soon as maternal antibodies are at a non-interfering level (normally 9 - 10 weeks) and certainly before exposure to adults. In some situations earlier vaccination schedules may be initiated (e.g. 3, 6 and 9 weeks of age). Although not licensed for use in the U.K. in kittens less than 12 weeks of age, the early use of the intranasal route (e.g. at one week and 3 - 4 weeks) may be useful because of the lack of interference from maternally-derived antibody.

Vaccine reactions and breakdowns

Possible reasons for this are discussed in more detail elsewhere (Gaskell, 1980; 1985), but the major points are listed below.

(i) Vaccine reactions

1. The most likely cause is that the animal is actually incubating the disease already: vaccination programmes are of course generally implemented in kittens just when their maternal antibody has waned.

2. The cat may already be a field virus carrier, and although the effect of vaccinating carriers is not clear, they are undoubtedly still of importance both to themselves and others as a source of infectious virus. It is also possible that for FVR, the mild "stress" of vaccination and the attendant disruption of routine, may initiate an episode of virus shedding.

3. Ideally, vaccine virus should be attenuated so that it does not itself cause disease. Nevertheless there may be one or two individuals in whom this is not so. This may be due to individual idiosyncrasy, to intercurrent disease (e.g. with feline leukaemia virus or panleucopenia virus), or perhaps to variations in microbial flora. However it does also seem that even in apparently normal cats, modified live vaccines given by the intransal route may cause mild signs of disease. In contrast, live systemic vaccines should be safe if administered properly, but there have been suggestions that if they are inadvertently given by the wrong route (e.g. by making an aerosol with a syringe or the cat licking the injection site), then signs of respiratory disease may develop (Povey, 1977).

(ii) Vaccine breakdowns

1. Firstly it should be emphasised that although the feline URD vaccines are generally effective in the majority of cats, even under ideal conditions protection is not necessarily entirely complete in all animals.

2. Factors which may adversely affect an individual's immune response include intercurrent disease; overwhelming challenge dose of virus; maternal antibody interference with the initial vaccination programme; infection with other respiratory pathogens (e.g. chlamydia) or possibly a variant strain of calicivirus.

3. In colonies with endemic disease, breakdowns may occur relatively commonly. There are two main reasons for this. The first is the variable and sometimes only short duration of passive antibody (particularly with FVR) which often leads to outbreaks of disease in young kittens. The second reason is that carriers are widespread in such colonies and are a continual source of virus. It is important to note also that although vaccinated animals are generally free from disease, they are not necessarily free from infection: such cats may already have been carriers before vaccination, or they may have become infected subclinically afterwards. Clearly such carriers may be of considerable epidemiological importance, particularly in colonies where the close contact encourages transmission.

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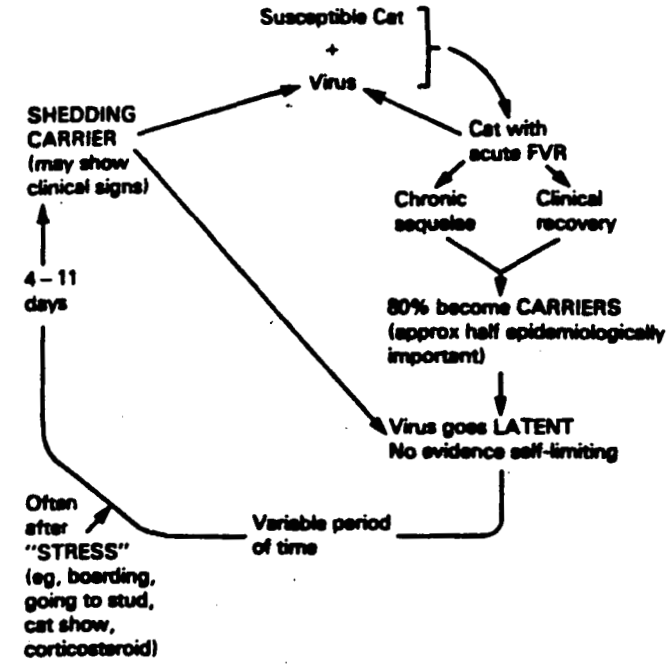
Table 1

Recommendations to prevent spread of the respiratory viruses in a boarding cattery.

1. Make sure all incoming cats are fully vaccinated.
2. House cats individually, unless from same household.
3. Build cattery with solid partitions between pens. Ensure frontages are at least 1.5m apart, and the surface of the pen is easily washable.
4. Arrange pen so food bowl and litter tray may be removed routinely without entering the pen, i.e. do not handle cats more than necessary.
5. Either wash hands in disinfectant bucket between visiting each pen; or have an individual pair of rubber gloves on a peg by each pen for use only with that pen. Disinfect thoroughly becore use with a new boarder.
6. Wear rubber boots and, if it is necessary to enter the pen, step into a disinfectant bath.
7. Either: use disposable food trays; or have two sets of feed bowls used on alternate days. Soak used set in 1 in 32 bleach/detergent solution for several hours, and then leave thoroughly rinsed and dried until re-use 24 hours later.
8. Prepare food in central area.
9. Replace badly soiled litter trays with another previously disinfected and pre-filled in a central area, i.e. a similar system to the feed bowls.
10. When cat goes home, thoroughly disinfect cage, allow to dry, and preferably leave empty for 2 days before re-using.
11. Put cats with any signs of a previous respiratory infection e.g. ocular discharge, chronic rhinitis), cats known to have had respiratory disease, and any suspect carrier cats from past experience, in one section, or at one end of the cattery, and feed last.
12. Feed cats in same order every day and attend to each pen completely before moving onto the next.
13. Reduce concentration of virus in environment by adequate ventilation, low relative humidity, and optimum environmental temperature.

(FVR carrier state: Epidemiology) Fig. 1



(FCD carrier state: Epidemiology) Fig. 2