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Review

Feline parvovirus infection and associated diseases

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ABSTRACT

Feline panleukopenia, caused by the single-stranded DNA virus feline parvovirus (FPV), is a highly contagious and often lethal disease of cats and other Felidae. FPV, but also canine parvovirus (CPV) can be isolated from both healthy and diseased cats. In Germany, CPV was detected in only approximately 10% of feline samples, but in Southeast Asia, reports estimated that up to approximately 80% of diseased cats were infected with CPV. Infection spreads rapidly, especially in cells with high mitotic activity, such as bone marrow, lymphoid tissue and intestinal crypt cells. Anorexia, vomiting, diarrhoea, neutropenia and lymphopenia are common in clinically affected cases. In utero or neonatal infection can result in cerebellar hypoplasia. Depending on the severity of clinical signs, mortality ranges from 25 to 100%. Effective vaccination and thorough disinfection are of the utmost importance in the prevention of disease transmission in multi-cat households and animal shelters. If clinical signs develop, supportive treatment should be commenced. The efficacy of feline recombinant interferon and FPV antibodies has not been clearly demonstrated. Commercially available vaccines should induce protective immunity when administered according to current guidelines. Recent studies suggest that in some kittens, maternally derived antibodies (MDA) can persist for much longer than has been previously recognised. FPV serum antibody tests are available, but protection status needs to be interpreted with caution in kittens with MDA and a negative titre in adult cats does not necessarily denote lack of protection.

Introduction

Feline panleukopenia virus (FPV) is a small, non-enveloped single-stranded DNA virus that infects domestic cats and other *Felidae* as well as species of the families *Mustelidae*, *Procyonidae*, and *Viverridae* (including raccoons, ring-tailed cats, foxes and minks). The virus causes feline panleukopenia, a disease characterised by severe reduction in circulating white blood cell count and enteritis with degeneration of the intestinal villi. Infection is highly contagious and is associated with high mortality and morbidity (Barker et al., 1983; Scott, 1987; Steinel et al., 2001), as very high concentrations of virus are shed from infected animals (up to 10^9 median tissue culture infective dose [TCID50]/g faeces).

Aetiology

Current taxonomy defines FPV and canine parvoviruses (CPVs) as one single taxonomic entity (Tattersall, 2006). In the late 1970s, CPVs evolved from FPV after crossing species barriers by acquiring five or six amino acid changes in the capsid protein gene (Truyen, 1999). Within 1 year, the first CPV (CPV-2) changed to the current subtypes, CPV-2a- and CPV-2a-derived strains. Whereas CPV-2 was not able to infect cats, the subtypes can cause clinical signs that cannot be distinguished from those caused by FPV (Truyen et al., 1995, 1996; Mochizuki et al., 1996).

The prevalence of CPV in cats with panleukopenia is not known. One study of cats with panleukopenia in Vietnam and Taiwan reported that CPV-2a- and CPV-2a-derived strains were isolated in up to 80% of diseased cats (Ikeda et al., 2000). In contrast, feline infections with CPV-2a- and CPV-2a-derived strains seem to be rare in Europe and the USA. However, CPVs are found sporadically in feline diagnostic material. In one German study, about 10% of the viruses isolated from cats with naturally occurring panleukopenia were CPV-2a- or a CPV-2a-derived strain (Truyen et al., 1996).

In the UK, CPVs were identified in 33% of faecal samples from clinically healthy cats in a cross sectional study of 50 cats in a feline-only shelter, and in 34% of faecal samples in a longitudinal study of 74 cats in a mixed canine and feline shelter (Clegg et al., 2012). Although many cats were shedding CPVs, clinical panleukopenia was not diagnosed, and canine faecal samples from the mixed kennel were negative for CPVs. Longitudinal sampling in one shelter showed that all cats shed the same virus strain each time, despite the lack of clinical signs. Fifty percent of the sequences of those feline strains were similar to those obtained from clinically ill dogs in the UK (Clegg et al., 2011).

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It is thought that clinically healthy cats can shed FPV as well as CPVs for prolonged periods of time, making them an important reservoir of infection for all carnivores. Recently, a case of co-infection with CPV-2a and FPV was described in a 3-month-old kitten in Italy with a parvovirus variant that contained FPV- and CPV-2a-specific epitopes. This variant was considered an intermediate between CPVs and FPV (Battilani et al., 2013).

Pathogenesis

Infection in cats older than 6 weeks

FPV is transmitted by the faecal-oral route, and is primarily spread through contact with infected body fluids, faeces, or other fomites, as well as by fleas. FPV is highly resistant in the environment and can survive at least up to 1 year in infected organic material (Poole, 1972). Fomite transmission is important and it is possible for cat owners to carry the highly contagious virus into the house on their hands, shoes or clothing, potentially infecting cats housed entirely indoors without access to other cats (Scott, 1987).

From 18–24 h after intranasal or oral infection, FPV initially replicates in the oropharynx, followed by viremia after 2–7 days, which distributes the virus throughout the body. All 'autonomous' parvoviruses require cellular DNA polymerases that synthesise a complementary DNA strand. FPV requires rapidly multiplying cells in the S-phase of division for its replication. Thus, viral replication primarily occurs in mitotically active tissues; lymphoid tissue, bone marrow and intestinal mucosa are most frequently affected in cats older than 6 weeks of age (Csiza et al., 1971b, 1971c; Parker et al., 2001).

By infecting lymphoid tissues, FPV causes immunosuppression through cellular depletion. Lymphopenia does not only arise directly as a result of lymphocytolysis, but also indirectly following lymphocyte migration into tissues. In the bone marrow, viral replication occurs in early progenitor cells, explaining the dramatic effect on virtually all myeloid cell populations (Parrish, 1995). This is also reflected by the eponymous panleukopenia in FPV-infected cats. FPV also damages rapidly replicating cells in the crypts of the intestinal mucosa, while the nondividing absorptive cells on the tips of the villi remain unaffected. The destruction of the crypt cells leads to damaged intestinal villi and in clinically affected cases, this eventually results in diarrhoea caused by malabsorption and increased permeability. Viral DNA can persist for long periods after infectious virus has been lost; thus, detection of DNA does not necessarily signify an active infection.

Foetal and neonatal infection

In utero infection in early pregnancy can result in foetal death, resorption, abortion, and mummified fetuses. In later pregnancy, FPV can cause damage to the neuronal tissue. Within an affected litter, some kittens can be clinically healthy, probably due to their innate resistance or the acquisition of maternally derived antibodies (MDAs). Still, these kittens can harbour the virus for up to 2 months after birth (Csiza et al., 1971a).

In late prenatal and early neonatal infection, the central nervous system (CNS), including the cerebrum, cerebellum, retina, and optic nerves, can be affected. Cerebellar damage resulting in cerebellar hypoplasia (Aeffner et al., 2006) has been frequently described, as the cerebellum develops during late gestation and early kittenhood. Thus, infections occurring up to 9 days of age can affect the cerebellum, interfering with cerebellar cortical development and resulting in reduced and distorted cell layers. FPV DNA has been detected by polymerase chain reaction (PCR) in the cerebellar tissue of affected cats (Resibois et al., 2007). Although neurons are considered to be terminally differentiated, parvoviruses seem to be able to replicate in these cells. One study detected parvovirus histochemically in the brains of adult cats that died of various diseases, including panleukopenia (Van Vuuren et al., 2000), but the clinical significance of this finding is not clear.

In dogs, a relationship between myocarditis and parvovirus infection has been described subsequent to neonatal infection and several studies have detected parvovirus-like particles in puppies affected by myocarditis (Gagnon et al., 1980; Van den Ingh et al., 1980). In cats, parvovirus-related myocarditis has not been proven, and the myocardium has not been shown to be a site of replication in neonatal kittens infected with FPV. However, FPV or FPV DNA has been identified in a significant number of adult cats that had died from cardiomyopathy (Miyazawa et al., 1999; Meurs et al., 2000). Thus, FPV might also play a role in the pathogenesis of cardiac disease in cats, but this theory remains unproven.

Clinical signs

Infection in cats older than 6 weeks

Not all cats infected with FPV develop clinical signs and the severity depends on age, immune status, and concurrent infections (Foley et al., 1999); the outcome ranges from subclinical to peracute infections with sudden death within 12 h. The most common is the acute form, which initially has non-specific signs, such as fever, depression, and anorexia (Addie et al., 1996). Vomiting unrelated to eating occurs commonly and, less often, cats develop watery to haemorrhagic diarrhoea later in the course of disease. Some cats show extreme dehydration, which, when combined with anorexia, vomiting and diarrhoea, can lead to progressive weakness and depression.

Cats typically die of complications associated with septicaemia, dehydration, and disseminated intravascular coagulopathy (DIC). In shelter cats with panleukopenia, the most commonly observed clinical signs in cats that survived FPV infection were anorexia, dehydration, fever, and diarrhoea. In cats with fatal infections, death was preceded by clinical signs of circulating shock (Litster and Benjanirut, 2013). If infected cats survive for longer than 5 days, they usually recover within days or weeks.

Foetal and neonatal infection

In new-born kittens, the main clinical signs of FPV infection are neurological, with ataxia, hypermetric movements and blindness predominating. In addition, there can be signs of cerebellar dysfunction, such as incoordination, or tremor with normal mental status, which is not progressive. Forebrain damage is much less common, and affected kittens present with seizures, behavioural changes, and normal gait despite postural reaction deficits. The severity of disease and the neurological signs present can vary among littermates (Czisa et al., 1971b).

One recent study reported that housing litters of kittens with their mother was not associated with an improved outcome in shelter cats with panleukopenia. This could be because if a queen becomes infected, she did not have a protective antibody titre and therefore is unlikely to provide her kittens with adequate MDA to protect them from the FPV that she is shedding (Litster and Benjanirut, 2013). Cats with mild cerebellar dysfunction can often learn to compensate and retain good quality of life despite residual deficits. FPV can also cause retinal degeneration with or without neurological signs in infected kittens (Percy et al., 1975).

Diagnosis

Early detection of FPV with accurate testing methods is very important to identify infected cats and to prevent disease transmission. Point of care tests for the detection of faecal CPV-2a-, CPV-2a-derived strains and/or FPV antigen are available. These tests are based on either enzyme-linked immunosorbent assays (ELISA) or immunochromatographic technology. The close structural and an-