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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-

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tigenic relationship between FPV and CPVs (Mochizuki and Akaboshi, 1988; Parrish, 1991) offers the potential to test cats for FPV (as well as CPV-2a- and CPV-2a-derived strains) using canine test kits. One study compared several point of care kits by testing 200 faecal samples from healthy cats ($n = 148$) and cats with diarrhoea ($n = 52$), using electron microscopy as the reference method (Neuerer et al., 2008). All tests had an acceptable sensitivity and specificity when compared with the reference method and were considered suitable as a screening tool for faecal parvovirus shedding in cats. Cats can test positive for up to 3 weeks after modified live (ML) vaccination, although detection of vaccine antigen on point of care kits is unusual and depends on the brand of test used (Neuerer et al., 2008).

In-house tests can be confirmed by PCR testing of faeces, blood, or infected tissues (Schunck et al., 1995; Schatzberg et al., 2003). Testing of whole blood is recommended in cats without diarrhoea or when no faecal samples are available (Ryser-Degiorgis et al., 2005). However, if a point of care test for faecal antigen test is positive (and the cat has not been vaccinated in the last 3 weeks), parvovirus infection can be considered confirmed. If the test is negative and the cat shows typical signs of panleukopenia, a PCR test should be performed on faeces or blood. In dogs, the sensitivity of in-house faecal CPV antigen tests is relatively poor when compared to faecal PCR, but there are no studies in cats comparing point of care faecal tests with PCR methods (Schmitz et al., 2009; Proksch et al., 2013).

Measurement of serum FPV antibody concentration is not useful for diagnosis because many cats have antibodies due to vaccination or previous subclinical infection. Cats that are presented with panleukopenia usually have negative or low antibody titres because humoral immunity provides good protection against FPV infection (Scott and Geissinger, 1999; Lappin et al., 2002).

Prognosis

Mortality is 25%–90% in cats with the acute form of the disease and up to 100% in peracute infections. However, the prevalence of subclinical infections is unknown. Kittens up to 12 months of age were once considered to have the highest morbidity and mortality (Addie et al., 1998; Cave et al., 2002). However, one German study reported no significant correlations between age and severity of clinical signs or outcome in 244 cats with panleukopenia (Kruse et al., 2010). In that study, 57% of cats were younger than 6 months of age, suggesting that young cats could be more susceptible to feline panleukopenia than older cats, but do not have an increased risk of death. No significant correlation was found between outcome and living conditions, vaccination status (unvaccinated vs. one or more vaccines administered) or severity of clinical signs, but leukocyte and thrombocyte counts, as well as albumin and potassium serum concentrations at the time of presentation, were prognostic markers.

Treatment and management

Cats diagnosed with feline panleukopenia should be hospitalised and kept in isolation for at least 2 weeks to avoid viral transmission. Intensive care and strict hygiene to prevent fomite transmission are essential (Lamm and Rezabek, 2008).

Disinfection

Due to the extreme stability of FPV, contaminated cages, litter trays, food dishes, water bowls, shoes and clothing can play a role in transmission, and proper sanitation is of utmost importance. FPV is resistant to many commonly used disinfectants, but is inactivated by products containing potassium peroxymonosulfate, accelerated hydrogen peroxide, peracetic acid, formaldehyde, sodium hypochlorite, or sodium hydroxide, provided thorough cleaning is performed first to remove organic matter and recommended contact times are observed. Sodium hypochlorite (household bleach, 1:30 dilution) can be used on surfaces such as litter trays, and formaldehyde gas can be used for room disinfection (Truyen et al., 2009).

Supportive therapy

Supportive therapy and good nursing care can decrease mortality in cats with feline panleukopenia (Truyen et al., 2009). As the gastrointestinal barrier is often destroyed in FPV-infected cats, intestinal bacteria can invade the blood stream and bacteraemia combined with neutropenia can lead to sepsis in these immunocompromised animals. Thus, prevention of sepsis is essential for all cases, and a broad-spectrum antibiotic with proven efficacy against Gram-negative and anaerobic bacteria is recommended. The combination of choice in cats with panleukopenia is amoxicillin/clavulanic acid used with a third generation cephalosporin. Due to renal toxicity, gentamycin should only be used in well hydrated cats. A broad spectrum can also be provided by combining amoxicillin/clavulanic acid with fluoroquinolones, but fluoroquinolones, with the exception of pradofloxacin, have been associated with retinal toxicity in cats and should be avoided (Gelatt et al., 2001). Antibiotics should be administered parenterally (preferably IV), but pradofloxacin is not currently available for parenteral use.

Antiemetics might be required to control persistent vomiting. Maropitant has proven efficacy in cats. Gastrointestinal protectants are also commonly administered. The use of anticholinergic medications is not indicated as they can produce sustained intestinal ileus and can lead to intussusception (Trepanier, 2010).

Oral caloric intake and water should only be withheld while cats are vomiting and should recommence as soon as possible, starting with frequent feeding of small amounts. The beneficial effects of early enteral nutrition have been reported in canine CPV infection (Mohr et al., 2003). A highly digestible diet is preferred for cats recovering from feline panleukopenia, but if the cat does not accept it, any diet is better than no food intake at all. Semi-moist foods with low fibre content could help firm the faeces of cats with diarrhoea.

Parenteral fluid therapy to restore hydration, electrolyte and acid–base balance is most important in supportive treatment. This is preferably administered IV as a continuous rate infusion. Vitamin B complex can be added to prevent thiamine deficiency, but this complication occurs infrequently. Hypoproteinaemic cats sometimes require plasma or whole blood transfusions to improve oncotic pressure. Plasma transfusions in combination with heparin can control DIC, as they supplement anti-thrombin III and other important plasma proteins. In anorectic cats, those with severe vomiting and/or diarrhoea, or with persistent hypoproteinaemia, parenteral nutrition is required, preferably via a central venous catheter in the jugular vein (Hartmann and Hein, 2002). Glucocorticoids should not be administered because of their immunosuppressive effects.

Antiviral therapy

Feline recombinant interferon-ω (feIFN-ω) has been successfully used to treat CPV infection in dogs in experimental studies and field trials. In one study, treatment reduced mortality of CPV infection by about fivefold (Esfandiari and Klingeborn, 2000). FeIFN-ω also inhibits replication of FPV in cell culture (Mochizuki et al., 1994; Miyazawa et al., 1999; Martin et al., 2002; De Mari et al., 2003), but the efficacy of feIFN-ω as a treatment for feline panleukopenia has not been proven.

To investigate a possible prophylactic effect, FeIFN-ω was administered to cats in a cattery before an outbreak of panleukopenia (Paltrinieri et al., 2007). Twenty-three kittens were injected with feIFN-ω (1 MU/kg SC, once daily for 3 days) and survival and blood parameters were compared with those of 17 untreated cats, but no significant difference in survival between the groups was found. Treated kittens had lower levels of α1-globulins and higher values

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Table 1
Recommendations for primary and booster vaccination against feline parvovirus (Truyen et al., 2009; Day et al., 2010; Scherk et al., 2013).

Primary vaccination		Booster vaccination
Vaccination series commencing at <12 weeks of age	Vaccination series commencing at >12 weeks of age	
Commence at 6–8 weeks old, then vaccinate every 3–4 weeks until 16 weeks of age, then revaccinate after 1 year	Twice, at an interval of 3–4 weeks, then revaccinate after 1 year	Every 3 years

of γ-globulins and immunoglobulins, suggesting that feIFN-ω might have stimulated antibody production. However, this did not lead to a better outcome (Paltrinieri et al., 2007).

Antibody therapy

In several European countries, immune sera containing FPV antibodies are commercially available and are used to treat and prevent infection in susceptible animals. In one study, dogs naturally infected with CPVs were treated with lyophilised canine IgG. Recovery time in these dogs was faster than in untreated dogs (Macintire et al., 1999). The results of this study suggested that antibodies might have a therapeutic effect on CPV infection in the dog but, so far, no studies have been conducted in cats.

Prophylaxis

In FPV infection, the presence of antibodies is a strong predictor of protection (Scott and Geissinger, 1999; Lappin et al., 2002).

Passive immunisation

The prophylactic efficacy of passively transferred antibodies has been demonstrated in dogs and is also expected, but has not been proven in cats. Commercial antibody preparations produced in horses are available in some European countries. For the prevention of disease due to FPV infection, kittens under 12 weeks of age are given 2 mL SC and cats over 12 weeks of age are given 4 mL SC. If the commercial product of equine origin is used, repeated administration (at >1 week intervals) is not recommended, as this could lead to anaphylactic reactions that could be fatal. Additionally, cats treated with anti-FPV serum should not be vaccinated within 3 weeks following passive immunisation, as anti-FPV titres could interfere with the response to vaccination. If anti-FPV serum is not commercially available, serum harvested from cats that have survived natural infection or that have recently been vaccinated can also be used.

Passive immunisation is recommended if rapid protection is of primary importance e.g. in disease outbreaks, or when entering a shelter where infection pressure is high. Passive immunisation can also be used to protect young kittens with incomplete vaccination histories, colostrum-deprived kittens, or unvaccinated adult cats.

Herd immunity

Panleukopenia is considered a core vaccine component according to the American Association of Feline Practitioners (AAFP), the European Advisory Board on Cat Diseases (ABCD) and other expert groups (Truyen et al., 2009; Day et al., 2010; Scherk et al., 2013). One guiding principle of vaccination recommendations is that as many animals as possible in a population should be vaccinated, but in individual cats vaccine should be administered only as often as necessary. This emphasises the point that herd immunity is the most important factor for prevention of epidemics. Populations where <70% of animals are protected are considered at risk for development of epidemics.

There have been several studies on the prevalence of antibodies against parvoviruses in cats. In one US study investigating the prevalence of antibodies in 267 client-owned cats (Lappin et al., 2002), the critical value of 70% was almost reached (67%). In a recent study of 350 cats in Germany, the prevalence of antibodies against FPV was 71% (Mende et al., 2013). In a study from Costa Rica, antibodies were detected in 93% of cats; the high number is probably explained by high environmental contamination due to high infection rates in dogs (Blanco et al., 2009). Remarkably, relatively low protective antibody rates of 40% were documented in cats entering a Florida animal shelter (DiGangi et al., 2012). Low prevalence rates were also found in North-Eastern France, with an average antibody prevalence of 25% (Hellard et al., 2011).

The divergent prevalence rates are most likely associated with different infection pressures and vaccination programs in each region. The inclusion of cats that had survived naturally acquired infection could be the reason why in the German study, 8/28 (29%) cats that had never been vaccinated had antibodies against FPV. Interestingly, 11/47 (23%) cats that had been vaccinated according to the current recommendations (Table 1) did not have detectable serum antibodies (Mende et al., 2013). This perhaps supports another study in Germany in which 37% of kittens did not develop antibodies despite vaccination at 8, 12 and 16 weeks of age (Jakel et al., 2012).

Current vaccination recommendations

Vaccines against feline panleukopenia are considered core vaccines according to current vaccination guidelines worldwide. Table 1 shows the current vaccination recommendations by international expert groups (Truyen et al., 2009; Day et al., 2010; Scherk et al., 2013).

Cats that respond adequately to primary vaccination against FPV (Table 1), administered according to current guidelines, maintain a solid immunity for 7 or more years (Scott and Geissinger, 1999; Lappin et al., 2002). Despite the duration of immunity (DOI) indicated in these studies, experts worldwide recommend a very similar vaccination program, indicating that revaccination after the primary series at intervals of 3 years or longer are advisable, unless special conditions apply, such as immunosuppression by feline leukaemia virus infection (Truyen et al., 2009; Day et al., 2010; Scherk et al., 2013).

It is unclear whether a single dose of ML virus (MLV) FPV vaccine can induce adequate immunity in adult FPV-naive cats. In one experimental study, 64 8–10 week old specific pathogen-free kittens were inoculated once with a multivalent vaccine containing inactivated FPV or MLV (Patterson et al., 2007). At 14 days after vaccination, 31% of kittens receiving the inactivated vaccines had protective FPV titres, whereas 85% of kittens receiving MLV vaccines had protective titres. Thus, with few exceptions, use of MLV is preferred. This is also the case in adult breeding cats, as high maternal antibody titres are required for optimal protection of kittens. The study by Patterson et al. (2007) also suggests that in the absence of MDA, a single dose of a MLV vaccine might be sufficient to provide some protection. However, in a recent field study, these findings could not be confirmed, as 40% of 244 cats with feline panleukopenia had received at least one dose of vaccine but nevertheless developed the disease. Possible reasons for this include the relatively long duration of interference by MDA in kittens, vaccination of immunecompromised cats, vaccination of cats already infected, or the administration of less immunogenic inactivated vaccines (Kruse et al., 2010).

Lack of development of protective immunity

Feline panleukopenia is still commonly diagnosed in many countries despite the widespread use of vaccination (Kruse et al., 2010) and clinical disease has been described in both vaccinated and non-

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vaccinated cats. Thus, a lack of immunity can occur even when the current recommended vaccination guidelines are followed (Table 1). In a German study, 25% of cats diagnosed with feline panleukopenia were older than 1 year, and 11% were older than 5 years. This is at odds with the common belief that older cats are either protected by vaccination or by immunity developed in response to previous subclinical infections. The relatively high infection rate in older cats in the German study could be explained by a lack of environmental virus exposure, as in that study indoor cats were overrepresented (Kruse et al., 2010). As natural contact with parvoviruses is less likely in indoor cats, there is reduced opportunity to build protective immunity after a subclinical infection (Gaskell et al., 2006). In addition, indoor cats are less commonly vaccinated, due to the misconception by their owners that it is not necessary.

It is well known that MDAs interfere with the development of active immunity after vaccination (Scott et al., 1970; Chappuis, 1998; Day, 2007). MDAs to feline panleukopenia have a half-life of 9.5 days. Until recently, MDAs were considered to decline to a level that could be overcome by MLV at 12 weeks of age (Scott, 1971), but current guidelines recommend vaccinations every 3–4 weeks until 16 weeks of age (Truyen et al., 2009; Day et al., 2010; Scherk et al., 2013). In 2008 and 2009, several outbreaks of feline panleukopenia in Norwegian Forest cats were reported in Germany (Hoffmann et al., 2010). Subsequently, a field study revealed that 37% of kittens did not develop antibodies despite three vaccinations at 8, 12, and 16 weeks of age. MDAs were found in most kittens beyond 12 weeks of age; in some cats, MDAs interfering with primary vaccination and preventing antibody development were detected until 20 weeks of age (Jakel et al., 2012). Thus, even if a course of vaccination is continued until 16 weeks of age, this is not sufficient to protect all kittens.

Evaluation of the best starting point for primary vaccination by antibody testing in individual kittens could be helpful to reduce incidence of feline panleukopenia. The optimum starting point is defined as the age when MDAs have dropped below a certain level (Jakel et al., 2012). Alternatively, future recommendations for primary vaccination might include continuing the kitten series of vaccinations until 20 weeks of age, or revaccinating at 6 months of age after the kitten series has been completed.

Antibody titre testing and booster vaccination

Although rare in cats, mild to severe adverse reactions can occur after vaccination; these include feline injection site sarcomas, which have a guarded prognosis because of frequent recurrence after surgical excision (McEntee and Page, 2001; Moore and HogenEsch, 2010). In previously vaccinated cats, detection of FPV-specific antibodies above certain pre-determined levels is predictive of protection, regardless of vaccine type or vaccination interval (Scott and Geissinger, 1999; Lappin et al., 2002). Therefore, yearly FPV antibody titre testing can be helpful in determining the susceptibility of individual cats and the response to vaccination before a decision is made regarding the need for booster vaccination (Schultz et al., 2002). However, in the interpretation of antibody test results, it should be remembered that while a protective titres against FPV denote immunity, cats with negative or low antibody titres might still be protected (DiGangi et al., 2012).

Studies have been conducted to investigate whether point of care tests can also be used for the detection of antibodies. One study investigated the accuracy of the ImmunoComb Feline VacciCheck antibody test kit (Biogal) in young (probably unvaccinated) cats entering a shelter in Florida (Digangi et al., 2011). Subsequently, the test was modified to increase its sensitivity. In a German study investigating the modified test, sera from 347 cats were evaluated, using haemagglutination inhibition (HI) as the reference method. The sensitivity of the test (87%) was higher than in the shelter study from Florida (49%). However, the specificity had decreased from 99% to 81% (Mende et al., 2012). If the intended use of the test is to aid in clinical decision making prior to the consideration of booster vaccination of individual cats in veterinary practice, minimising the number of false positive test results is important, because it is necessary to accurately identify unprotected animals. Thus, high test specificity is most important.

Conclusions

Feline panleukopenia is a common disease with high morbidity and mortality. Despite the frequent use of effective vaccines, studies have shown that the feline population is not well protected in many countries. Recently published reports of FPV antibody prevalence and vaccine protection rates should underpin a reconsideration of current vaccination guidelines. Adequate primary vaccination of kittens, resulting in protective immunity, is still the most important strategy for the prevention of FPV infection.

Conflict of interest statement

None of the authors of this paper has a financial or personal relationship with other people or organisations that could inappropriately influence or bias the content of the paper.

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