Review Article

BASJVETRES

BASRAH JOURNAL OF VETERINARY RESEARCH, 2025, 24(2):141-165.
https://bjvr.uobasrah.edu.iq/

Feline Calicivirus: A Comprehensive Review

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DOI: https://doi.org/10.23975/bjvr.2025.158296.1207

Received: 16 March 2025 Accepted: 26 May 2025

Abstract

A highly mutated RNA virus known as feline Kalisi virus (FCV) is commonly found in domestic cats. However, its potential for transmission to humans is minimal. FCV has significant genetic and antigenic diversity in cat populations. Severe acute systemic FCV infection (VS-FCV) manifests itself as lingual ulcers for upper respiratory tract diseases, gingivitis, alopecia, skin, mouth, ear problems, and claudication syndrome. Other symptoms include necrosis of the pancreas, liver, and spleen, bronchointerstitial pneumonia, and subcutaneous edema which contribute to the high mortality rate of infected cats. The diagnosis of FC virus is carried out using two techniques: reverse transcription-polymerase chain reaction (RT-PCR) and virus isolation. The frequency of antibodies is usually high in cat populations due to vaccination and symptomatic diseases. Therefore, the presence of certain antibodies does not always indicate the presence of a prolonged infection. The mainstay of FCV treatment is supportive care, although there are no authorized antiviral drugs specifically designed for the virus. However, drugs such as misuripine and nitazoxanide have proven their antiviral effectiveness in the laboratory. The types of vaccines available include inactivated and modified vaccines. Vaccination is necessary for the prevention of FCV virus. Preventing the spread of FCV requires proper hygiene and effective disinfection, especially in versatile environments.

Keyword: Calicivirus, Cat, Ulceration, gingivostomatitis

Introduction

Domestic cats are commonly infected with the highly mutagenic RNA virus known as feline calicivirus (FCV) (1). The only hosts it affects are Felidae, and it has no zoonotic potential. According to (2), FCV exhibits significant genetic and antigenic variation in feline populations. Upper respiratory tract disease (URTD), necrotizing pododermatitis with serocellular crusts, alopecia, lingual ulcerations, gingivostomatitis, ulceration of the skin, oral cavity, pinnae, nares, and Limping syndrome are all indicators of a severe systemic FCV infection. Infection-related complications in cats include pancreatic, hepatic, and splenic necrosis, subcutaneous edema, and a high mortality rate (3).

Techniques for diagnosing FCV include electron microscopy, immunohistochemistry, RT-PCR, virus isolation in cell culture, and antibody detection. It is possible to identify FCV antibodies using both virus neutralization assays and the enzyme-linked immunosorbent test (ELISA).

Because of vaccination and natural infections, antibody prevalence is typically high in cat populations. Therefore, certain antibodies are not a sign of an active infection (4). Although there are no approved antiviral medications specifically for FCV, supportive care is the mainstay of treatment; however, substances such as mizoribine and nitazoxanide have demonstrated antiviral activity in vitro (5). The management of FCV requires vaccination; different vaccine types, such as modified-live and inactivated vaccines, are available (6). Controlling the spread of FCV requires hygienic practices and efficient disinfection, particularly in multicat settings (7,8). The objective is to provide an overview of the virus's current status, investigate new research, and offer insights into its pathogenesis, clinical manifestations, epidemiology, diagnosis, prevention, and available treatments.

Etiology

In the family Caliciviridae, FCV is a member of the order Picornavirales. The term refers to the cup (from Latin calyx) shaped depressions" that appear on the surface of the virion when examined under an electron microscope (9). Vesivirus, Lagovirus, Minovirus, Nacovirus, Nebovirus, Norovirus, Recovirus, Salovirus, Sapovirus, and Valovirus are among the eleven genera that comprise the family Caliciviridae (9)

Fish are infected by the genera Lagovirus, Norovirus, Nebovirus, Recovirus, Sapovirus, Valovirus, and Vesivirus, whereas birds are infected by Minovirus and Salovirus, and mammals are infected by Bavovirus and Nacovirus. The author (9) claims that FCV belongs to the Vesivirus genus, which also contains the swine virus's vesicular exanthema (9).

Caliciviruses from the genus Lagovirus, which comprises the viruses that cause rabbit hemorrhagic sickness and European brown hare syndrome, also have an impact on veterinary care. Some of the caliciviruses found in sea lions, dogs, and minks are thought to be vesivirus-like viruses, but none of them have been described. Vesivirus virions are icosahedral

in symmetry, non-enveloped, and range in diameter from 27 to 40 nm. The FCV is a positively polar single-stranded RNA virus. The virus contains three open-reading frames (ORF) and a roughly 7.0 kb genome (9).

Post-translational cleavage of the polyprotein encoded by ORF 1 results in the production of non-structural proteins, including the viral protease and polymerase complex, the RNA-dependent RNA polymerase, the NTPase enzyme, and the viral protein genome (Vp-G)-linked (10). The production of infectious viruses and binding to the host cell depend on the major capsid protein (VP1) and the minor capsid protein (VP2), which are encoded by ORF2 and ORF3, respectively (11,12). According to (9), the VpG connected at the 5' end acts as a primer during the synthesis of viral RNA. According to (13), the poly-A tail at the 3' end of the genome is essential for viral RNA stability and translation.

The capsid gene can be divided into six sections, A through F, according to (14). Region E consists of the center conserved region, the 3' prime hypervariable region (HVR), and the 5' prime HVR. During recurrent FCV infections, the entire HVR E is believed to be a target for immune evasion, and the 5' HVR has epitopes that neutralize monoclonal antibodies (15,16). The viral protease breaks down the precursor capsid protein into two different forms: the smaller leader capsid protein and the bigger main VP1 capsid protein (17).

It was found that the leader capsid protein is necessary for the development of viruses that can cause a cytopathic effect in feline kidney cell culture (18). For FCV attachment, entrance, and other downstream activities, it has been determined that the cellular receptor junctional adhesion molecule 1 (JAM 1) is a functioning receptor (19,20). Cats' endothelium and epithelial cells' cell-cell junctions are the main locations of the feline JAM 1 (fJAM 1) receptor. The vesicular and ulcerative lesions observed after an FCV infection are indicative of the disruption of intercellular connections (21).

Nonetheless, virulent-systemic (VS)-FCV isolates and isolates from pneumonia-stricken kittens were inactivated following in vitro incubation with the fJAM 1 ectodomain, and the FCV F9 vaccine isolate was resistant to receptor-mediated inactivation by fJAM 1, indicating that there are differences amongst FCV isolates concerning receptor interaction (22).

According to (23), FCV is a highly mutagenic virus that evolves at frequencies of 1,32×10-2 to 2,64×10-2 substitutions per nucleotide annually for strains that circulate throughout a population and 3,84×10-2 to 4,56×10-2 substitutions per nucleotide annually for strains that circulate within people.

For RNA viruses, this is one of the quickest rates of evolution that has been discovered. Due of the large genetic variation across related isolates revealed by sequence analysis, FCV is thought to live as a so-called quasispecies within the host (16). Nucleotide and amino acid studies of the HVRs of various isolates have led to the division of FCV into two genogroups; isolates from genogroup II are solely from Japan (24). Based on studies of the genetic diversity and evolution of FCV, it is generally believed that 20% of the genetic

distance allows strain differentiation (25, 26).

Epidemiologically unrelated isolates differ from one another by over 20% in terms of nucleotide and amino acid levels in the variable regions C and E of the capsid gene. For this reason, they are regarded as distinct strains. According to (26,27&28), isolates that are epidemiologically related, such as those found in acute and virulent systemic illness outbreaks, are considered variants of the same strain since they are practically identical. Where FCV is prevalent in cat colonies, a single strain of the virus might differ by as much as 18% (23).

FCV strains show considerable genetic and antigenic strain complexity both spatially and temporally, and no single field strain is more common than the others (29). Long-term survival within a susceptible community or individual is facilitated by recombination, sequential reinfection, and the cumulative accumulation of random mutations inside a single isolate, indicating that viral evolution is not only reliant on isolate competition (23,30, and 31).

Research has shown that two distinct strains of FCV can co-circulate in a cat shelter and that two strains of the virus can infect a single cat simultaneously (28). The above strategies could make FCV strains more genetically diverse until new strains appear; this high genetic plasticity could help with the evasion of the immune system (23). According to (3,32, and 33), despite the fact that genetic heterogeneity results as well in antigenic diversity, the strains appear to be sufficiently similar genetically to provide some degree of cross-protection by vaccination.

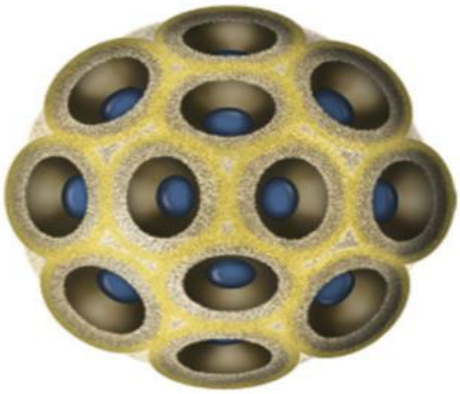

Figure 1: Schematic of the structure of the capsid of feline calicivirus (34).

Epidemiology

Other than wild cats, there are no known reservoirs or hosts for FCV. The risk of infection in humans is low. Although they probably have a minimal impact on the epidemiology of FCV in dogs and cats, researchers have isolated viruses that resemble FCV from dogs (35). The majority of cats with acute disease release FCV through their nasal and oral secretions, even though it is found in their blood, urine, and feces. After an infection, many cats shed; some do so for years or even their whole lives. Despite extended exposure to FCV, a tiny proportion does not shed, perhaps as a

result of host genetic or immune-mediated traits (36).

In general, cats are frequently infected with FCV. A study conducted in Italy on colonies of stray cats found that 85% of the cats had positive FCV antibody tests (37). An Australian study found no correlation between the socioeconomic status of the owners and the prevalence of FCV infection, in contrast to FIV infection (38). The presence of a few persistently infected cats and the reinfection of other cats with a different strain or a variation of the same strain of FCV are linked to high FCV strain diversity and high FCV prevalence within a colony (23,39). The viruses that infect each cat colony or group are usually distinct and come from various ancestors (39,40). Several strains of FCV can infect a cat simultaneously (23, 28). When new strains are introduced into a cat colony or when individual cats are infected with different strains, recombination events can occur, increasing genetic variety. In cats, endemic infection may be possible due to the selection of antigenic variations that avoid the collective immune response (23,30).

The study by (41) found that there is a notably high genetic variability of FCV in shelter environments with high cat turnover, which often leads to the introduction of new virus strains. However, in stable multicat homes, the diversity of FCV viruses was low (41). Maintaining proper hygiene and biosecurity can prevent the spread of FCV in cat shelters (31). Direct contact with the secretions of cats who are carriers and acutely infected is typically how infection is contracted (42).

However the virus can live and spread on dry surfaces for up to a month at room temperature; it can live even longer in colder climates (43, 44, 45). Additionally, facilities with cats that shed FCV have been observed to have aerosol propagation, and the virus lives longer in moist environments than in dry ones (46).

It is possible for indirect transmission to occur, particularly in the small space of a cattery where secretions could contaminate cages, cleaning and feeding supplies, or staff. As has been reported for VS-FCV variants, facilities also need to be ready for indirect transmission through humans or fomites (47, 48, 49).

One study found that the primary source of a VS-FCV outbreak was the hands of caregivers. Even with the use of washing and disinfection procedures, it was predicted that one student could infect up to ten cats during a physical examination or when giving oral medication (50). Additionally, kittens may experimentally contract FCV by coming into contact with infected fleas or their feces due to the fact that flea feces can harbor the virus for as long as eight days (51,52).

Pathogenesis

FCV is a valuable model for studying molecular pathogenesis because, in contrast to other Caliciviridae family members, it can be cultivated in cell culture (3). Investigating viral pathogenesis and disinfection methods now requires this organism. The oropharynx is the primary site of FCV infection in cats, resulting in transient viremia that facilitates FCV's spread to other tissues. Cat epithelial and endothelial cells are infected by VS-FCV, which results in cell death, vascular damage, and significant mortality, according to

immunohistochemistry and electron microscopy (53).

Targeting the receptor fJAM-A at epithelial and endothelial cell junctions, VS-FCV strains cause leakage and breakdown of these junctions. The researcher stated that the identification of FCV RNA in the blood adds credence to the notion that FCV is hematogenous distributed, as demonstrated by the presence of the fJAM-A receptor on feline platelets and blood leukocytes (21). Studies have demonstrated that fJAM-A expression systems can convert non-permissive cells into FCV-permissive cells, underscoring the vital receptor role of the fJAM-A proteins (19). Aside from the usual oral symptoms, FCV can also result in lameness, or limping syndrome. The affected joints have FCV, and the onset of limping syndrome is associated with immune complexes. It has been proposed that polyarthritis, which manifests as fever and lameness, is a type III hypersensitivity reaction that cats may experience following an FCV infection or vaccination (54). Antigen-antibody complexes develop and build up in the joints during this reaction, causing an acute inflammatory response. Despite being linked to FCV, the primary causes of the syndrome are believed to be co-infections with the FCV field strain or, in rare instances, vaccine strain reactivation (55). Infected cats have higher levels of cytokines, especially IL-10, TNF-alpha, and MIP-1alpha, which may indicate a systemic inflammatory response (56). Membrane blebbing and cell rounding are two typical cytopathic effects of FCV infection in cell culture (57). In cell culture, FCV infection activates the mitochondrial pathway, which in turn causes apoptosis and caspase activation, according to molecular research (58).

FCV employs antigenic drift and shifts as strategies to overcome the host's defenses. Recombination events between different strains of FCV have also been documented (antigenic shift), and mutations accumulate continuously (antigenic drift) due to the error-prone RNA polymerase in FCV. These changes could lead to significant changes in viral epitopes, which could either decrease recognition by neutralizing antibodies or increase FCV's ability to bind to cell receptors and thereby increase its infectivity. Another immune evasion strategy used by FCV is host "shut-off," which is the inhibition of host protein synthesis (59). By restricting protein synthesis to just the necessary proteins for viral replication, this mechanism undermines the host's ability to fight off infections (60,8).

Clinical Manifestations

Acute symptoms in the upper respiratory tract and mouth can be caused by an FCV infection. At the same time, it has been associated with persistent gingivostomatitis, a condition thought to be brought on by an immune reaction. There is also evidence of VS-FCV illness and the "foot and paw" conditions

.1-**Upper Respiratory Tract and Acute Oral Illness**

The clinical presentation may vary based on the specific FCV strain, the age of the infected cats, and factors related to their care and management. Although infection can be asymptomatic in certain situations, it often

presents with a characteristic presentation of lingual ulcers (as shown in Figure 2) and a relatively moderate acute respiratory disease (61). Acute respiratory and oral illnesses are the main conditions that kittens show symptoms of. The incubation period lasts anywhere between two and ten days (62). Fever is a commonly observed symptom, and the main indications are oral ulcerations, sneezing, and serous nasal discharge (63). Anorexia is often more obvious than rhinitis symptoms, especially when combined with excessive salivation (Figure 3) brought on by mouth sores (mostly on the tongue) (1). When treatment is focused on symptom relief, symptoms usually get better in a few days (64). Young kittens may develop pneumonia, which manifests as fever, coughing, depression, and dyspnea (65)

2. Feline Chronic Gingivostomatitis (FCGS)

The characteristic feature of FCGS is inflammation that permeates beyond the mucogingival junction and reaches the alveolar mucosa and other soft tissues. If the gingival tissues are the only area inflamed, FCGS cannot be diagnosed (66,67,68). Acute sickness characterized by fever, lethargy, oral ulcers, gingivitis, rhinitis, and/or conjunctivitis is thought to occur when kittens are intentionally administered FCGS because it causes an immune system reaction to FCV (and possibly other oral antigens) (69).

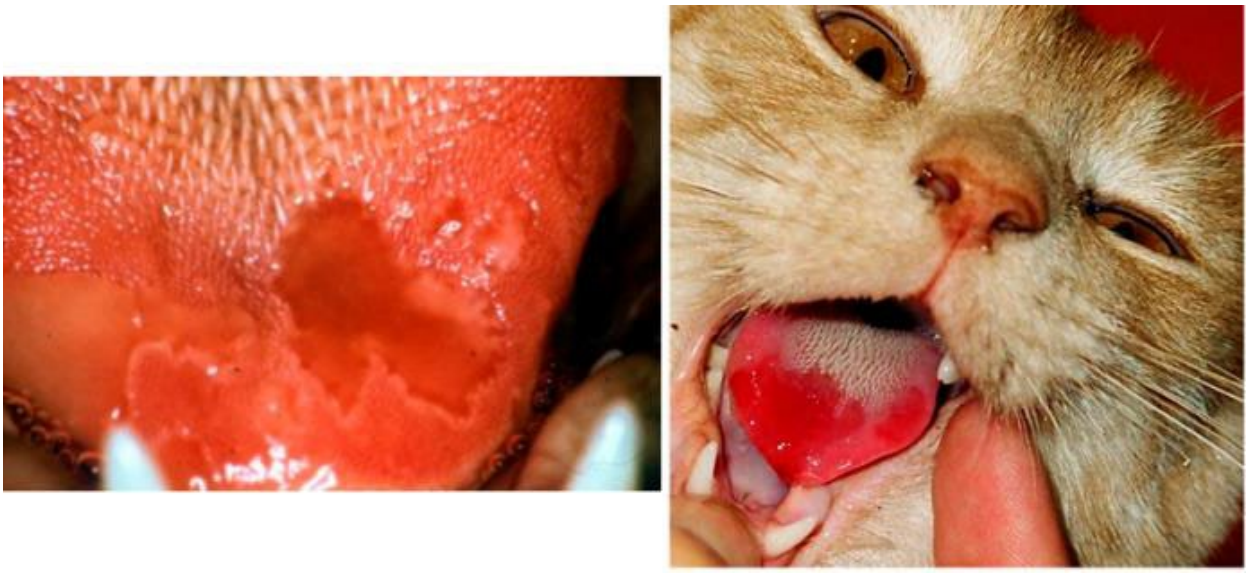

Figure 2: Cats infected with FCV have ulcerative sores on their tongues (52).

Figure 3: Excessive salivation in a feline with feline calicivirus (FCV) infection. Tongue ulceration can cause significant pain, resulting in difficulties in consuming food and liquids and excessive production of saliva (52)

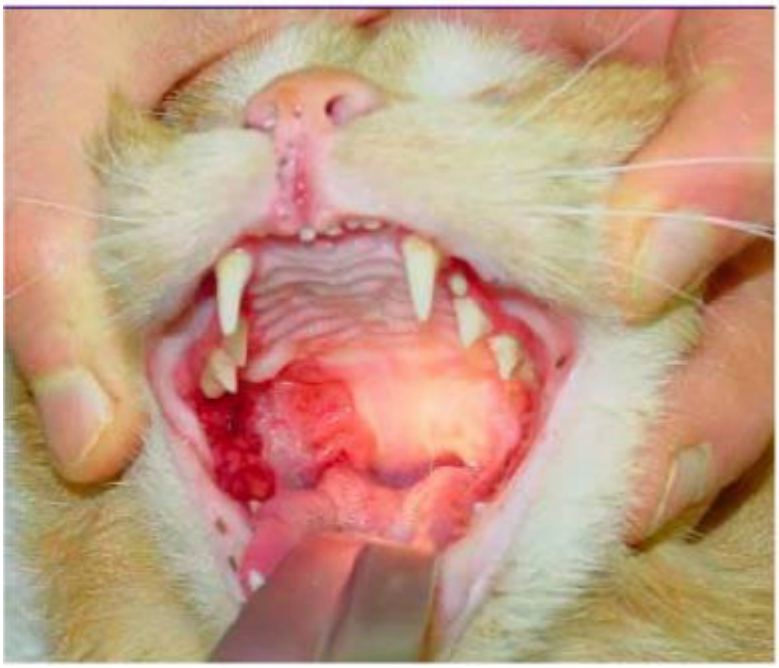

Figure 4: Feline chronic gingivostomatitis (52).

3. Limping Syndrome

A sudden and transient lameness (as shown in Figure 5) may happen after receiving an FCV vaccination or be associated with an FCV infection. There is a fever associated with this condition. Since immunization was often coincidental and FCV field strains different from the vaccine strains were also found in many cases, the limping is not always due to the vaccine strain alone (52). In cases of spontaneous infection, limping syndrome may appear a few days or weeks after the first respiratory or oral symptoms. The lameness can spread quickly between limbs and may be severe. Without assistance, full recovery frequently occurs in 24 to 48 hours (70).

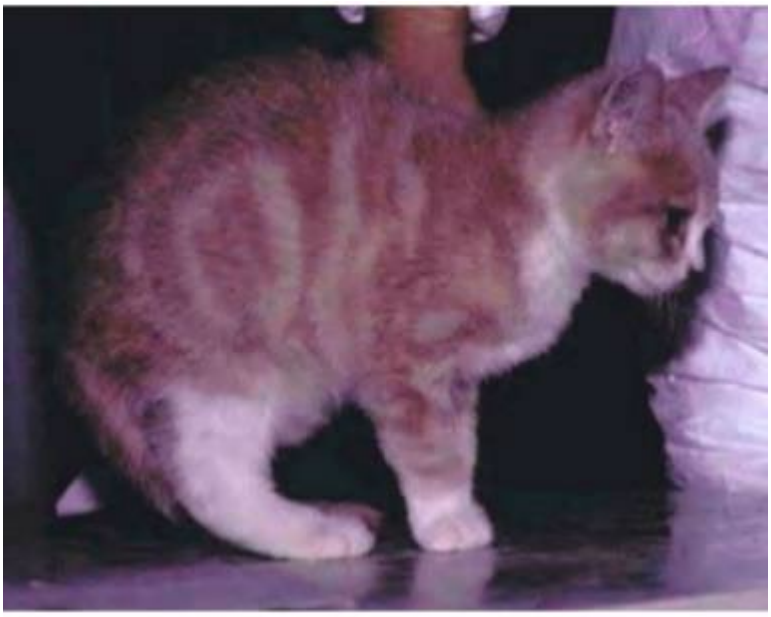

Figure 5: Calicivirus infection limping syndrome. (52)

4. Paw and Mouth Disease

At first, the illness showed a lot of similarities to VS-FCV, but neither the death rate nor the disease's prevalence increased significantly. In addition to fever, depression, and appetite loss, notable clinical symptoms include skin swelling and open sores on the paws, head, mouth, and around the anus (71). The head and limbs are where the edema most commonly shows up. Although there have been fatal cases, the fatality rate is usually low. In contrast to VS-FCV, "paw and mouth" illness does not have a widespread outbreak and is only seen in one or a small number of cats. (72, 73).

5. Virulent Systemic Feline Calicivirus Infection (FS-FCV)

The disease was first known as "hemorrhagic-like fever" when domestic cats contracted the extremely virulent and frequently lethal FCV virus (47). However, because hemorrhages were infrequent, the term "highly virulent feline calicivirus disease" was later proposed as a more

accurate description (74). The term "virulent systemic feline calicivirus" (VS-FCV) is commonly used to describe the predominant viral strains responsible for the disease (48).

In phylogenetic analyses of VS-FCV and FCV from cats with various FCV-associated clinical signs or cats without symptoms, there was no grouping based on the various clinical presentations. Consequently, no specific mutations that could differentiate between different clinical manifestations were found, and there was no discernible relationship between the virus's genetic makeup and the severity of the illness (6). Common strains of VS-FCV that cause systemic illness include those that cause multiorgan failure, severe systemic inflammatory response syndrome, disseminated intravascular coagulation (DIC), and often death. The mortality rate is significant, ranging from about 30% to 70% (56). A VS-FCV infection can present with a variety of clinical symptoms. Often, the preliminary results exhibit characteristics of a severe acute upper respiratory tract infection. Cutaneous edema and ulcerative sores on the skin and paws are two characteristic symptoms. The head and limbs are where the edema is mostly found (Figure 6). The nose, lips, ears, and periocular region exhibit crusted lesions, ulcers, and hair loss (Figure 7), as well as on the tongue, oral cavity, and footpads (Figures 8 and 9) (62).

Figure 6: Cats infected with VS-FCV may have edema of the head and limbs (52).

Figure 7: Cats with VS-FCV infections have crusted lesions and ulcers (52).

Additionally, similar initial clinical signs, including edema, mouth and skin ulcers, and elevated body temperature, distinguish the presence of paw and mouth disease from FCV infection (72). Jaundice, which can be brought on by diseases like pancreatitis or hepatic necrosis, is a symptom of VS-FCV disease in some cats (Figure 10). Additionally, pulmonary edema can cause severe respiratory distress in certain cats. Petechiae, ecchymosis, epistaxis, or bloody stools can be symptoms of thromboembolism and coagulopathy brought on by disseminated intravascular coagulation (75).

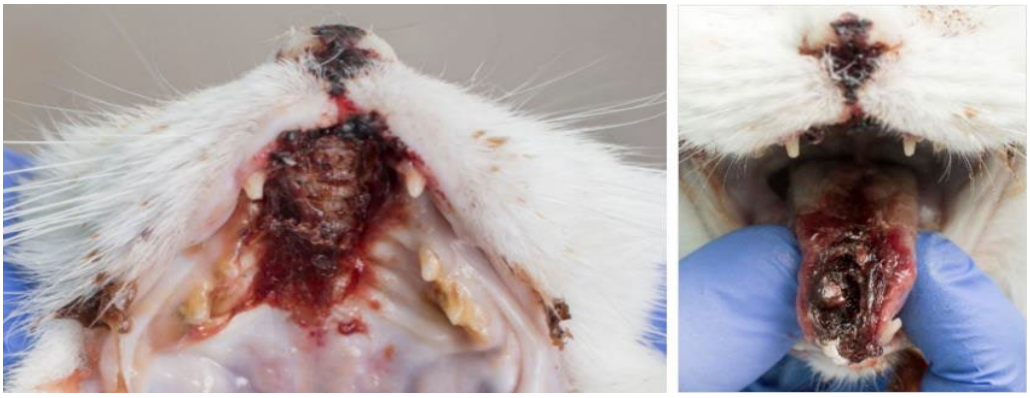

Figure 8: severe necrosis in a cat infected with VS-FCV (philtrum, palate, tongue, and lip commissure) (52).

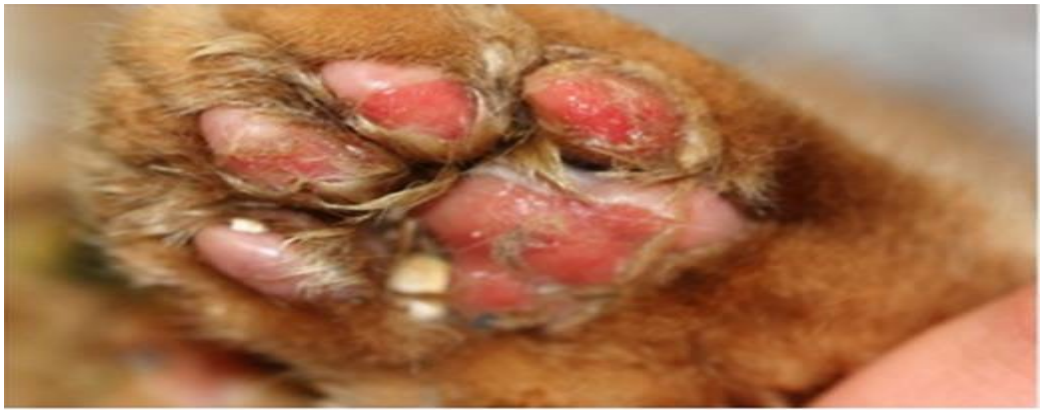

Figure 9: Foot excoriations in a feline infected with VS-FCV (52).

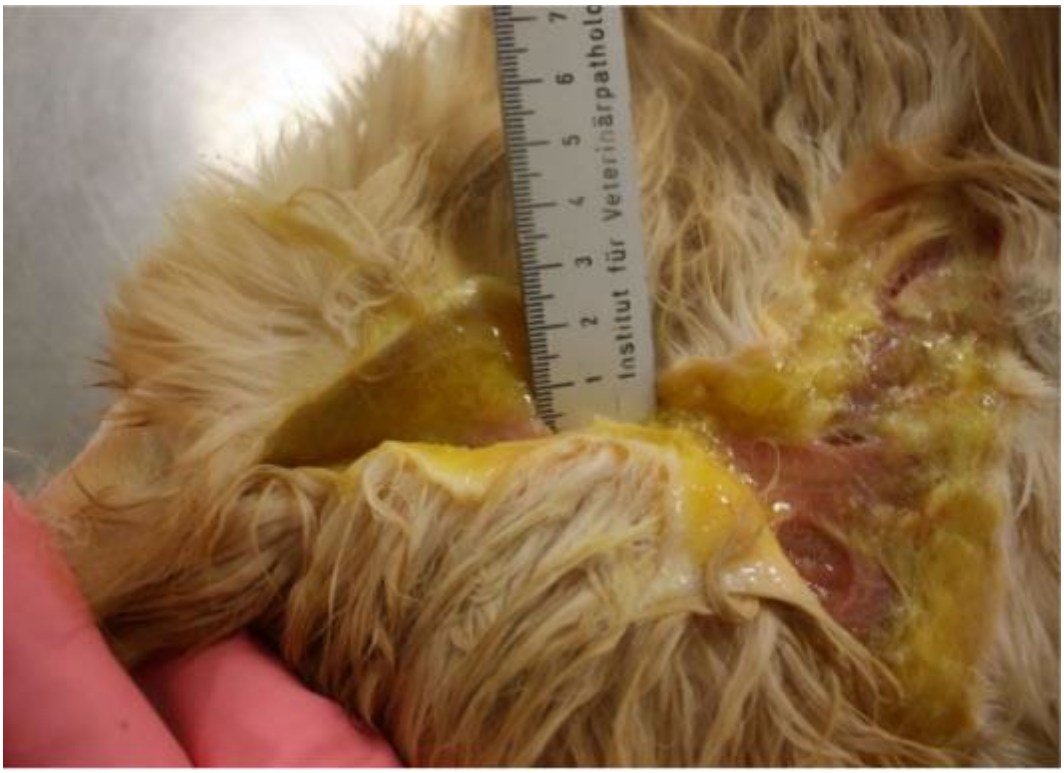

Figure 10: At necropsy, a cat with a VS-FCV infection had jaundice and oedema of the subcutaneous region (52,72).

6. Other Clinical Presentations

Although there is not enough evidence to support these links, FCV has also been connected to other conditions like cystitis and polyps in the tympanic bulla (76). Additionally, FCV was isolated from the feces of cats who had intestinal inflammation, which lends credence to the theory that some strains of FCV might become favored for the intestines and act as intestinal pathogens (77).

Host Immune Response to FCV

1. Innate immune response

A cat's innate immunological response, which is the first line of defense, is rapidly triggered by a feline calicivirus (FCV) infection. By identifying common patterns, the innate immune system can identify viral components, including viral RNA, which sets off a series of immune reactions (78). This includes the production of interferons by the host cells and the activation of natural killer (NK) cells, which can directly destroy virus-infected cells (79).

2. Adaptive immune response

By providing a more focused and accurate defense mechanism, the adaptive immune response strengthens the innate immune response. After identifying and responding to specific FCV antigens, B cells develop into plasma cells that produce anti-FCV antibodies (79). By binding to virus particles, these antibodies prevent the virus from infiltrating host cells and mark the particles for phagocytic cells to recognize and eliminate (78).

3. Immunological memory and immune evasion

The host immune system creates immunological memory to defend against FCV after it first attacks. After the initial infection, memory B cells and memory T cells remain in the host for a long time (78). These memory cells can proliferate quickly and generate a quicker and more effective immune response when the same or similar FCV antigens are encountered again. However, as shown in Figure 11, FCV has immune evasion mechanisms that allow it to evade detection by host antibodies. This is primarily because the virus's surface proteins are so diverse (79,80).

Diagnosis

After receiving a modified live virus vaccination, cats may occasionally release feline calicivirus (FCV), even if they do not exhibit any symptoms (81).

1. Detection of Nucleic Acids

Several RT-PCR assay types, including conventional, nested, and real-time, have been developed to detect FCV RNA in a variety of samples, including conjunctival and oral swabs, blood, skin scrapings, or lung tissue. The selection of the sample is based on the specific clinical manifestation and progression of the disease (82). The RT-PCR's diagnostic sensitivity is dependent on the strain being identified and the primer selection because the viral genome is highly diverse. To maximize molecular assays and reduce the possibility of false-negative results, it is crucial to test a range of strains (83).

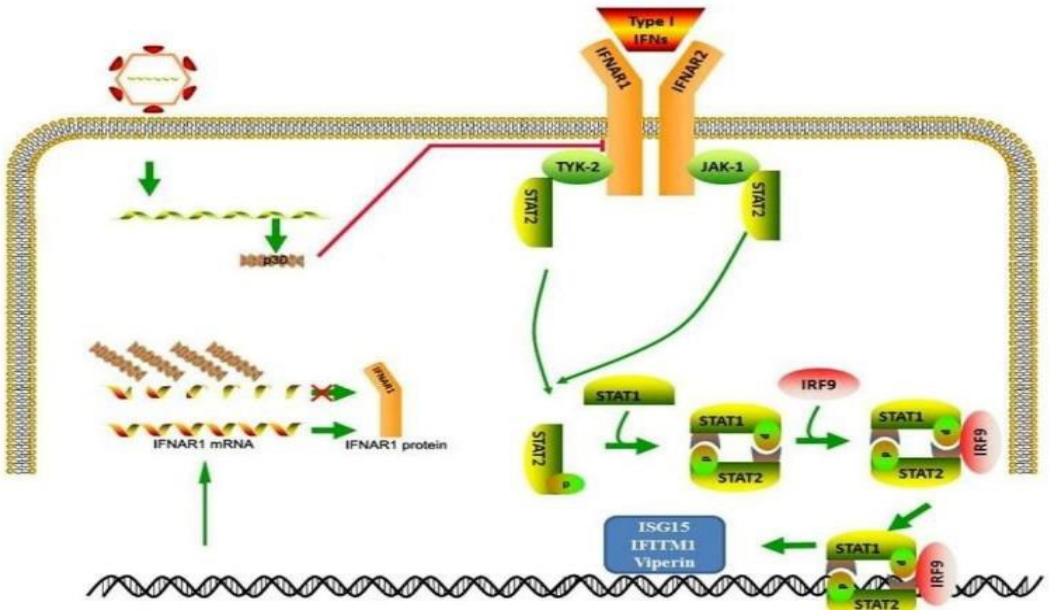

Figure 11: FCV immune escape mechanism (Explores the impact of Feline calicivirus (FCV) on the immune response of host cells by examining its immune evasion mechanisms. The diagram illustrates the interferon signaling system, which is a vital mechanism for cellular response to viral infection. Initially, the cell detects the existence of the virus, which triggers the synthesis and secretion of Type I interferons (IFNs.), (80).

Furthermore, improvements have been made to multiplex PCR/RT-PCR assays that can detect Feline Calicivirus (FCV), Feline Herpesvirus (FHV), and occasionally Chlamydia felis simultaneously. But it's important to remember that these tests might be less sensitive. Apart from its capacity to identify FCV infection, RT-PCR can also be used to pinpoint the virus strain. Molecular epidemiology and epidemic investigations have benefited from this method (84). The higher sensitivity of real-time RT-PCR makes it generally superior to traditional RT-PCR assays in diagnostic settings. Quantitative assays, if at all possible, are also recommended since they can reveal the quantity of virus found in positive samples. Interpreting the diagnostic FCV RT-PCR results in light of the clinical symptoms is crucial (85).

2. Virus Isolation

The technique of virus isolation is useful in detecting FCV infection. It verifies the existence of a virus that is actively reproducing and is less impacted by strain diversity than RT-PCR. FCV multiplies in feline cell lines, and its quick growth in tissue culture may make it more difficult to identify concurrent FHV infection (84). In order to maximize the likelihood of successfully isolating the virus, it is recommended to obtain swabs from both the conjunctiva and the oropharynx. The virus

can be extracted from swabs obtained from the nose, eyes, or oropharyngeal site (86).

3. Antibody Detection

The ELISA or virus neutralization can be used to detect the presence of FCV antibodies. Due to both vaccination and spontaneous infection, cat populations usually have a high incidence of antibodies (4). As a result, identifying specific antibodies is not a trustworthy way to diagnose an infection (87). A cat's level of protection can be determined by looking at their virus-neutralizing antibody levels. However, if the cat's virus-neutralizing antibodies do not react with the particular laboratory strains used in the test, false-negative results could result, so care must be taken when interpreting these levels (88).

4. Diagnosis of FCV Infections

Particularly in highly virulent strains associated with VS-FCV, there are still no established genetic markers that reliably indicate virulence (29). By examining the amino acid properties of the E region of VP1, it was discovered that seven residue sites (438, 440, 448, 452, and 455 in the E30 HVR; 465 in the E-conserved area; and 492 in the E50 HVR) varied amongst the various pathotypes (89). According to one study, a highly contagious FCV isolate's capsid gene was the cause of its quicker growth kinetics in a lab setting (20). But in two other outbreaks, the VS-FCV showed either an intermediate configuration or the pattern commonly observed in a respiratory pathotype (6).

Disease Management

1. Treatment of FCV infection

Despite not having a phospholipid bilayer envelope, FCV is very persistent. Infected cats' surroundings have been found to contain FCV RNA by RT-qPCR up to twenty-eight days after shedding ceased, although no replication-competent virus was ever found (46). Shelters and animal hospitals are at serious risk because of FCV's prolonged environmental stability and the continuous admission of fresh cats with unclear immunological, immunization, and illness histories. All surfaces must be well cleansed before employing disinfectants such as potassium peroxymonosulfate (1%, 10 min), sodium hypochlorite (2,700 ppm, 1 min), accelerated hydrogen peroxide (35,000 ppm, 10 min), or aldehydes (2%), as these are known to be effective against FCV (90).
According to (91), virucidal disinfectants that are effective against human norovirus are also suitable for FCV because the two viruses have comparable viral characteristics. Supportive management for cats with FCV-induced URTD comprises intravenous fluids, nebulization therapy, non-steroidal anti-inflammatory drugs, and the provision of very attractive food to maintain nutrition (7).
Clinical signs and FCV replication have been demonstrated to be reduced by recombinant feline interferon omega (feIFN-ω) (92). Research has looked into possible treatment options even though there aren't any direct antiviral medications against FCV on the market at the moment. The combination of mefloquine and feIFN-ω has

demonstrated potential in preventing FCV replication. Mizoribine and nitazoxanide have shown antiviral activity in vivo and in vitro, indicating possible therapeutic advantages (5). Drug screening has shown that handling suppresses HSP70 expression in vitro, which inhibits FCV infection (93). Though their effectiveness in vivo is still unknown, a number of antiviral medications, such as ribavirin, polysodium 4-styrenesulfonate, mefloquine, and natural compounds, have shown inhibitory effects against FCV replication in vitro (52). Acute viral feline URTD has been successfully treated with sera that contain antibodies against FCV, feline herpesvirus (FHV), and feline parvovirus (FPV) (94).

For the treatment of feline chronic gingivostomatitis (FCGS) brought on by an FCV infection, a number of strategies have been investigated, including mesenchymal stem cell therapy, corticosteroid therapy, and oral hygiene. When secondary bacterial infections occur in patients with FCGS, broad-spectrum antibiotics are used to treat them. In addition to lowering stress, improving the atmosphere, and upholding good hygiene, prednisolone and felFN-ω are taken into consideration for managing FCGS in multi-cat households (95).

2. Immunization

Vaccination is a crucial part of treating FCV infections, which lowers viral shedding and prevents severe clinical symptoms and inflammation. Vaccines that are inactivated or modified-live are both commercially accessible (96). Despite their potential, new vaccine technologies like mRNA and subunit vaccines are not yet commercially available. To establish the proper immunization kinds and intervals, vaccination guidelines include variables such as age, health, lifestyle, and housing conditions (96).

Clinical symptoms and an in anti-FCV IgM antibodies correlated with FCV shedding (97). IgM levels rose initially when the virus-neutralization activity started, but IgG antibody levels quickly increased after that. The neutralizing capacity is mostly contributed by IgG antibodies, however, IgM antibodies seem to play a significant role in the early stages of the neutralizing response. However, distinguishing vaccine-induced antibodies from wild-type antibodies can be challenging. Studies have also been conducted on the presence of IgA antibodies in serum and saliva. Studies have indicated that salivary IgA antibody peak levels were detected earlier than serum levels, suggesting that salivary IgA may contribute to early defense against FCV (3). After the first dosages at ages 8–9 and 12 weeks, the standard vaccination schedule recommends annual booster shots. The effectiveness of this regimen is debatable, and evidence suggests that some kittens may not develop adequate immunity by 12 weeks due to residual MDA. Immunity lasts for different lengths of time, especially when heterologous challenges are included. The increasing trend of vaccinations focuses on improving cross-reactivity, decreasing challenge viral shedding, and limiting persistent infection since antigenic diversity among FCV strains can occasionally lead to vaccine failures. The effectiveness of vaccines must be assessed through

challenging experiments because there are no biomarkers for protection (10).

Vaccines against FHV or FPV are frequently combined with those against FCV. There is a chance that adjuvant-containing inactivated vaccines could cause injection-site sarcomas (52). In Europe and Japan, however, there is a non-adjuvanted, inactivated vaccine against FCV that is made by combining two inactivated virus strains. Another factor to take into account is the use of modified-live vaccines, which have the potential to spread FCV strains and aid in the emergence of immune-evasive variations. Antibody testing has limited predictive value for protection because it might not match real-world field exposure. It is advised that healthy cats who have recovered get vaccinated. Considering MDA and risk factors, the vaccination approach varies for older cats and kittens. There is disagreement over the best intervals between revaccinations; Triennial boosters are advised in low-risk situations, while annual boosters are advised in high-risk situations (52).

Conclusion

The highly contagious feline calicivirus can cause oral diseases and respiratory infections in cats, ranging from mild to severe. It frequently infects young cats and is particularly prevalent in shelters and breeding colonies. However, those criteria make the disease more important nowadays, Therefore, control measure strategies and early diagnosis are too important as a control steps.

Conflicts of interest

The authors declare that there is no conflict of interest.

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فيروس كاليسيفيروس القطط: مراجعة شاملة

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الخلاصة

فيروس كاليسيفيروس القطط هو فيروس الحمض النووي الريبي مطفرة للغاية التي كثيرا ما توجد في القطط المنزلية. إنه مضيف خاص بعائلة السنوريات ولديه الحد الأدنى من الإمكانات الحيوانية المنشأ. يظهر فيروس التهاب الكبد الوبائي درجة عالية من التنوع الجيني والمستضدي في مجموعات القطط. الحاصة ، الجلدية ، عن طريق الفم ، الصيوان ، ناريس ، التهاب الجلد الناخر مع القشور المصلية ، أمراض الجهاز التنفسي العلوي، تقرحات لغوية ، التهاب اللثة ، ومتلازمة العرج هي بعض من علامات خبيثة النظامية فيروس كاليسيفيروس القطط العدوى. تساهم الأعراض الأخرى مثل الالتهاب الرئوي القصبي ، والوذمة تحت الجلد ، ونخر البنكرياس والكبد والطحال في ارتفاع معدل وفيات القطط المصابة. طريقتان لتشخيص هي عزل الفيروس والنسخ العكسي تفاعل البوليميراز المتسلسل. بسبب التطعيم والأمراض المتفرقة ، عادة ما يكون لدى مجموعات القطط انتشار مرتفع للأجسام المضادة. وبالتالي ، فإن وجود أجسام مضادة محددة لا يعني دائما عدوى مستمرة. على الرغم من عدم وجود أدوية مضادة للفيروسات معتمدة خصيصا لفيروس التهاب الكبد الوبائي ، إلا أن الرعاية الداعمة هي حجر الزاوية في العلاج. ومع ذلك ، فقد أظهرت أدوية مثل نيتازوكسانيد وميزوريبين فعالية مضادة للفيروسات في المختبر. هناك عدة أنواع من اللقاحات المتاحة ، بما في ذلك اللقاحات الحية المعطلة والمعدلة ؛ التطعيم أمر بالغ الأهمية للسيطرة على فيروس التهاب الكبد الوبائي. التطهير الفعال والنظافة الجيدة ضروريان لمنع انتشار فيروس التهاب الكبد الوبائي ، خاصة في البيئات متعددة القطط.

الكلمات المفتاحية: فيروس الكاليسي، قطة، تقرحات، التهاب اللثة والفم.