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SEVERE MULTISYSTEMIC FELINE CALICIVIRUS INFECTION WITH EXTENSIVE ORAL AND DERMATOLOGICAL INVOLVEMENT: A CASE REPORT

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Abstract

Feline calicivirus (FCV) commonly causes upper respiratory and oral disease, but rare cases may involve severe systemic manifestations. This report describes an atypical multisystemic FCV infection in a 5-year-old neutered male cat, marked by mucosal necrosis, peripheral oedema, hepatic dysfunction, and non-regenerative anaemia. Clinical signs included lethargy, anorexia, severe lingual ulceration, and limb swelling, with trauma ruled out radiographically. FCV infection was confirmed by PCR. The cat required intensive hospitalisation with fluid therapy, antibiotics, immunomodulation, vitamin supplementation, and assisted enteral nutrition. Complications included hypoalbuminemia, elevated liver enzymes, ulcerative skin lesions, and non-regenerative anaemia requiring erythropoietin therapy. After three months of multidisciplinary management, the cat made a full clinical recovery. This case highlights the potential severity of systemic FCV and the importance of early, comprehensive treatment.

Key words*: anaemia, calicivirus, oedema, multisystemic, ulceration.*

INTRODUCTION

Feline Calicivirus (FCV) is a highly prevalent, non-enveloped, single-stranded RNA virus of the family *Caliciviridae* and a major contributor to feline upper respiratory tract disease worldwide (Radford et al., 2007; Sykes et al., 2014). Marked genetic heterogeneity and prolonged environmental survival facilitate viral persistence in both household and multi-cat settings, despite widespread vaccination (Radford et al., 2009).

Typical FCV infection manifests as oral ulceration, upper respiratory signs, and transient lameness associated with synovitis; however, clinical outcomes are heterogeneous, ranging from subclinical infection to chronic inflammatory oral disease and persistent carrier states (Hurley and Sykes, 2003; Lommer and Verstraete, 2003; Radford et al., 2007).

Rarely, highly virulent systemic variants of FCV give rise to virulent systemic feline calicivirus (VS-FCV) disease, a fulminant syndrome characterised by acute fever, peripheral oedema, cutaneous ulceration, icterus, coagulopathies, and multi-organ failure, with reported mortality rates exceeding 30-50% in outbreak settings (Pedersen et al., 2000; Hurley et al., 2004; Coyne et al., 2006). The pathogenesis of this entity remains incompletely elucidated and is believed to reflect strain-specific virulence rather than predictable host factors (Radford et al., 2007).

Because no targeted antiviral therapy exists, survival depends on early recognition and aggressive, multidisciplinary supportive care (Hurley and Sykes, 2003; Sykes et al., 2014).

Here, we report an atypical multisystemic FCV infection in an adult cat with prolonged hospitalisation and full clinical recovery, underscoring the diagnostic complexity and therapeutic intensity required to successfully manage severe systemic disease.

MATERIALS AND METHODS

A 5-year-old neutered male domestic shorthair cat (body weight: 7.1 kg) was presented with a 3-day history of progressive lethargy, anorexia, hypersalivation, swelling, and pain in the humero-radio-ulnar joint of the front left limb. The cat had indoor and outdoor access and was reportedly vaccinated annually with a modified-live core vaccine, including feline calicivirus. No recent trauma, toxin exposure, or dietary changes were reported.

At presentation, rectal temperature was 39.5°C (reference interval [RI]: 38.0-39.2°C). The patient was estimated to be 5-7% dehydrated based on mucous membrane tackiness and skin turgor. Heart rate was 180-190 bpm (RI: 140-220 bpm), and respiratory rate was 40 breaths/min (RI: 20-30 breaths/min).

Severe ulcerative lesions were observed affecting the ventral and lateral aspects of the tongue, with surrounding mucosal necrosis (Figure 1). Mild peripheral lymphadenomegaly was detected. No cardiac abnormalities or pulmonary crackles were auscultated.

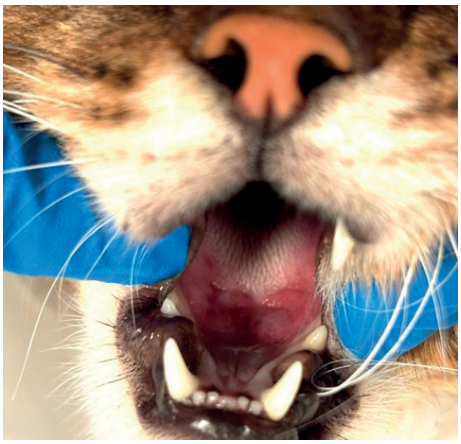


Figure 1. Severe lingual ulceration and mucosal necrosis at initial presentation (5-year-old DSH cat, VS-FCV)

Thoracic and appendicular radiographs were obtained using standard orthogonal views. No fractures, luxation, or osteolytic changes were identified (Figure 2). Soft tissue swelling was evident without underlying skeletal abnormalities.

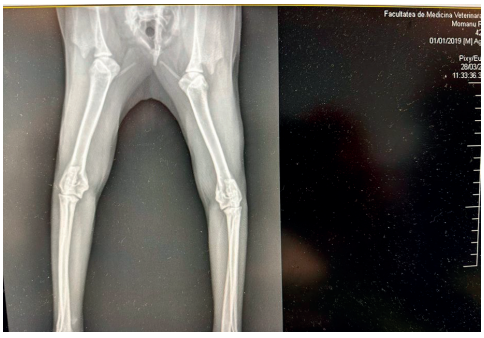


Figure 2. Orthogonal radiographic views of the thoracic limbs showing soft tissue swelling without skeletal abnormalities

Abdominal ultrasonography revealed mild hepatomegaly and diffusely hypoechoic hepatic parenchyma. No free abdominal fluid was detected. Oropharyngeal swabs were collected using sterile nylon swabs. Samples were submitted to a reference diagnostic laboratory for molecular confirmation of feline calicivirus infection. Real-time polymerase chain reaction (RT-PCR) was performed on oropharyngeal and conjunctival swabs using a commercial assay targeting conserved regions of the FCV capsid gene (VP1). FCV RNA was detected, with amplification curves consistent with a positive result, confirming active infection at the time of sampling.

Given the severity and multisystemic nature of the clinical presentation, the diagnostic approach was guided by the need to differentiate between the main viral agents associated with oral and systemic disease in cats.

Feline herpesvirus type 1 (FHV-1) was initially considered due to its common involvement in feline upper respiratory tract disease. However, the absence of characteristic ocular lesions, such as keratoconjunctivitis and corneal ulceration, and the lack of a predominantly oculo-nasal clinical pattern made this diagnosis less likely.

Feline immunodeficiency virus (FIV) was also a key differential diagnosis due to its well-recognised association with chronic oral disease, including gingivostomatitis and periodontitis, and its role in progressive immunosuppression with secondary infections. However, the temporal pattern of disease was not compatible with FIV infection, as FIV-associated oral pathology is typically chronic and progressive, whereas this case presented as an acute, rapidly developing multisystemic ulcerative syndrome with extensive dermatological involvement. In addition, FIV infection was excluded based on a negative enzyme-linked immunosorbent assay (ELISA) result.

Following systematic evaluation of these differentials, the detection of FCV RNA by RT-PCR, in combination with the acute multisystemic clinical phenotype and the exclusion of FHV-1 and FIV, supported feline calicivirus as the most probable primary aetiological agent in this case.

A complete blood count (CBC) at admission (day 0) was performed (Table 1).

Table 1. Complete blood count (CBC) parameters at the time of admission (Day 0) in a cat with suspected systemic feline calicivirus

Parameter	Reference interval	Results
Haematocrit	30.3-52.3%	36.8
Haemoglobin	9.8-16.2 g/dL	11.3
RBC	6.54-12.20 M/µL	8.41
Reticulocytes	3.0-50.0 K/µL	26.1
WBC	2.87-17.02 K/µL	21.24
Neutrophils	2.30-10.29 K/µL	15.43
Lymphocytes	0.92-6.88 K/µL	5.21
Platelets	151-600 K/µL	151

The patient was hospitalized in an isolation ward and managed with intensive supportive therapy. Intravenous isotonic crystalloid solution (Lactated Ringer's Solution) was administered at an initial rate of 4 mL/kg/h for correction of estimated dehydration (5-7%) and maintenance requirements. Fluid therapy was subsequently adjusted according to body weight, urine output, perfusion parameters, and serial electrolyte measurements.

Empirical broad-spectrum antimicrobial therapy was initiated due to the presence of extensive mucosal ulceration and risk of secondary bacterial infection. Amoxicillin–clavulanic acid was administered subcutaneously at 25 mg/kg every 24 hours. Treatment duration was 10 days and was reassessed based on clinical progression and laboratory findings (Table 1).

Analgesic management consisted of buprenorphine administered at 20 mcg/kg intravenously every 12 hours and meloxicam given at 0.1 mg/kg every 24 h subcutaneously. Pain was assessed using a validated feline pain scoring system (Glasgow Composite Measure Pain Scale - Feline), and analgesic dosing was adjusted accordingly. Due to persistent anorexia and severe lingual ulceration impairing voluntary food intake, a nasal feeding tube was placed. Enteral nutrition was initiated using a commercially available recovery diet at 25-33% of the calculated resting energy requirement (RER) on day 1 and progressively increased to full caloric requirements over 5 days. In addition to intensive supportive care, the patient received parenteral vitamin supplementation consisting of B-complex vitamins, including thiamine (vitamin B1, 10 mg/kg), pyridoxine (vitamin B6, 10 mg/kg), and cyanocobalamin (vitamin B12, 0.5 mg/kg), administered to support metabolic function, appetite recovery, and neurological and haematopoietic homeostasis during the acute phase of disease.

The diet was supplemented with an immunonutritional support formulation containing polysaccharides such as arabinogalactan, which exerts immunomodulatory effects by enhancing natural killer (NK) cell activity and also extracts from *Ganoderma lucidum* (Reishi mushroom), which have demonstrated potent immunomodulatory properties through the stimulation of lymphocyte activity. Local management of oral ulcerations included gentle cleansing of the oral cavity and topical application of a gel containing lactoferrin and chlorhexidine, intended to reduce bacterial load, limit secondary infection, and promote mucosal healing. Lactoferrin modulates the host immune response by enhancing the activity of neutrophils, macrophages, and natural killer (NK) cells, while regulating cytokine production to reduce excessive inflammation. It has been shown to support mucosal immunity and promote tissue repair and epithelial healing, particularly in inflamed or ulcerated tissues. Hypoalbuminemia was monitored through serial serum biochemistry analyses. Nutritional support was optimized, and fluid therapy was adjusted to avoid exacerbation of peripheral oedema. Hepatoprotective supplementation (e.g., S-adenosylmethionine) was introduced following detection of elevated hepatic enzyme activity. Progressive non-regenerative anaemia prompted the initiation of recombinant human erythropoietin at 100 IU/kg subcutaneously every 48 hours, in combination with iron supplementation. Hematologic parameters were monitored weekly to assess response and detect potential adverse effects.



Figure 3. Progression of vasculopathic lesions on the distal limbs, characterized by focal epidermal necrosis and marked peripheral oedema

Progressive ulcerative and necrotic cutaneous lesions developed during hospitalization,

initially affecting the distal thoracic limbs and subsequently involving all four limbs.
Lesions were characterized by focal epidermal necrosis, sero-cellular exudation, and peripheral oedema (Figures 3 and 4).
Wound management was conducted under aseptic conditions and followed principles of moist wound healing. Lesions were gently irrigated with sterile isotonic saline solution to remove surface debris and exudate. Mechanical debridement was limited to clearly demarcated non-viable tissue in order to preserve viable granulation tissue.



Figure 4. Extensive digital pad necrosis and ulcerative dermatitis affecting the thoracic limbs

Topical treatment consisted of medical-grade honey–based ointment (L-Mesitran® Soft). L-Mesitran contains standardized medical honey enriched with hypoallergenic lanolin, polyethylene glycol, propylene glycol, and vitamins C and E. The high osmolarity of honey promotes autolytic debridement and reduces local oedema through osmotic fluid shift, while its low pH and hydrogen peroxide–mediated activity contributes to broad-spectrum antimicrobial effects. Additionally, honey has been shown to stimulate angiogenesis, fibroblast proliferation, and epithelialization, thereby supporting granulation tissue formation and wound contraction (Vogt et al., 2021). A sterile non-adherent contact layer was applied over the ointment, followed by secondary absorbent padding and conforming bandage material. Bandages were changed every 48 hours, or earlier in cases of saturation, leakage, or contamination. At each bandage change, wound surface area, degree of exudation, presence of necrotic tissue, and granulation quality were assessed and documented. As lesions progressed to involve all four limbs, care was taken to avoid excessive compression in order to prevent vascular compromise in the context of peripheral oedema. Limb perfusion and distal temperature were monitored at each dressing change.
Systemic antimicrobial therapy and analgesia were continued concurrently, and nutritional support was maintained to optimize tissue repair. Progressive epithelialization and reduction of inflammatory oedema were observed over subsequent weeks, and no surgical wound closure was required (Figure 5).



Figure 5. Clinical resolution of full-thickness cutaneous ulceration following protocolized topical medical-grade honey therapy (5-year-old DSH cat, recovery phase)

RESULTS AND DISCUSSIONS

Following admission, the patient demonstrated rapid clinical deterioration characterized by progression from lingual ulceration and pyrexia to peripheral oedema and multifocal ulcerative dermatitis. Within the first two weeks of hospitalization, erythematous distal limb lesions evolved into focal epidermal necrosis and full-thickness ulceration, ultimately affecting all four limbs (Figures 3-5). The anatomical distribution, bilateral symmetry, and absence of radiographic evidence of trauma supported a systemic inflammatory and vasculopathic process rather than mechanical injury.
Virulent systemic feline calicivirus (VS-FCV) has been described as a severe manifestation of FCV infection, characterized by cutaneous ulceration, facial and limb oedema, hepatic dysfunction, and high mortality rates in outbreak settings (Pedersen et al., 2000; Hurley et al., 2004; Coyne et al., 2006). The cutaneous lesions observed in the present case - particularly distal extremity necrosis and digital pad involvement - are consistent with previously reported vasculopathic and endothelial injury patterns associated with highly virulent FCV strains (Hurley et al., 2004; Radford et al., 2007) (Table 2).

Serial hematologic monitoring revealed a progressive decline in haematocrit from 36.8% to a minimum value of 22.6%, consistent with non-regenerative anaemia. Reticulocyte counts remained low during the acute phase, indicating insufficient bone marrow response. Non-regenerative anaemia in systemic viral infections may be multifactorial, potentially involving inflammatory cytokine-mediated suppression of erythropoiesis, transient bone marrow suppression, or anaemia of inflammatory disease (Weiss and Goodnough, 2005). Gradual hematologic recovery following erythropoietin administration suggests reversible marrow suppression rather than irreversible aplasia (Figure 6).

Table 2: Comparative analysis of clinical manifestations between the present case and reported literature for virulent systemic feline calicivirus (VS-FCV)

Parameter	Reported VS-FCV (Literature)	Present Case
Fever	Common	Yes
Peripheral oedema	Frequent	Yes
Cutaneous ulceration	Reported	Severe, 4 limbs
Hepatic involvement	Variable	Elevated enzymes
Anaemia	Occasionally reported	Non-regenerative
Mortality rate	30-50%	Survival
Outbreak setting	Common	Isolated case

The clinical presentation of the present case shows several similarities with virulent systemic feline calicivirus (VS-FCV) reported in the literature. Fever, peripheral oedema, and cutaneous ulceration are commonly described features of VS-FCV infection and were also present in this case, consistent with previously reported outbreak-associated cases (Pedersen et al., 2000; Hurley et al., 2004; Coyne et al., 2006). Hepatic involvement has been variably reported in severe systemic infections and was reflected in this case by increased liver enzyme activity. Anaemia has been occasionally described in VS-FCV cases, although it is not considered a consistent clinical feature; in the present case, a non-regenerative anaemia was identified. A central pathogenic mechanism is generalised vascular endothelial damage, which results in increased vascular permeability, capillary leakage, and tissue oedema. This explains the frequent clinical findings of peripheral oedema, facial swelling, and cutaneous ulceration. Histopathological studies have demonstrated systemic neutrophilic vasculitis and fibrinoid vascular necrosis, suggesting that endothelial injury is a key driver of disease severity (Pedersen et al., 2000; Hurley et al., 2004).

The resulting capillary leak syndrome leads to hypovolaemia, hypoperfusion, and secondary shock, which can progress to multi-organ failure. In parallel, epithelial necrosis in multiple tissues (oral cavity, skin, and occasionally internal organs) reflects both direct viral effects and immune-mediated damage (Hurley et al., 2004; Radford et al., 2007).

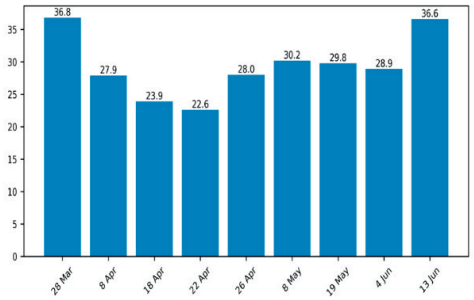


Figure 6. Temporal evolution of haematocrit (%) in a cat diagnosed with systemic feline calicivirus infection over a three-month monitoring period

Biochemical analysis demonstrated progressive hypoalbuminemia concurrent with the development of peripheral oedema. Albumin is the principal determinant of plasma colloid oncotic pressure; therefore, decreased serum albumin concentration likely contributed to interstitial fluid accumulation (DiBartola, 2011). In systemic inflammatory states, hypoalbuminemia may result from decreased hepatic synthesis secondary to acute-phase protein redistribution, increased vascular permeability, and enhanced extravascular protein loss (Radford et al., 2007). The temporal correlation between declining albumin levels and worsening oedema in this patient supports a pathophysiologic contribution of oncotic imbalance in addition to inflammatory endothelial dysfunction (Figure 7.).

Serial monitoring revealed a concurrent decline in haematocrit and serum albumin during the

acute phase of disease. Haematocrit decreased from 36.8% to 22.6% (38.6% relative reduction), while albumin reached a minimum value of 1.9 g/dL, below the reference interval (2.2-4.4 g/dL). The lowest albumin concentration temporally coincided with maximal peripheral oedema and progression of distal limb lesions.

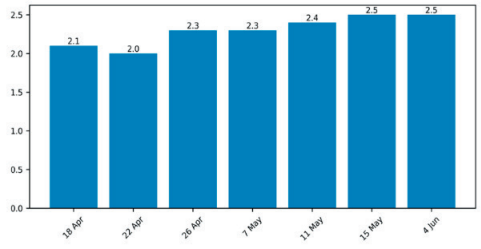


Figure 7. Fluctuations in serum albumin concentration (g/dL) in relation to the clinical development of peripheral oedema

This parallel deterioration supports a systemic inflammatory process. Pro-inflammatory cytokines are known to suppress hepatic albumin synthesis and inhibit erythropoiesis, contributing to hypoalbuminemia and anaemia of inflammation (Weiss & Goodnough, 2005). Increased vascular permeability likely facilitated transcapillary albumin loss, reducing plasma oncotic pressure and promoting interstitial fluid accumulation (Table 3).

Biochemical improvement preceded full hematologic recovery, consistent with restoration of hepatic synthetic function prior to bone marrow regenerative response. These findings indicate coordinated inflammatory, hepatic, and hematopoietic involvement in severe systemic FCV infection and support the reversibility of inflammation-mediated dysfunction under sustained supportive therapy.

Despite the severity of multisystemic involvement, the patient achieved complete clinical recovery following prolonged intensive supportive management. Mortality rates in reported VS-FCV outbreaks have ranged from 30% to over 50% (Hurley et al., 2004; Coyne et al., 2006), underscoring the clinical significance of this favourable outcome. The successful resolution of full-thickness limb ulcerations without surgical intervention further emphasizes the importance of optimized wound management, including the use of medical-grade honey-based topical therapy, which has demonstrated antimicrobial, anti-inflammatory, and pro-angiogenic properties in veterinary wound care (Molan, 2001; Dart et al., 2015).

Table 3. Chronological correlation between clinical progression, hematologic shifts (HCT), and biochemical markers (ALB, ALT)

Time Point	Clinical Findings	HCT (%)	ALB (2.2-4.0 mg/dl)	ALP (14-111 U/L)	Comments
Admission	Lingual ulcer	36.8	2.4	170	Stable
Week 2	Limb oedema	23.9	2.1	215	Anaemia progressing
Week 3	Full limb lesions	22.6	1.9	255	Minimum value
Week 8	Healing phase	30.2	2.0	218	Recovery

HCT – haematocrit; ALB – albumin; ALT – alanine aminotransferase.

This case illustrates the complex interplay between systemic inflammation, vascular permeability alterations, oncotic imbalance, and secondary tissue injury in severe FCV infection. Moreover, it highlights that early recognition, aggressive supportive therapy, and sustained multidisciplinary monitoring can significantly improve prognosis, even in cases exhibiting features consistent with virulent systemic disease.

CONCLUSIONS

This case highlights the potential for severe multisystemic involvement in feline calicivirus infection, extending beyond the classical presentation of upper respiratory and oral disease. The progression to peripheral oedema, full-thickness cutaneous ulceration, hepatic dysfunction, and non-regenerative anaemia

underscores the complex pathophysiology associated with virulent systemic FCV variants. Early recognition of systemic manifestations, close hematologic and biochemical monitoring, and aggressive multidisciplinary supportive management were critical to achieving a favourable outcome in this case. The temporal association between hypoalbuminemia and peripheral oedema further emphasizes the importance of understanding oncotic and inflammatory mechanisms in severe viral disease.

Despite features consistent with virulent systemic FCV, prolonged intensive therapy resulted in complete clinical recovery without surgical intervention. This case reinforces the necessity for prompt diagnosis and sustained therapeutic support in managing severe FCV-associated systemic disease.

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