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Virulent Systemic Feline Calicivirus Infection in a Kitten

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Abstract : A 5 month old Korean domestic short haired male kitten (weighing 1.7 kg) was presented with primary complaints of upper respiratory disease (URD) signs and skin ulceration and edema on face, feet and footpad with lameness. Diagnostic test revealed leukopenia, lymphopenia, pancreatitis and feline calicivirus (FCV) infection. Diagnosis of virulent systemic FCV (VS-FCV) was made on clinical signs, isolation of calicivirus via PCR and exclusion of other causes of acute upper respiratory disease. Therapeutic strategies were directed to lessen URD signs and to treat secondary bacterial infection and antiviral infection. One month after this therapy, skin lesions on face and feet and URD signs were much improved, although the lameness persisted mildly. To author's best knowledge, this is the first case reporting VS-FCV infection in a kitten in Korea.

Key words **:** feline calicivirus, virulent systemic FCV, upper respiratory disease, nasal discharge, cat.

Introduction

Feline calicivirus (FCV) is single-stranded RNA virus, which is responsible for upper respiratory disease in cats (3). The usual field strains of FCV causes relatively mild symptoms. However, lethal variant of FCV, called as virulent systemic FCV (VS-FCV; recently isolated in USA and some European countries) causes an acute and severe syndrome with mortality rates ranging between 33% and 60% (1,4,6,8,9). Clinical features of VS-FCV infection include profound fever, anorexia, marked subcutaneous edema (especially of the limbs and face), icterus, alopecia, crusting, and ulceration of the nose, lips, pinnae, and feet, along with the common clinical signs of infection from usual field FCV strain (e.g. ulceration and nasal or ocular discharge) (1,4,6,8,9). This case report described unusual infection of VS-FCV infection in a kitten.

Case

A 5 month old Korean domestic short haired male kitten (weighing 1.7 kg) was presented with primary complaints of anorexia, fever, nasal/ocular discharge, alopecia/crusting/ulceration of the nose, lips, pinnae, and feet, marked subcutaneous edema, and lameness. The kitten was vaccinated twice before presentation and had medical history of upper respiratory disease (URD) partially responsive to doxycycline and famciclover at the age of 4 month. On physical examination, the kitten was weak, depressed and wasting (body conformation score 1/5). The kitten was limping due to hind limb lameness, and was febrile (40.5°C), tachycardic (180 beats per min) and open mouth breathed (by severe nasal discharge). His systolic blood pressure was 150 mmHg (measured by Doppler detector). Laboratory tests revealed leukopenia (4.4×10^9/L, reference interval [RI] 5.5-19.5 $\times 10^9$/L), lymphopenia (0.5×10^9/L, RI 1.2-3.2 $\times 10^9$/L), decreased hemoglobin (8.6 g/dL, RI 9-15 g/dL) and hematocrit (27%, RI 30-47%), increased serum amylase (1500 U/L RI 300-1100 U/L), and decreased blood urea nitrogen (9 mg/dL, RI 10-30 mg/dL). Feline pancreatic lipase immonoactivity (fPLI) test was positive, suggesting pancreatitis. There was edema on the face and 4 limbs (Fig 1A and C). There were alopecia, crusting, and ulceration of the nose, lips, pinnae, feet and foot pad (Fig 1B and D). Skin impression on ulceration region of the face found predominant infiltration neutrophils with fewer macrophages and dead epithelial cells and Gram negative rod bacteria. Nasal discharges were mucopurulent and were cytologically consisted of degenerate neutrophils and macrophages with cell debris. The nasal discharge was submitted for PCR detection of feline upper respiratory pathogens to a commercial lab (IDEXX, Korea), and found negatives for *Chlamydophila felis*, *Bordetella bronchiseptica, Mycoplasma felis*, feline herpesvirus (FHV), H1N1 influenza virus, but positive for feline calicivirus (FCV; Fig 3). In addition, the kitten was negative for feline immune deficiency virus (FIV) and feline leukemia virus (FeLV) on an in-house diagnostic test (SNAP®, IDEXX, USA). Diagnosis of VS-FCV was made on clinical signs, isolation of calicivirus via PCR and exclusion of other causes of acute upper respiratory disease (e.g., FHV, *Chlamydophila felis*).

Initial therapeutic strategies were directed to lessen URD signs (Oxymetazoline hydrochloride drops; Afrin Pediatric Nasal Drops®, USA) and to treat secondary bacterial infection (doxycycline, 5 mg/head, PO, q12hr; Vibravet, Pfizer, USA) and antiviral infection (famciclover 15 mg/head, PO, q12hr; Famvir, Novartis, USA). The skin ulcer was treated with saline/povidone lavage). Despite this treatment, clinical signs

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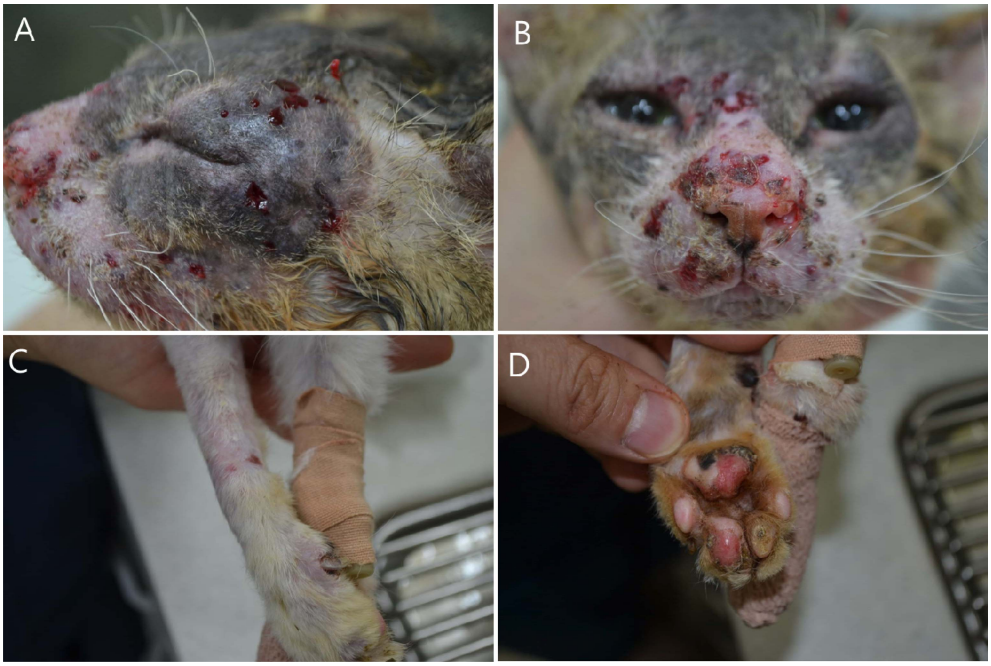

Fig 1. Skin lesions on the first presentation. A: There was marked facial edema with alopecia, crusting, and ulceration of the nose, lips and pinnae. B: Marked ulceration was noticed on nose and lips. C: There was mild limb edema with alopecia, crusting, and ulceration of feet and toes. D: Marked ulceration was noticed on footpad.

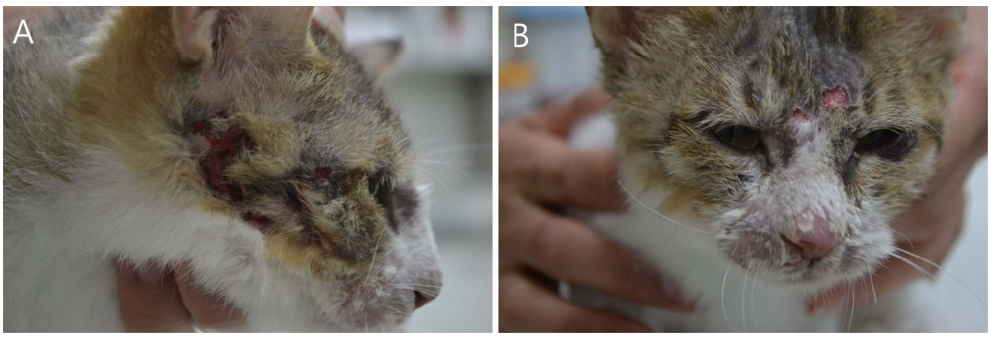

Fig 2. Skin lesions after 1 month of therapy. A: Skin lesions (including edema) on face were much improved. B: Ulcerations on nose and lips were much improved.

were poorly improved except URD signs. Therefore, doxycycline was changed to azithromycin 10 mg/kg, PO, q24hr PO; Zithromax, Pfizer, USA) and dose of famciclover was increased to 60 mg/head, PO, q12hr. Because the kitten started to show semi-moist stool, oral metronidazole (20 mg/kg, PO, q12hr) was co-administered. To prevent cachexia, we forced to feed the kitten with a commercial diet (a/d, Hills, USA) with a multi-vitamin tablet. Later we stopped administering famciclover, because the PCR test ruled out FHV infection. One month after this therapy, skin lesions (including edema) on face and feet and URD signs were dramatically improved (Fig 2A and B), although the lameness persisted mildly. The kitten was eating well without forced feeding.

Discussion

The pathogenesis of VS-FCV is quite different from usual strain of FCV (1). The VS-FCV is characterized by widespread vasculitis, multiorgan failure and high mortality in cats (1,4,6,8,9), although the exact the pathogenesis of VS-FCV infection is still under the investigation. Viral evolution, immune-mediated components, and environmental and management factors were suggested in the pathogenesis of VS-FCV infection (3). Diagnosis of VS-FCV is challenging, because there is no way to differentiate VS-FCV strain from field FCV strains. Therefore, the diagnosis is based on progression of disease (more severe disease) and severe clinical signs from vasculitis (including marked edema and/or extensive hair loss, oozing and ulceration of the skin), isolation of calicivirus via PCR and exclusion of other causes of acute upper respiratory disease. In this case of kitten, we firstly suspected FCV infection complicated with FHV and other secondary bacterial infections. However, the PCR testing for upper respiratory pathogens led us to rule out concurrent infection. Then we suspected FIV/FeLV infection, which cause immune suppression in cats. Surprisingly, this kitten was also negative for FIV/FeLV infection. The clinical presentation of this kitten was closely similar to those in VS-FCV infection. The cat was febrile and limping with severe ulceration and edema on face, feet and footpad. Those signs were due to

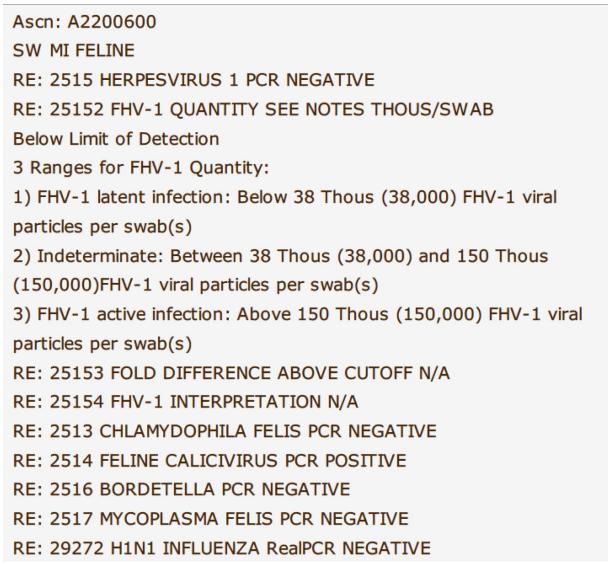
Ascn: A2200600
SW MI FELINE
RE: 2515 HERPESVIRUS 1 PCR NEGATIVE
RE: 25152 FHV-1 QUANTITY SEE NOTES THOUS/SWAB
Below Limit of Detection
3 Ranges for FHV-1 Quantity:
1) FHV-1 latent infection: Below 38 Thous (38,000) FHV-1 viral particles per swab(s)
2) Indeterminate: Between 38 Thous (38,000) and 150 Thous (150,000)FHV-1 viral particles per swab(s)
3) FHV-1 active infection: Above 150 Thous (150,000) FHV-1 viral particles per swab(s)
RE: 25153 FOLD DIFFERENCE ABOVE CUTOFF N/A
RE: 25154 FHV-1 INTERPRETATION N/A
RE: 2513 CHLAMYDOPHILA FELIS PCR NEGATIVE
RE: 2514 FELINE CALICIVIRUS PCR POSITIVE
RE: 2516 BORDETELLA PCR NEGATIVE
RE: 2517 MYCOPLASMA FELIS PCR NEGATIVE
RE: 29272 H1N1 INFLUENZA RealPCR NEGATIVE

Fig 3. Test result of PCR detection of feline upper respiratory tract infection.

vasculitis, which was unusual in general field strains of FCV. According to literature, gold standard of diagnosis of VS-FCV is the immunohistochemical examination of tissues demonstrating the presence of the virus in the viscera and its association with lesions (3). However, the kitten was survived from the infection, and thus we could not precede this test.

Clinical presentation from VS-FCV infection is responsible from the combination of epithelial (cytolytic) and endothelial injury (5). Although it is rare, it often causes severe respiratory distress, jaundice and gastrointestinal signs (4). Clinical signs are generally more severe in adults (4). Because our case was young kitten, he might survive from VS-FCV infection.

Since there is no definitive cure for FCV infection, severely affected cats with VS-FCV are generally treated with supportive care (e.g. fluid therapy, antibiotics) and immune modulating drugs (e.g. steroids and interferon). To date, no controlled clinical studies have been evaluated for specific treatment for VS-FCV. Although antiviral drugs such as ribavirin were suggested to be effective for inhibit FCV replication, the drug is not recommended for treatment, because it is very toxic to cats (7). Although several studies found feline interferon-ω could inhibit FCV replication *in vitro* (3,10), no controlled studies have yet been published, to date.

Unfortunately, commercial vaccines against FCV do not provide full protection against infection. They can only lessen the severity of disease. Recently, Boeringer-Ingelheim produces a vaccine incorporated two strains of calicivirus (traditional FCV strain plus VS-FCV strain; 2). Unfortunately, there is no evidence that this new vaccine can provide better protection against VS-FCV. It only provides broader cross protection against all FCV strains.

In conclusion, the case presented here had typical feature of VS-FCV infection in a cat. Although the diagnosis was challenging, we made a diagnosis based on typical clinical presentation of VS-FCV infection, isolation of calicivirus via PCR and exclusion of other causes of acute upper respiratory disease. To author's best knowledge, this is the first case reporting VS-FCV infection in a kitten in Korea.

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새끼고양이에서 발생한 고독성 칼리시바이러스(VS-FCV) 감염증

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요 약 : 5개월령 코리안 숏헤어 새끼 고양이(몸무게 1.7 kg)가 상부호흡기 증상, 얼굴과 발/발바닥의 궤양과 부종 및 파행을 주증으로 내원하였다. 실험실 검사상, 백혈구감소증, 림프구 감소증, 췌장염 및 칼리시바이러스 감염증이 확인되었다. 진단은 고독성 칼리시바이러스 감염증에 대한 특이 임상증상과 PCR을 통한 칼리시바이러스 동정 및 다른 상부호흡기 감염을 배제함으로써 내려졌다. 상부호흡기 증상완화와 이차 감염에 대한 처치 및 보조요법을 약 한 달간 실시하였고, 환자는 이러한 치료를 통해 임상증상이 크게 개선되었다. 본 증례는 고독성 고양이 칼리시바이러스에 대한 첫 번째 발병증례이다.

주요어 : 고양이 칼리시바이러스, 고독성 칼리시바이러스, 상부호흡기 질환, 비루, 고양이