

The second method of a third eyelid flap is less frequently used. It has the advantage of allowing the nictitans to move with the eyeball, minimizing motion between corneal epithelium and conjunctiva. Its disadvantage is its tendency to pull the sutures through the bulbar conjunctiva. Method: three to four horizontal mattress sutures are placed through the horizontal part of the cartilage and the dorsal bulbar conjunctiva. A soft suture material is always used (e.g. silk 5-0) and the knots are tied at the palpebral side of the nictitans.

Sutures are left in place for 7 to 14 days or even longer if the situation allows it. When placed correctly, a nictitans flap is well tolerated by dogs and cats. An Elisabethan collar is advisable for at least the first days.

In both methods, the bulbar side of the nictitans can be debrided, so that blood vessels are placed directly onto the corneal surface. This can be advantageous to the healing process. The conjunctiva will be firmly attached to the cornea when the sutures are removed, dissection will be necessary.

CONJUNCTIVAL FLAPS

Conjunctival flaps are used in corneal abrasions, ulcers, descemetocoeles and minor lacerations.

They provide a better vascularisation of the underlying tissue than third eyelid flaps, but require more surgical experience. Conjunctival pedicle grafts are sutured to the cornea, using 8-0 to 10-0 material, require a high magnification, microsurgical instruments and a surgeon trained in corneal surgery. They lie beyond the scope of this paper.

A 360° fornix-based conjunctival flap is easier to perform. General anaesthesia is required. Using tenotomy scissors, the bulbar conjunctiva is dissected at the limbus. The conjunctiva is undermined for approx. 5 millimetres and pulled over the cornea. Horizontal mattress sutures (3 to 5) of 5-0 or 6-0 silk are placed. The sutures should be tied in a way that the suture material has no contact with the cornea, to prevent abrasions.

The cornea is covered by conjunctiva completely. The sutures can be left in place for 12 to 21 days, then they are removed. If the conjunctiva retracts prematurely, holes will appear. These can be sutured under local anaesthesia. After removal of the sutures, the conjunctiva will retract spontaneously from initially intact cornea. Conjunctival tissue will be adhered to corneal defects. At first, blood vessels will be present. Later, these will retract and the tissue will become translucent.

FOLLICULAR CONJUNCTIVITIS

In most younger dogs, an enlargement of lymphoid follicles is present at the bulbar surface of the nictitans. This can be regarded as a normal reaction to antigenic stimuli such as viruses and bacteria. A mucoid discharge can be present, but discomfort is absent. In more severe cases, the palpebral side of the nictitans and the palpebral and scleral conjunctiva are affected. These dogs can be slightly photophobic and more mucoid discharge is present. Spontaneous regression is common. Follicular conjunctivitis can also be in concurrence with allergic skin diseases or chronic irritation of the eye (e.g. entropion). If treatment is necessary, it should be focused on the primary cause. Topical corticosteroids can be helpful, but it may take several weeks until some regression can be noticed. In severe cases, removal of the follicles at the bulbar side of the nictitans by careful resection is possible. Electrocautery can be done, but requires magnification and a steady hand, since the cornea is near. Post-operatively topical corticosteroids are applied for 2 to 3 weeks, together with an antibiotic ophthalmic ointment in the first week. Thermocautery is not suitable, it causes a painful and extreme swelling of the conjunctival membranes.

REFERENCES

1. Gelatt KN (Ed). *Veterinary Ophthalmology*. 2nd edition, Lea & Febiger, Philadelphia 1991.
2. Slatter DH. *Fundamentals of Veterinary Ophthalmology*. 2nd edition, Saunders, Philadelphia 1990.
3. Slatter DH. *Textbook of Small Animal Surgery*. Saunders, Philadelphia 1985.

THE DIAGNOSIS AND EPIDEMIOLOGY OF FELINE IMMUNODEFICIENCY VIRUS (FIV) INFECTION IN THE NETHERLANDS

Herman F. Egberink¹, Paula Hendriks¹, Robert J. Slappendel², and Marian C. Horzinek¹

INTRODUCTION

In 1987, the discovery of a new retrovirus isolated from diseased cats was reported. Based on its morphology, biochemical characteristics, nucleotide sequence homology and antigenic cross-reactivities the virus was classified as a member of the lentivirus genus (1-7). In the first report it was referred to as feline T-lymphotropic lentivirus (FTLV), but it is now known as feline immunodeficiency virus (FIV). Since it causes an immunodeficiency syndrome in cats, the virus is of considerable

veterinary importance. Also, it shares many physical and biological properties with HIV, the causative agent of AIDS in man. Besides the veterinary importance, its potential model character has made this virus the most extensively studied feline pathogen during the last years.

During FIV infection a transient primary illness can be recognized with fever, neutropenia and lymphadenopathy, followed by a long period of clinical latency (8). During this stage impairment of immune function develops, eventually leading to an immunodeficiency syndrome (9). The incubation period for this stage can be as long as 5 years. Many FIV infected cats are presented for the first time showing vague signs of illness:

¹ Institute of Virology, Department of Infectious Diseases and Immunology and Department of Clinical Sciences of Companion Animals, Faculty of Veterinary Medicine, Utrecht University, Yalelaan 1, NL-3584 CL Utrecht, the Netherlands

recurrent fevers, emaciation, inappetence, lymphadenopathy, anaemia, leucopenia and behavioral changes. Several signs of chronic secondary or opportunistic infections can be present, e.g. chronic stomatitis/gingivitis, enteritis, upper respiratory tract infections, and infections of the skin (10).

DIAGNOSIS OF FIV INFECTION

As all lentiviruses, FIV causes chronic lifelong infections (11). An infection can be diagnosed by isolation of the virus from the blood. This is possible throughout the course of disease, although isolation during the initial and terminal stages is more often successful as compared to attempts during the period of clinical normality (10). The isolation procedure is very laborious: it can take 1-8 weeks before a culture becomes positive. In contrast to infections with feline leukaemia virus (FeLV), viral antigen cannot be demonstrated in blood cells or in the plasma. Probably insufficient amounts of FIV antigen are circulating, or the antigen is captured in immune complexes. Because of the persistent nature of the infection determination of antibodies is of great diagnostic importance. Cats do not eliminate FIV in the course of the infection, and the presence of antibodies proves that they harbour the virus. Most cats mount an antibody response early after infection and stay seropositive for their entire lives (12). Therefore, FIV is routinely diagnosed by detection of antibodies using an enzyme-linked immunosorbent assay (ELISA) or immunofluorescence assay (IFA) (13,14), both of which are sensitive and specific.

Only few false-positive results can be expected in ELISA and IFA. However, positive test results, especially in cats from low-risk groups (e.g. healthy cats in catteries), should be confirmed using an independent assay principle. In low-risk cats the incidence of FIV infection is expected to be <1%, and the prediction reliability of the ELISA is consequently low. The 'Western blot' is mostly used for confirmation, but it can only be performed in specialized laboratories. Using this assay the antibody response directed against the individual FIV proteins is visualized. Of course, false-negative reactions can also occur. In a previous study we have found only one seronegative, virus-positive cat (1 out of 64 cats tested). Such cats can only be detected using virus isolation or by demonstration of the viral nucleic acid using the 'polymerase chain reaction'.

EPIDEMIOLOGICAL STUDIES

Infections with FIV have been found worldwide. Reported incidences vary between 1% and 15% in healthy animals and between 3% and 44% in diseased cats (15,10). The incidence in the Netherlands was estimated to be about 1% in healthy and 3% in diseased cats, as we have shown before (16). The lower incidence could be due to the presence of a smaller population at risk. Bites seem to be the most efficient and important mode of transmission, which could explain the higher incidence in male, free roaming cats as has been found in many studies (14,17,18). Horizontal transmission through contact alone probably also occurs (19,1), but it is inefficient (14).

In a recent survey we have studied the incidence of FIV in cats presented at the Department of Clinical Sciences of Companion Animals of the Utrecht Veterinary Faculty, in stray cats and in cat shelters. The animals were tested using IFA, and the positive tests confirmed by Western blot. FIV infection was found in

about 4% of the diseased animals. In stray cats, the incidence in different groups ranged between 1 and 20% (mean 4.4%); more infections were found in older male cats in agreement with epidemiological data presented by others. The significance of these figures will be discussed with respect to the different populations that had been studied.

REFERENCES

1. Pedersen NC, Ho EW, Brown ML, and Yamamoto JK. Isolation of a T-lymphotropic virus from domestic cats with an immunodeficiency-like syndrome. *Science* 1987; 235: 790-3.
2. Yamamoto JK, E Sparger, EW Ho, PR Andersen, TP O'Connor, CP Mandell, L Lowenstine, R Munn, and NC Pedersen. Pathogenesis of experimentally induced feline immunodeficiency virus infection in cats. *Am J Vet Res* 1988; 49: 1246-58.
3. Olmsted RA, AK Barnes, JK Yamamoto, VM Hirsch, RH Purcell, and PR Johnson. Molecular cloning of feline immunodeficiency virus. *Proc Natl Acad Sci USA* 1989; 86: 2448-52.
4. Olmsted RA, VM Hirsch, RH Purcell, and PR Johnson. Nucleotide sequence analysis of feline immunodeficiency virus: genome organization and relationship to other lentiviruses. *Proc Natl Acad Sci USA* 1989; 86: 8088-92.
5. Talbott RL, EE Sparger, KM Lovelace, WM Fitch, NC Pedersen, PA Luciw, and JH Elder. Nucleotide sequence and genomic organization of feline immunodeficiency virus. *Proc Natl Acad Sci USA* 1989; 86: 5743-47.
6. Steinman R, J Dombrowski, T O'Connor, RC Montelaro, Q Tonelli, K Lawrence, C Seymour, J Goodness, NC Pedersen, and PR Andersen. Biochemical and immunological characterization of the major structural proteins of feline immunodeficiency virus. *J Gen Virol* 1990; 71: 701-6.
7. Egberink HF. FIV infection: an animal model for AIDS. Ph.D. dissertation Utrecht 1991
8. Ishida T, and I Tomoda. Clinical staging of feline immunodeficiency virus infection. *Jpn J Vet Sci* 1990 52: 645-8.
9. Barlough JE, CD Ackley, JW George, N Levy, R Acevedo, PF Moore, BA Rideout, MD Cooper, and NC Pedersen. Acquired immune dysfunction in cats with experimentally induced feline immunodeficiency virus infection: comparison of short-term and long-term infections. *J Acq Immune Defic Syndr* 1991; 4: 219-27.
10. Pedersen NC, JK Yamamoto, T Ishida, and H Hansen. Feline immunodeficiency virus infection. *Vet Immunol Immunopathol* 1989; 21: 111-229.
11. Narayan O, and JE Clements. Biology and pathogenesis of lentiviruses. *J Gen Virol* 1989; 70: 1617-39.
12. Egberink HF, Keldermans CEJM, Koolen MJM, and MC Horzinek. Humoral immune response to feline immunodeficiency virus in cats with experimentally induced and naturally acquired infections. *Am J Vet Res* 1992; 53: 1133-38.
13. O'Connor TP Jr., S Tanguay, R Steinman, R Smith, MC Barr, JK Yamamoto, NC Pedersen, PR Andersen, and QJ Tonelli. Development and evaluation of immunoassay for detection of antibodies to the feline T-lymphotropic lentivirus (feline immunodeficiency virus). *J Clin Microbiol* 1989; 27: 474-9.
14. Yamamoto JK, H Hansen, EW Ho, TY Morishita, T Okuda, TR Sawa, RM Nakamura, and NC Pedersen. Epidemiologic and clinical aspects of feline immunodeficiency virus infection in cats from the continental United States and Canada and possible mode of transmission. *J Am Vet Med Assoc* 1989; 194: 213-20.
15. Egberink HF, and MC Horzinek. Animal immunodeficiency viruses. *Vet Microb* 1992; 33: 311-31.
16. Lutz H, H Egberink, P Arnold, G Winkler, C Wolfensperger, O Jarrett, AL Parodi, NC Pedersen, and MC Horzinek. Felines T-lymphotropes Lentivirus (FTLV): Experimentelle Infektion und Vorkommen in einigen LAndern Europas. *Kleintierpraxis* 1988; 33: 455-9.
17. Ishida T, T Washizu, K Toriyabe, S Motoyoshi, I Tomoda, and NC Pedersen. Feline immunodeficiency virus infection in cats of Japan. *J Am Vet Med Assoc* 1989; 194: 221-5.
18. Shelton GH, RM Waltier, SC Connor, and CK Grant. Prevalence of feline immunodeficiency virus infections in pet cats. *J Am Anim Hosp Assoc* 1989; 25: 7-12.
19. Hosie MJ, C Robertson, and O Jarrett. Prevalence of feline leukaemia virus and antibodies to feline immunodeficiency virus in cats in the United Kingdom. *Vet Rec* 1989; 125: 293-7.