FELINE LEUKAEMIA VIRUS INFECTION*

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All aspects, including aetiology, incidence, transmission, immunity, clinical signs, pathogenesis, pathology and diagnosis of feline leukaemia virus infection in domestic cats are considered with a bias towards the more clinical aspects for the benefit of private practitioners. Non-neoplastic disease and possible methods of control are also discussed.

INTRODUCTION

The Feline leukaemia virus (FeLV) was discovered in 1964 by Jarrett and his co-workers in his laboratory in Glasgow. It has been shown that the FeLV is responsible for a variety of diseases, both neoplastic (leukaemia and lymphosarcoma) and non-neoplastic, and that it is horizontally transmitted by contact between unrelated cats.

Although the incidence of infection and its manifestation in the overall cat population is relatively low, it becomes a matter of importance in high density households, catteries or breeding establishments where there are large numbers of cats in close contact with one another. If an infected cat is introduced into such an establishment, the virus can spread rapidly with disastrous results.

Concern arose when it was discovered that FeLV could be grown in human tissue culture cells, but to date no evidence has been found that FeLV produces or is associated with any disease in man. The FeLV occurs throughout the world.

AETIOLOGY

The FeLV is a member of the oncovirus (Oncorna) virus group and replicates in the cell cytoplasm. The new virus particles are released from the cell by a process of budding, but the cell is not destroyed by the virus10 11 14. The FeLV grows in many different types of cells in the body not all of which may later become malignant. It can replicate in the epithelial cells of the buccal and nasal cavities, trachea, urinary bladder, intestinal tract, the salivary and mammary glands and the pancreas, as well as in the precursor cells of the lymphoid, erythroid and myeloid series in the bone marrow or lymphoid tissues of the body11.

INCIDENCE

Incidence (as indicated by the presence of serum antibodies to the virus) depends largely on the density of the cat population in any one area and varies from about 50% in large cities where contact is high, to about 5% in rural areas. Incidence is much higher in infected catteries, breeding colonies or high density households. The actual incidence of feline leukaemia, lymphosarcoma or other FeLV-associated diseases is much lower, possibly 0.05% or less of the total cat population, but leukaemia or lymphosarcoma accounts for approximately 20% of all neoplasms in cats, whereas in man they account for only 5% of all neoplastic conditions9 14.

TRANSMISSION

FeLV has been found in the urine, blood, saliva, nasal secretions and milk of infected cats15. Initially it was thought that FeLV was transmitted vertically from one generation to the next. It has since become apparent that the FeLV is readily transmissible between unrelated cats coming into contact with one another. It is thought, however, that there is little danger of transmission of FeLV infection between cats where contact is brief. It appears that contact must be relatively close and of some duration before infection can be established14 15 22.

a) Natural Transmission

(i) The virus may be transmitted by licking, grooming, fighting, contamination of food or water bowls with saliva or litter trays or sand boxes with urine. The virus may be inhaled or ingested. Aerosol transmission is possible14 15.

(ii) Intra-uterine (transplacental) infection seems possible as the virus has been found in newborn kittens where the queen was viraemic15.

(iii) Transmammary infection of nursing kittens has not yet been demonstrated although the virus is excreted in the milk of infected queens15.

(iv) There appears to be little danger of horizontal transmission of FeLV to dogs or man8.

b) Artificial Transmission

(i) This has been achieved using cell-free isolates from lymphosarcoma cats or gradient purified virus, and has resulted in FeLV infection in previously healthy, uninfected cats15.

(ii) FeLV infected blood, transfused into a non-viraemic cat, has resulted in a persistent viraemia in the recipient15.

(iii) The virus has been experimentally transmitted by intra-nasal inoculation15.

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SOURCE OF INFECTION

Any FeLV positive cat (viraemic) is a potential source of infection to any uninfected cat with which it may come into contact. Fortunately the environment is not important as a direct source of infection because the virus will survive only a few days in moist conditions and only a few hours in a dry environment. It is also readily susceptible to common household detergents.

IMMUNITY

Factors which determine the response of a cat exposed to FeLV infection are degree of exposure (the amount of virus and the time period involved) and the immunological competence of the cat, which in turn is influenced by age, nutritional status, general health and individual variation. Kittens are not yet fully immunocompetent and are therefore fully susceptible to FeLV infection and possible ultimate development of lymphosarcoma and other FeLV-associated disease, although it appears that kittens born to immune queens are afforded a degree of protection by transplacental or colostral antibodies derived from the queen. The immune response to FeLV infection in the cat can result in the production of neutralising antibodies to the type-specific viral antigens and/or feline oncornavirus associated cell membrane antigen (FOCMA) antibodies. Neutralising antibodies inactivate the virus. Most cats in the general population, when infected with FeLV, respond by the production of neutralising antibodies and are only transiently viraemic and do not develop leukaemia or lymphosarcoma. Anti-FOCMA antibodies act against the membrane antigens of transformed cells. If they are present at sufficiently high levels, the cat will not develop leukaemia or lymphosarcoma although it may still be viraemic. These cats may remain asymptomatic carriers and are as much a source of infection to the healthy cat population as are clinically affected viraemic cats.

SUSCEPTIBLE HOSTS

All cats previously unexposed to FeLV infection are fully susceptible. Younger cats are more susceptible to FeLV infection than older cats, there being a higher incidence of lymphosarcoma in cats under 5 years of age. It seems that there is also a breed susceptibility in that there is a higher incidence of this disease in Siamese and Burmese cats. This is probably a reflection of the fact that these are exotic breeds which are often housed in relatively high density breeding establishments where the infection can readily spread once introduced. Males (entire) are apparently more susceptible than females but this may be due to their greater tendency to roam. Stud males are also subjected to a higher degree of exposure to possible sources of infection and are therefore more likely to become infected.

PATHOGENESIS

The latent period from the time of initial infection until the ultimate development of overt disease is very variable and may be as long as 4 years in some instances or as short as 1 month in others. The average length of time between detection of FeLV infection and the manifestation of disease would appear to be somewhere between 6 and 12 months.

Initial viral infection is followed shortly (after about 10 to 21 days) by a viraemia and a generalised immunosuppression which may persist in susceptible cats until the development of leukaemia, lymphosarcoma or some other FeLV-associated disease. It is thought that the reduction in immunocompetence is brought about by an immunosuppressive protein derived from the FeLV. It is thought that the virus multiplies first in the cells of the bone marrow and then spreads via the blood stream to other tissues of the body, including epithelial cells. The role of epithelial cells in the pathogenesis of this disease is not altogether clear, although it seems that they may be important as a source of infection for other cats. Ultimately a chronic viraemia results in the development of leukaemia, lymphosarcoma or some other FeLV-associated disease, depending on the target cell.

PATHOLOGY

FeLV-associated diseases are defined as those caused by or associated with the FeLV. They are not necessarily neoplastic (FeLV induces neoplasia only in the cells of the haemopoietic or lymphoid organs) and may be classified as follows:

1. Lymphosarcoma (LSA)
   (i) Alimentary
   (ii) Thymic/cranial mediastinum
   (iii) Multicentric
   (iv) Other

2. Leukaemia
   (i) Lymphoid/Lymphocytic leukaemia
   (ii) Myeloid/Granulocytic leukaemia

3. Non-neoplastic diseases
   (i) Non-regenerative anaemia
   (ii) Glomerulonephritis
   (iii) Thymic atrophy
   (iv) Abortion and foetal resorption
   (v) Panleukopaenia-like syndrome
   (vi) Immunosuppression and secondary infection

1. Lymphosarcoma

Lymphosarcomas are solid tissue tumours involving the cells of the lymphoid series, including the histiocytes or reticulum-cells lining the sinuses and are usually aleukaemic, although tumour cells may appear in the circulation intermittently or terminally, in which case the term "leukaemic" or "sub-leukaemic" may be used to describe the condition. It is characteristic of the cat that LSA may occur in organs not primarily of the lymphoid system, such as the kidneys or the intestine.

In the intestinal form of LSA it is usually the terminal portion of the ileum which is involved over a distance of several centimeters but occasionally the stomach, duodenum, caecum, colon or rectum may be the main sites of involvement. Occasionally the tumour takes the form of a diffuse thickening of a large portion of the intestine, or there may be numerous small nodules in...
the intestinal wall\textsuperscript{13,14}. The affected portion of the bowel is distended and the wall thickened by white tumour tissue. This may result in obstruction of the lumen and dilatation of the proximal gut\textsuperscript{13,14}. The regional mesenteric lymph nodes are frequently involved simultaneously but may be the site of the predominant lesion, with little or no involvement of the gut or other organs. They may become so massively enlarged as to impair intestinal function. Other organs such as the liver and spleen and especially the kidneys may also be involved\textsuperscript{11,14}. The peripheral lymph nodes are not enlarged\textsuperscript{14}.

The kidneys are often the site of the predominant lesion or may be involved together with the intestine. Occasionally there may be only one or two nodules with more significant lesions elsewhere. When there is any major involvement of the kidneys, it is almost invariably bilateral and the kidneys are usually markedly enlarged\textsuperscript{6}.

Tumours of the cranial mediastinum may develop in the mediastinal lymph nodes or the thymus. A firm, pale, pear-shaped mass fills the cranial and ventral portions of the thoracic cavity and may even bulge forward through the thoracic inlet. The trachea and oesophagus may be displaced upwards and are sometimes surrounded. The lungs and heart are displaced caudally and dorsally. Occasionally the wall of the aorta may be invaded. The chest wall and diaphragm may be involved, with infiltration of the intercostal muscles and ribs\textsuperscript{5}. There is often a hydrothorax and the fluid is usually clear and straw-coloured. Occasionally there is an obstruction of the lymphatic drainage, resulting in a milky appearance of the fluid due to the presence of chyle\textsuperscript{8}.

Thymic or cranial mediastinal LSA must be differentiated from a thymoma which is usually a benign tumour, involving both the epithelial and the lymphoid elements of the normal thymus. A diagnosis can be made by histopathological examination\textsuperscript{8}.

There is seldom massive involvement of all the lymph nodes throughout the body in the cat, as is common in dogs and cattle. In the cat it is the mesenteric lymph nodes which are most commonly involved, whether grossly or microscopically, and occasionally this is the predominant lesion. Other lymph nodes are often involved but not necessarily with any apparent relationship to lesions elsewhere. Affected lymph nodes are enlarged\textsuperscript{8}.

The spleen is frequently involved to a variable degree in all the lymphoid malignancies of the cat\textsuperscript{6}. There may be slight to moderate splenomegaly with increased prominence of lymphoid follicles, especially on cut surface. Occasionally splenomegaly is severe.

The liver, like the spleen, is involved to a varying degree in many cats with lymphoid malignancies. The liver may be slightly or moderately enlarged, with focci of round cells or round cell infiltration in the portal and perilobular areas. Pale, discrete nodules and masses of any size are seen occasionally\textsuperscript{16}.

Lymphosarcomas do not occur as commonly in other organs but have been described in the nasopharynx; pharyngeal tonsils, larynx, lungs, tongue, oesophagus, uterus, eyes, brain and spinal cord, salivary gland, pancreas, adrenals and thyroids\textsuperscript{8}.

2. Leukaemias

True leukaemias are generally regarded as having their origin in the bone marrow and are also called myeloproliferative disorders. They are thought to arise from uncontrolled proliferation and defective maturation of the bone marrow cells\textsuperscript{2,12}. These leukaemic cells are found in the bone marrow and in the blood. They metastasize via the blood rather than the lymphatic system and invade other organs of the body such as the liver, spleen or lymph nodes.

The leukaemias are classified according to the cell series involved. The different cell types are thought to arise from a multipotential stem-cell in the bone marrow and may be of the granulocytic, monocytic, erythroid or megakaryocytic series, either singly, sequentially or in various combinations. Unlike LSA, there is often considerable overlap between the different forms of leukaemia, both in individual animals and in the course of the disease in a particular animal\textsuperscript{10,20}.

Leukaemias may also be described as leukaemic, subleukaemic or aleukaemic. Leukaemic leukaemia is characterised by a leukocytosis, and subleukaemic leukaemia by a normal or low white cell count. In both instances there are sufficient numbers of malignant cells present in the blood to allow a diagnosis to be made by examination of a blood smear. An aleukaemic leukaemia cannot be diagnosed by blood smear examination, but leukaemic cells can be found in the bone marrow, lymph nodes or other tissue samples. A LSA could thus be termed an aleukaemic lymphocytic leukaemia but the term lymphosarcoma is generally preferred\textsuperscript{11}.

Lymphoid or lymphocytic leukaemia is the most common form seen but is not usually regarded as a true leukaemia as it does not have its origin in the bone marrow. It is thought to be a leukaemic form of LSA\textsuperscript{2,14}.

Myeloid leukaemia involves neoplastic proliferation of cells of the granulocyte series, usually neutrophils, and is therefore a true leukaemia. Differential staining is essential for accurate diagnosis\textsuperscript{9,11}. Myeloid leukaemia involving cells of the eosinophil or basophil series is rare. Basophilic leukaemia must be differentiated from mast cell leukaemia or mastocytosis\textsuperscript{9,11}.

Anaemia is always severe\textsuperscript{6}. The spleen is massively enlarged and paler than normal due to infiltration of the red pulp by leukaemic cells. The Malpighian bodies are not visible\textsuperscript{8,11,14}. The liver is usually pale and enlarged due to diffuse infiltration of the portal areas and sinusoids by leukaemic cells\textsuperscript{9,11,14}. The bone marrow is always involved and is greatly expanded, filling the medullary cavities of the long bones. It is pale pink in colour and highly cellular in consistency\textsuperscript{9,11,14}. The lymph nodes are usually moderately enlarged and are less cellular on cut surface than with LSA\textsuperscript{8,11,14}.

**CLINICAL FINDINGS**

The clinical signs seen with FeLV-associated diseases tend to be rather variable, depending on the organs or systems affected. General symptoms frequently associated with these conditions are anorexia, emaciation, dehydration, listlessness and depression, intermittent fever and anaemia\textsuperscript{9,10}. Some or all the lymph nodes may be enlarged to a variable extent. Hepatomegaly and splenomegaly are common findings\textsuperscript{9,10}. The blood picture is very variable. It may be completely normal or there may be indications of a non-regenerative anaemia, leukenopaenia or leukocytesis and thrombocytopenia.
Lymphosarcoma in the cat may be alimentary, multicentric, thymic (cranial mediastinum) or may involve other organs such as the skin, the eye or the nervous system.  

(i) Alimentary lymphosarcoma

This, and LSA involving the kidney, are the most common forms of LSA in the cat. If the lesion is in the upper small intestine, the main clinical signs are usually vomiting, anorexia and depression, while if the lower bowel is affected, there is usually diarrhoea and emaciation. The faeces are sometimes blood-tinged and there is loss of condition, anaemia and dehydration. These tumours are usually easily felt on abdominal palpation but should not be confused with faecal masses, foreign bodies or intussusception. A radiograph will facilitate diagnosis; areas of increased density and alterations in lumen diameter will be revealed. Barium radiographs may show a constriction or obstruction of the lumen at the site of the tumour with dilatation of the intestinal lumen proximally. Anaemia may be severe and leukaemia occurs in a few cases. Splenomegaly and hepatomegaly may be present. If the kidneys are involved there may be a nephrotic syndrome with proteinuria, hypoproteinaemia and oedema, or uraemia with stomatitis, halitosis, anorexia, vomiting, polydipsia, polyuria and an elevated blood urea nitrogen (BUN) and creatinine. The kidneys may be markedly enlarged and, in an emaciated cat, may even be visible as bulges in the flanks.

(ii) Multicentric lymphosarcoma

This is a less common form of LSA than the alimentary form and is characterised by a bilateral and more or less symmetrical involvement of the lymph nodes. Certain groups of lymph nodes may be more markedly involved than others. The enlarged superficial lymph nodes should be readily felt on palpation. Splenomegaly and hepatomegaly are common and may be felt on abdominal palpation. Other organs or systems commonly affected are the kidneys, lungs, heart, gastro-intestinal tract and bone marrow. Anaemia may be present. Leukaemia may be present in some cases and occasionally there is a neutrophilia. Aﬀected cats show few specific clinical signs when suffering from this form of LSA. Generally all that is reported is anorexia, progressive depression and emaciation. The diagnosis may be conﬁrmed by a lymph node biopsy and histopathology.

(iii) Thymic (Cranial mediastinum) lymphosarcoma

This is again a less common form of LSA than the previous one and is usually seen in cats less than 3 years old. The tumour is thought to arise in the thymus and/or cranial mediastinal lymph nodes. Other organs in the body such as the liver or spleen are always involved to a greater or lesser extent. The prescapular and axillary lymph nodes are sometimes also involved. A hydrothorax usually develops, resulting in partial collapse of the lungs. This, together with physical obstruction by the tumour, causes respiratory embarrassment, dyspnoea with slow abdominal respiration, cyanosis, exercise intolerance and possibly mouth-breathing. Coughing and vomiting shortly after swallowing solid food may also be noted. The heart sounds are muffled or inaudible. The apical impulse may be absent and lung sounds are only audible in the upper caudal portion of the chest. The chest may be barrel-shaped and abnormally firm on palpation.

Although the liver and spleen are usually involved, they are seldom suﬃciently enlarged for this to be determined by abdominal palpation. If anaemia is present in this form of the disease it is usually mild.

Differential diagnoses are pneumothorax, empyema, hydrothorax as a result of cardiac or hepatic disease, chylothorax and diaphragmatic hernia with the presence of abdominal viscera in the thoracic cavity. All these can result in respiratory distress. The diagnosis may be confirmed radiographically and by thoracocentesis. The tumour is usually visible in radiographs and results in displacement of the oesophagus, trachea and heart. Thoracocentesis usually yields a clear, straw-coloured fluid. If this is centrifuged and the sediment examined microscopically, malignant lymphoblasts are usually found. If the tumour is necrotic or if a blood vessel is punctured during the procedure, cell debris or red blood cells may be found in the sediment. If the fluid is milky, this may be due to obstruction of lymphatic drainage.

(iv) Unclassified forms of lymphosarcoma

These forms of LSA are relatively rare and the clinical signs vary depending on the organs involved.

(a) Skin

Lymphoid tumours of the skin are very varied in their appearance and may take the form of single or multiple, non-pruritic, nodular lesions or irregular areas of thickened skin. They may also involve the subcutis and underlying muscle and are usually present elsewhere in the body as well. The diagnosis can be made on a skin biopsy.

(b) Eye

Primary intra-ocular LSA is unlikely and any involvement of the eye is usually secondary. Hyphema, hypopyon and panophthalmitis are also seen in association with intraocular LSA. Orbital infiltration and retrolublar tumours resulting in exophthalmia have also been reported.

(c) Nervous system

There may be involvement of the brain, spinal cord or peripheral nerves, resulting in motor and/or sensory deficits or seizures.

(d) Nasal passages

LSA of the nasopharynx may result in a constant nasal discharge and asymmetry of the external nares. On radiographical examination, there will be increased

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density in the affected areas\(^1\). Involvement of the pharyngeal tonsils, larynx and the lungs has also been reported\(^8\).

(e) Other
Involvement of the tongue, salivary glands; uterus, heart muscle and valves, pancreas, adrenal and thyroid glands has been reported\(^6\).

2. Leukaemia
Cats suffering from leukaemia are usually ill for a number of months, but an animal may only be presented for examination subterminally. There is usually a history of intermittent fever, anorexia and depression\(^1\). There is almost invariably a severe myelophthisic anaemia due to progressive destruction of the normal bone marrow constituents. The megakaryocytes are also destroyed, resulting in a thrombocytopenia and petechial haemorrhages may be present in the skin and mucous membranes\(^9\). Because leukaemic cells are present in the blood stream, the liver and spleen are heavily infiltrated and there is always a marked splenomegaly and often a hepatomegaly. The lymph nodes are never involved to the same extent as they are with LSA but may be slightly to moderately enlarged. Enlargement of the superficial lymph nodes may be noticed on palpation\(^11\). A blood smear must always be carefully examined for other possible causes of anaemia such as Haemobartonella felis infection. In a leukaemic cat, the white cell count may be normal or decreased but is usually raised, and abnormal or immature cells of the myeloid series are seen in greater numbers than normally expected\(^9\)\(^14\).

3. Non-neoplastic diseases
Unlike the other oncogenic viruses, FeLV is the cause of several non-neoplastic conditions in the cat\(^3\).\(^14\).

(i) Anaemia
Anaemia is commonly associated with FeLV infection and may occur as a primary clinical entity on its own in the absence of neoplasia or as part of the feline leukaemia/lymphosarcoma complex\(^18\). Two distinctly different types of anaemia are known to occur. One is a macrocytic, normochromic anaemia of haemolytic origin, while the other is a normocytic, normochromic aplastic (non-regenerative) anaemia in which there is no indication of bone marrow suppression\(^11\)\(^14\). The clinical signs are typical of anaemia (pale mucous membranes, tachypnoea, water-hammer pulse and tachycardia, lethargy and low exercise tolerance) and affected cats usually die of heart failure due to the severe anaemia. The packed cell volume is very low and may even fall below 10%\(^14\).

(ii) Glomerulonephritis
The incidence of glomerulonephritis in FeLV positive cats is much higher than expected. It has been reported to occur in association with LSA in cats and has also been experimentally induced. There has also been evidence of FeLV infection in cats with glomerulonephritis but which did not have neoplasia\(^14\). Trapping of circulating antigen-antibody complexes in glomerular basement membranes may result in a membranous or proliferative glomerulonephritis. This interferes with the glomerular filtration process and results in a uraemic syndrome in the former instance or a nephrotic syndrome in the latter\(^11\)\(^14\). In the uraemic form, there may be initial polydipsia and polyuria, which eventually transform into an oliguria or anuria and are followed by a typical uraemic crisis. There may also be halitosis, mouth ulcers and vomiting as well as raised BUN and creatinine levels\(^14\). In the nephrotic form there is a marked proteinuria, hypoproteinaemia and generalised oedema. The BUN remains normal until late in the disease\(^14\)\(^18\).

(iii) Thymic atrophy
Thymic atrophy has been seen in young kittens experimentally inoculated with FeLV as neonates. This results in immunodepression, particularly of cell-mediated immunity, and a “fading kitten” syndrome, due to any of several secondary bacterial or viral infections. Bronchopneumonia, upper respiratory tract infections and various forms of enteritis are commonly seen and ultimately result in the death of the kitten before there is any indication of leukaemia or LSA\(^11\)\(^14\)\(^15\).

As there are other virus infections which may result in thymic atrophy (feline panleukopaenia virus and calicivirus) and because the secondary infections can mask the primary cause, it is advisable, if possible, to exclude FeLV infection in the kittens and the queen. This could be of particular importance in a breeding establishment if there are problems in raising healthy kittens\(^14\)\(^15\).

(iv) Abortion and foetal resorption
FeLV infection has been implicated as a possible cause of infertility, foetal resorption and abortion where no other cause can be found. Bacteriological cultures were negative and those cats tested for toxoplasmosis were also negative\(^3\)\(^11\)\(^14\)\(^18\).

(v) Panleukopaenia-like syndrome
Panleukopaenia may occur concurrently with FeLV-associated disease but occasionally a severe form of enterocolitis with lesions similar to those of panleukopaenia is seen in association with FeLV infection. This condition if often associated with stress and is characterised by vomiting, haemorrhagic diarrhoea, dehydration, fever and anorexia\(^3\)\(^11\)\(^18\).

(vi) Immunosuppression
Apart from thymic atrophy, FeLV infection is known to result in immunodepression in older animals, with suppression of the humoral as well as the cell-mediated response. This may result in secondary bacterial or viral infection with manifestations such as non-healing wounds, stomatitis, respiratory infections, enteritis and recurrent fever. There is often a leukaemia instead of the expected leukocytosis. FeLV infection should be considered when conditions such as these do not respond to treatment. Other conditions predisposed to by the immunodepressive effect of the FeLV are feline infectious peritonitis, feline infectious anaemia, granulomatous disease and possibly toxoplasmosis\(^3\)\(^11\)\(^18\).

DIAGNOSIS
A diagnosis is generally suggested on history and clinical signs and should be confirmed by microscopic study of the blood, bone marrow and other body fluids or...
tissues. commercially available diagnostic tests include the Feleuk (pitman moore) test for FeLV which is a sensitive fixed cell immunofluorescence test for the detection of FeLV antigens in the white blood cells and platelets of peripheral blood. This correlates with the presence and active production of infective FeLV particles in the blood and is reputedly accurate even in blood smears which are some months old. there is also the leukassay-F (ethnor laboratories) test which is an enzyme-linked immunosorvent assay (ELISA) test which is reputedly highly sensitive, specific, quick and simple to perform. the leukassay-F test is able to detect FeLV antigens at an earlier stage of infection than the Feleuk test. A cat which is Feleuk negative but leukassay-F positive will almost certainly be Feleuk positive if retested 7-14 days later. the leukassay-F test can readily be carried out in a private practitioner's laboratory, whereas for the Feleuk test blood smears must be sent to a specialist laboratory.

It is important that these tests should be correctly interpreted. if a test is positive, it indicates that the cat is infected with FeLV, but it does not necessarily mean that the cat has the disease or even that it will ultimately develop the disease, nor does it indicate immunity or the likelihood of immunity developing. a clinical diagnosis should not be based on the results of a test alone. the latter should supplement the clinical and laboratory findings and should be used to confirm and not make a diagnosis. a negative FeLV test indicates that the cat is not viraemic at the time of the test. it does not mean that the cat is immune to the virus or the disease, or that it does not have the disease. cats with LSA may not necessarily be viraemic. the virus may be present in the cells of a LSA and later be reactivated, resulting in a viraemia. in certain cases a cat may not be viraemic but the virus may be present in other body fluids, such as the urine. a cat occasionally reverts from FeLV positive to negative but this is rare.

### TREATMENT

There are 3 alternatives available to the veterinarian who is prepared to undertake treatment of a cat suffering from leukaemia or LSA. These are surgery, irradiation or chemotherapy, which may be used individually or in combination. No more than about 10% of cats with leukaemia or LSA are likely candidates for treatment, and it should be born in mind that these animals may still shed virus and be a source of infection to other cats. the condition of the cat and its blood picture must be carefully monitored in any treatment involving the use of cytotoxic drugs. a complete physical examination and blood test should be done before commencing treatment and every 5-7 days thereafter for at least 2 months. the veterinarian should check for anorexia, lethargy, dehydration and possible secondary infection during treatment. a complete blood count, packed cell volume and platelet and reticulocyte count as well as urinalysis should be carried out on each visit.

(i) **Chemotherapy**

Drugs which are used in the treatment of leukaemia or LSA are corticosteroids, Cyclophosphamide, Vinca alkaloids, L-Asparaginase and 6-Mercaptopurine. Corticosteroids (Prednisolone, Betamethazone) in general have few side effects in cats and are therefore good drugs to use in this species. A high dose (Predni- solone 5 mg twice daily) should be given to start with. this can then be reduced according to effect.

Cyclophosphamide can be used if corticosteroids are or become ineffective or can be used together with, alternately with or instead of corticosteroids. It has an advantage in that it can be administered orally. it should not be used if there is any renal involvement, and its effect must be carefully monitored as it causes a marked and sudden drop in the number of circulating leukocytes, the red blood cells and platelets.

The Vinca alkaloids have been used with relative success but have the disadvantage of having to be given intravenously. they are highly irritant and extravasation causes severe sloughing.

L-Asparaginase has not been used to any extent in treatment of leukaemia or LSA in cats but seems promising nonetheless. it is administered intraperitoneally at an average daily dose of 400 IU/kg for 10 days followed by a weekly maintenance dose. no adverse effects in cats have been reported.

6-Mercaptopurine causes drastic bone marrow depression as well as reducing the number of circulating malignant cells. Doses should therefore be minimal and the blood carefully monitored.

(ii) **Surgery**

Surgery is seldom successful unless the LSA is confined to a single circumscribed lesion, which is rare in the cat. Surgery should not be undertaken if there is any involvement of the blood or bone marrow. Removal of a lymphomatous spleen is seldom successful, nor is removal of a single kidney as involvement is almost always bilateral.

(iii) **Irradiation**

Use of irradiation for the treatment of leukaemia or LSA has been of limited and transient value in cats. as far as is known, total inactivation of the bone marrow by irradiation, followed by a bone marrow transplant, has not been attempted in cats.

### CONTROL

Two possibilities may be considered here. One is the identification of FeLV positive cats and their possible elimination by isolation or euthanasia. This is applicable in breeding establishments and catteries where large numbers of cats are in close contact with one another, rather than to the individual cat owner. the other possibility is vaccination. It is not recommended that all cats should be tested for FeLV as a matter of routine but any cat suspected of having FeLV infection as well as those suffering from chronic undiagnosed diseases which do not respond to treatment or which have been exposed to another FeLV positive cat, should perhaps be tested. in the latter instance 2 tests should be done, 3 months apart, to allow for the long incubation period. any cats which are due to be imported or used as blood donors should also be tested.

Testing of all cats and the removal of positive cases from catteries and breeding establishments have been advocated. FeLV positive animals are strictly isolated or euthanased, while the remainder are quarantined and retested 3 months later in case any were in the incubation stage. this procedure should continue until all uninfected cats remain negative on 2 con-
secutive tests 3 months apart. Thereafter, all stud

cats brought into a cattery should be quarantined and
tested before being introduced.

Various FeLV vaccines have been developed and

tested for the prevention of FeLV viraemia and FeLV
disease, but as yet none of these are commercially

available.

CONCLUSION

A final important aspect of this disease is informing and

advising the owner, always bearing in mind that the

final decision as to what course of action to take must

lie with the owner. In attempting to give a prognosis, it

should be remembered that if a cat is FeLV-positive but

does not have a FeLV disease (i.e. is an asymptomatic

carrier), then, apart from being a potential source of

infection to other cats, it may develop leukaemia or

LSA within 6 months to 2 years, although until tests for

anti-FOCMA antibodies become commercially avail­

able, it will not, in fact, be possible to predict definitely

which cats will develop FeLV-associated disease and

which not. Isolation of such cats is a consideration,

but a cat used to roaming freely may not adapt well to

living indoors, and it is not always possible to isolate it

effectively from other cats in the household. If an

owner chooses to risk infection of other cats in his

household, there should be no contact with neighbours' cats. His cats will have to be kept indoors and neigh­

bours' cats should have no access to them. Euthanasia

may be advised, depending on the circumstances of the

cat's environment and its degree of contact with other cats. Cats suffering from FeLV-associated diseases may

respond temporarily to chemotherapy, and a remission

lasting some months may result. Treatment is, how­

ever, prolonged and expensive and extensive follow up

care on the part of the owner and the veterinarian is

required. Few cats are in fact suitable candidates for

chemotherapy in terms of their general condition and

ability to withstand the side effects of the drugs.

Chemotherapy does not destroy the virus. The cat

therefore remains a potential source of infection and

the disease may even become aggravated or manifest in

other forms.

Prognosis, with or without treatment, is poor and the

quality of life of the animal can deteriorate rapidly.

Euthanasia is probably the best course to suggest in

cases manifesting overt disease, but the emotions and

ultimate decision of the owner must always be re­

spected.

REFERENCES

1. Bennet A M, 1979 The fluorescent antibody test for the feline

leukaemia virus (FeLV), Australian Veterinary Practitioner 8:

49-51

2. Carpenter J L and Holzworth J, 1971 Treatment of leukaemia in

the cat. Journal of the American Veterinary Medical Association

158: 1130-1134

3. Cotter S M and Hardy W D, 1975 Association of FeLV with

lymphosarcoma and other disorders in the cat. Journal of the

American Veterinary Medical Association 166: 449-454

4. Ethnor Laboratories, Advertising letter, February 1980

5. Fink M A, Sibal L R and Plata E J, 1971 Serologic detection of

feline leukaemia virus antigens or antibodies. Journal of the

American Veterinary Medical Association 158: 1070-1075


Hardy W D, 1980 Increased risk for lymphoma and glomerulo­
nephritis in a closed population of cats exposed to FeLV. Ameri­

can Journal of Epidemiology 111: 337-346


of the American Veterinary Medical Association 158: 1119-1122


I. Lymphoid malignancies. Journal of the American Veterinary

Medical Association 136: 47-69


II. Journal of the American Veterinary Medical Association 136:

107-121

10. Howell, Prof. P., Dept. Infectious Diseases, Faculty of Veterin­

ary Science, Onderstepoort, Virology lecture notes, 1980

11. Jarrett W F H 1975, Cat leukaemia and its viruses. Advances in

Veterinary Science and Comparative Medicine 19: 165-193

12. Kahn D E, 1980 Field evaluation of Leakassay-F an FeLV detec­

tion test kit. Feline Practice 10: 41-45


Electron microscope detection of viruses in feline lymphosar­

coma. Journal of the American Veterinary Medical Association

158: 1109-1118

14. Mackey L, 1975 Feline leukaemia virus and its clinical effects in

cats. Veterinary Record 96: 5-11

15. Norsworthy G D, 1977 The feline leukaemia virus. Feline Prac­
tice 7: 52-58


17. Norsworthy G D, 1977 The feline leukaemia virus associated
diseases: Part 2 – The leukaemias. Feline Practice 7: 35-40

diseases: Part 3 – Non-neoplastic diseases. Feline Practice 7:

38-41


1979. A report to Practitioners – Immunoprevention of feline

leukaemia. Feline Practice 9: 16-20


in the cat associated with C-type leukovirus particles in the bone

marrow. Journal of the American Veterinary Medical Associa­

tion 157: 1686-1696


to leukaemic leukaemias. Feline Practice 6: 32-34

22. Schneider R S 1971, Comments on epidemiologic implications of

feline leukaemia virus. Journal of the American Veterinarian

Medical Association 158: 1125-1129

23. Weijer K and Daams J H, 1976 The presence of leukaemia (lym­

phosarcoma) and feline leukaemia virus (FeLV) in cats in the


24. Weijer K and Daams J H, 1978 The control of lymphosarcoma

leukaemia and feline leukaemia virus. Journal of Small Animal

Practice 19: 631-637