Clinical feline toxoplasmosis: parasitological, haematological and serological findings in retroviral infected and uninfected

E. A. Okewole^{1*}, and M. O. Akpan²

¹Department of Veterinary Medicine, University of Ibadan, Ibadan, Nigeria ²Veterinary Teaching Hospital, University of Ibadan, Ibadan, Nigeria

OKEWOLE, E. A., M. O. AKPAN: Clinical feline toxoplasmosis: parasitological, haematological and serological findings in retroviral infected and uninfected cats. Vet. arhiv 72, 67-79, 2002.

ABSTRACT

Reports of the parasitological, haematological and serological diagnoses of clinical feline toxoplasmosis in 10 middle-aged Siamese cats are made with special emphasis on the reconciliation of different diagnostic findings in the process of making a definitive diagnosis. Three of the cats were immuno-competent while the remaining 7 were immuno-compromised due to retroviral co-infections. Major clinical signs included anorexia, fever (>39.8 °C), hyperpnoea, dyspnoea, pneumonia, emaciation, lethargy, icterus, regenerative anaemia and lymphopenia, while the immuno-compromised animals additionally had non-regenerative anaemia and some ocular disorders. The immuno-competent cats responded positively to the 4-week treatment protocol, while the immuno-compromised did not and were consequently eliminated from use in further trials. The immuno-competent group had a higher mean live mass gain of 1.8 kg in 55 days and a concurrent above normal haemogram and were thus recommended for uses other than in drug trial study, since the latter could involve the administration of the immunosuppressive drugs that could reactivate oocysts shedding - a zoonotic risk. Counsels were provided on the hygienic mode of managing these cats from a zoonotic aspect.

Key words: toxoplasmosis, haematology, serology, retroviral infections, immunodeficiency, cat

ISSN 0372-5480 Printed in Croatia

^{*} Contact address:

Dr. E. A. Okewole, Department of Veterinary Medicine, University of Ibadan, Ibadan, Nigeria, Phone: +234 2 8102461 64-Ext.1507; Fax: +234 2 8103043; E-mail: b-olufemi@hotmail.com

Introduction

Toxoplasma gondii is an obligate intracellular coccidia that can be found in most animals, although cats and other *Felidae* are the only definitive hosts (FRENKEL et al., 1969; FRENKEL and HOLZWORTH, 1987; DUBEY, 1986). Toxoplasmosis is extremely common throughout the world but its sero-prevalence in cats and man varies by region and country, often around 30-40% (LAPPIN, 1999). Clinical signs in cats are various, with both fatal and sub-lethal syndromes being recognized. Clinical diagnosis is easy to make but a definitive diagnosis could be more difficult, if not impossible, especially in immuno-compromised cats.

Immuno-suppression by retroviruses, pregnancy and corticosteroid therapy are amongst the factors influencing the clinical course, immune response, treatment response and prognosis of clinical toxoplasmosis in cats and man.

The retroviruses are a group of RNA viruses that possess the reverse transcriptase enzyme that copy their single-stranded RNA genome into the complementary single-stranded DNA of their hosts (FENNER, 1976). The feline leukaemia virus (FeLV) and the feline immunodeficiency virus (FIV) are members of the *Retroviridae* family that are common pathogens of the cat.

Persistent FeLV infection of cats is characterized by a reduction in the numbers of lymphocytes, neutrophils, reduced T-cell blastogenesis, cutaneous anergy and impaired antibody production which predispose to secondary infections (PERRYMAN et al., 1972; HARDY, 1982). Similarly, as with FeLV, FIV has been reported to have a tropism for T-cells and also a progressive deterioration in immune functions with depletion of CD4+(T-helper) lymphocytes (features that are also typical of HIV infections in humans) have been documented in infected cats (SPARKES et al., 1993).

In view of the potential zoonotic risk of both complicated and uncomplicated toxoplasmosis, veterinarians are often consulted for their opinions on the desirability or otherwise of keeping an infected cat, a task that demands a sound understanding of both the biology of the parasite and the disease process in normal and immuno-compromised cat hosts.

This paper reports the diagnosis and treatment of feline toxoplasmosis in a group of 10 cats. It also stresses the significance of reconciliation of diagnostic results, as well as the influences of viral co-infection on the serological pattern, treatment responses, and prognosis of the disease.

Materials and methods

Case History. In November, 1998, ten 1½-3½ year-old Siamese cats (C₁-C₁₀) were brought to the Veterinary Teaching Hospital (VTH) from the Pharmacology Department of the College of Medicine, University of Ibadan for blood and intestinal parasitological examination following an observed general unthriftiness. All cats had been previously purchased from an open-air market in Ibadan. Seven of the animals had a history of recurrent diarrhoea, anorexia, occasional vomiting, dyspnoea, polydypsia, lethargy, sero-mucous nasal discharges and enlarged abdomen.

Clinical examination findings. Nine cats had fever (39.8-40.1 °C), seven had pneumonia of different grades, with dyspnoea, polypnoea, abdominal respiration, dark-coloured diarrhoea and dehydration. Ocular involvements in these cats were in the form of ocular discharges, iritis and hyphema. All cats had pale mucosae, enlarged and palpable lymph nodes, slight splenomegaly and hepatomegaly. Six animals had many purulent skin wounds on their necks and shoulders.

Laboratory investigations. The 10 cats were weighed, number-tagged and admitted to the Veterinary Teaching Hospital (VTH) cattery to facilitate clinical investigation and care.

Parasitological examination. Fresh faecal samples from the cats taken 2 days after admission were emulsified and floated in sucrose solution (specific gravity 1.18). Isolated oocysts were washed and examined under a light microscope as earlier described by DUBEY and BEATTIE (1988). Using an ocular micrometer (Cope7, A. J. Cop & Son Ltd., England), morphometric measurements of parameters for *T. gondii* identification were taken as described by FRENKEL (1973). The oocyst-shedding cats were grouped as A, while non-shedding were grouped as B. Group A cats were $1\frac{1}{2}$ to 2 years of age, group B cats were over 2 years of age.

Haematology. Two millilitres of blood was taken from the marginal ear vein of each cat and haematological analysis was performed as described by SCHALM et al. (1975). Results are expressed in age-matched groups A, B and C to facilitate reconciliation with parasitological results. Group A cats were aged 1½ to 2 years, Group B 2 to 3 years and Group C over 3 years. Group means of haematological parameters were statistically compared by the Student's t-test at a 5% level of significance.

Serology. Sera samples were collected on days 2, 9, 16 and 24 after admission and stored at 20 °C until after examination.

Serological screening. A latex agglutination test (LAT) for Toxoplasma antibodies was performed on day 2 sera using the commercial kit Toxo test⁷ (Elken Chemical Co., Tokyo, Japan and Syn Kit Inc., Chatsworth, California) as described by LAPPIN (1996). Sera for days 2, 9, 16 and 24 were also later screened for T. gondii specific antibodies, as earlier described by LAPPIN et al. (1989b) using the enzyme-linked immunosorbent assay (ELISA) commercial kit No. EIA504-A and EIA508-A (Sigma-Aldrich Co. Ltd., U.K.) for respective immunoglobin G (IgG) and immunoglobulin M (IgM) assays, on a four-fold dilution series. Storage at 20 °C facilitated the simultaneous test-run on day 24 and reduced the usual day-to-day and test-to-test variations (FRENKEL and HOLZWORTH, 1987). The day 2 sera samples were also tested for the presence of the feline leukaemia viral (FeLV) and feline immunodeficiency viral (FIV) antibodies using the commercial combined A in-house membrane form of the ELISA (Cite⁷-Idexx, Portland, Me) test kit using the method described by HOSIE et al. (1989).

Results

Parasitological examination. Young cats aged 1½ to 2 years shed only a small number of oocysts on day 2, whereas the older group (>2 years) did not shed at all.

Haematological examination. Results are shown in Table 1. Haematocrit values were below normal in all groups on day 2, but the values were much lower in groups B and C. However, on day 55 haematocrit

values increased more in group A cats than it did in groups B and C. Statistical pairings of the mean haematocrits on day 55 for group A versus B had a value of P<0.01; group B versus C had a value of P>0.05, and group A versus C had a value of P<0.01. Also, neutrophilia was observed in groups A, B and C on day 2. However, by day 55 the neutrophilia had resolved in group A although not in groups B and C. Eosinophilia was consistently observed in the 3 groups from days 2 to 55 (Table 1).

Serological assay: virological screening and LAT results. Cats C_4 , C_6 and C_7 were sero-negative for both FeLV and FIV but positive for T. gondii, coincidentally these were the same set of cats in the parasitological group A. Cats C_1 , C_2 , C_5 , C_9 and C_{10} were sero-negative for FIV but positive for FeLV and T. gondii, coincidentally these were the same set of cats in the parasitological group B. Only cats C3 and C8 were sero-nega-

Table 1. Haemogram and live mass changes (mean \pm sd) of cats on days 2 and 55 of admission

| | | Group A (3) | | Group B (5) | | Group C (2) | | |
|---------------------|---------------------|--|----------------|---|------------------|---------------------------------|----------------|-------------------|
| Haematological | | C ₄ . C ₆ . C ₇ | | C ₁ . C ₂ . C ₅ C ₉ . C ₁₀ | | C _{3-,} C ₈ | | Normal range of |
| parameters | Units | Day 2 | Day 55 | Day 2 | Day 55 | Day 2 | Day 55 | value* |
| Haemoglobin conc. | g/dl | 7.1 ± 1.2 | 11.2 ± 1.4 | 6.7 ± 1.6 | 9.5 ± 2.0 | 6.3 ± 1.0 | 9.9 ± 1.3 | 8-15 |
| Haematocrit | % | 24.2 ± 2.2 | 33.5 ± 1.8 | 20.2 ± 2.3 | 28.4 ± 1.9 | 20.5 ± 1.3 | 28.3 ± 0.7 | 30-45 |
| Total counts WBC | n/fl | 19.992 ± 1004 | 18.053 ± 908 | 29.736 ± 2.812 | 19.813 ± 1.025 | 20.458 ± 1.224 | 20.217 ± 2.004 | 5.500- 19.500 |
| Total counts RBC | 10 ⁶ /fl | 4.9 ± 1.1 | 6.7 ± 0.8 | 4.3 ± 1.4 | 4.9 ± 1.0 | 4.7 ± 0.7 | 5.1 ± 0.5 | 5.0-10.0 |
| Reticulocyte counts | % | 0.9 ± 0.2 | 2.9 ± 1.0 | 0.5 ± 0.1 | 3.1 ± 1.1 | 0.4 ± 0.2 | 1.5 ± 0.5 | 1.4-10.8** |
| Seg. Neutro. | n/fl | 16.212 ± 1006 | 12.484 ± 908 | 16.987 ± 1027 | 15.750 ± 1025 | 16.852 ± 2042 | 16.760 ± 1602 | 2.500 - 12.500 |
| Band Neutro. | n/fl | 211 ± 32 | 58 ± 09 | 58 ± 05 | 32 ± 08 | 69 ± 02 | 72 ± 03 | 0-300 |
| Lymphocytes | n/fl | 4.010 ± 213 | 794 ± 101 | 794 ± 107 | 1.471 ± 98 | 760 ± 32 | 917 ± 43 | 1.500 - 7.000 |
| Eosinophiles | n/fl | 683 ± 41 | 1.913 ± 97 | 1.913 ± 95 | 1.821 ± 102 | 1.784 ± 310 | 1.911 ± 45 | 0-1.500 |
| Monocytes | n/fl | 765 ± 104 | 753 ± 93 | 984 ± 82 | 739 ± 66 | 993 ± 56 | 622 ± 71 | 0-850 |
| Basophiles | n/fl | - | - | - | - | - | - | Rare |
| Live mass | kg | 15.6 ± 1.1 | 17.4 ± 1.3 | 16.1 ± 1.5 | 17.5 ± 1.6 | 18.3 ± 1.0 | 19.8 ± 1.3 | - |

^{*} Schalm et al. (1975)

^{**} Cramer and Lewis (1972)

Table 2. Toxoplasma gondii specific antibody titre in cats

| Groups | Groups Mean antibody titre | | | | | | | |
|------------------------------|----------------------------|--------|--------|--------|--------|--|--|--|
| | Assay | Day 2 | Day 8 | Day 16 | Day 24 | | | |
| Group A (3) | IgM | 1:502 | 1:482 | 1:305 | 1:108 | | | |
| C_4, C_6, C_7 | IgG | 1:1084 | 1:1244 | 1:2048 | 1:2052 | | | |
| Group B (5) | IgM | 1:74 | 1:63 | 1:52 | 1:30 | | | |
| $C_1, C_2, C_5, C_9, C_{10}$ | IgG | 1:1260 | 1:973 | 1:973 | 1:970 | | | |
| Group C (2) | IgM | 1:77 | 1:58 | 1:49 | 1:34 | | | |
| C_3, C_8 | IgG | 1:1184 | 1:882 | 1:880 | 1:865 | | | |

Group A were positive for T. gondii only;

Group B were positive for T. gondii and FeLV but negative for FIV;

Group C were positive for T. gondii and FIV but negative for FeLV

tive for FeLV, but were positive for FIV and T. gondii, coincidentally the same cats in the age-matched haematological group C (Table 2 footnotes).

Reconciliation of results. The shedding of a small number of oocysts $(<0. = 485 \pm 37 \text{ gm})$ by cats in group A was suggestive of a recent infection probably spanning 2 to 4 weeks earlier than day 2. The older groups, B and C, that were retroviral co-infected either did not shed oocysts or had their shedding earlier. IgM titre declined from a value of 1:502 on day 2 to a value of 1:168 on day 24, while IgG titre appreciated from a value of 1:244 to 1:2052 at the same rate. In retroviral co-infected groups B and C, both IgM and IgG titres declined, although the former declined to an almost sero-negative endpoint on day 24, while the latter declined only slightly. These trends suggested either an earlier *T. gondii* infection in these older groups than in the younger group A cats, or a recent reactivation of a chronic T. gondii infection by the immuno-suppressive effect of the coinfecting retroviruses with a terminal phase of IgM transient rise in titre and a steady post-peak indefinite decline in IgG titre, which could last well over 6 years post-infection. Haematologically, mean haematocrit, total red cell and reticulocyte counts were below normal values in the 3 groups, and even much lower in the immuno-compromised groups B and C on day 2 (Table 1). Similarly, neutrophilic leucocytosis, lymphopenia and

eosinophilia were prominent features of the haemogram, with monocytosis additionally only in retroviral co-infected groups B and C. However, on day 55 (i.e. 30th day-post treatment) these parameters were reversed in group A cats, but insignificantly in cats in groups B and C.

Serological assay had IgM titre for group A cats at 1:502 (reference value 1:64) and IgG titre over the 22-day admission period increased from a value of 1:244 to 1:2052 (reference value is a four-fold increase) (Table 2). However, cats in groups B and C had a slightly declining IgG titre and in addition an IgM titre above 1:64. Some clinical signs referable to toxoplasmosis manifest some positive responses (at least haematologically and in live mass evaluation) to *T. gondii*-specific treatment protocols.

Definitive diagnosis. Based on the history of purchase at different openair markets, the observed clinical signs referable to toxoplasmosis, neutrophilic leucocytosis, lymphopenia, eosinophilia, demonstration of specific antibody in serum, an IgM titre above 1:64, a four-fold increase in IgG titre within a 22-day period, and positive responses to the specific toxoplasmosis treatment protocols, a definitive diagnosis of clinical toxoplasmosis was made in the 10 cats with a more fulminating manifestation (including ocular) than in the 7 animals that were retroviral co-infected.

Management protocols. In the meantime, treatment of the 3 groups of cats began on day 25 and continued for 28 days as follows: because we were handicapped by the absence of donor cats for the whole blood transfusion of all sick cats, which we considered an ideal treatment, group A cats had oral clindamycin phosphate, folic acid, prednisolone and parenteral testosterone propionate with dextrose-saline rehydration additionally for the diarrhoeic animals. However, groups B and C additionally had parenteral vitamin B complex, a tapering regimen of oral prednisolone, local dressing of their skin wounds with 1% chlorhexidine digluconate and topical treatment of ocular manifestation with 1% prednisolone eye drops.

Clinical reassessment and counselling. On day 5 group A cats were alert, active and healthy with a mean live mass gain of 1.8 kg and a mean haematocrit gain of 9.3%. However, cats in groups B and C showed no

satisfactory improvement in their health, with persisting neutrophila which was probably due to persisting subclinical pneumonia and lower mean live mass gains of 1.4 kg and 1.5 kg, and mean haematocrit gains of 8.2% and 7.8% respectively. In the light of this we eliminated the 10 cats from the drug trial study. For group A cats, which no longer constituted a zoonotic risk, having undergone primary oocyst shedding, a reactivation of oocyst shedding was possible should they receive an immuno-suppressure drug during the course of the study (DUBEY and BEATTIE, 1988, LAPPIN et al., 1992a). Groups B and C were also eliminated because of their possibiity of developing secondary immunodeficiency syndromes in the course of drug trial and were finally euthanized at the owners request. We however, approved the use of the group A cats for other studies different from drug trial, with a proviso that good personal hygiene be maintained in their management.

Discussion

This report has again corroborated the inadequacies in the exclusive use of either the parasitological or serolgical methods for the diagnosis of feline toxoplasmosis as earlier observed by FRENKEL and HOLZWORTH (1987), LAPPIN et al. (1989a, 1999).

The declining values of the IgM antibody class titre in the group A cats in the first 3 weeks of admission represented the characteristic decline phase of primary transient rise in recent *T. gondii* infection in cats and this is consistent with the earlier reports of FRENKEL and HOLZWORTH (1987), LAPPIN et al. (1989b, 1992a), DUBEY (1995) and LAPPIN (1999). This decline is often recommended as a reliable guide to the course of current clinical disease (LAPPIN et al., 1989a). The transient rise of the IgM itself represented the cats immune responses to the highly invasive blood or lymph-borne tachyzoites acquired possibly from their preys or home-fed tissue cysts before purchase. If the immune responses were strong enough the tachyzoite replication is attenuated to become the slow-replicating and possibly encysted bradyzoites which locate in the muscle, viscera or the central nervous system with lower antigenicity. The later attribute of the bradyzoites which might not be unrelated to their encystment, non-haematogenic course and lower replication rate has probably contributed

in part to the consistently declining level of the IgG class antibody, especially in the retroviral infected cats (Table 2).

Since only a very few oocysts (<485 gm) were shed on day 2 by the group A cats, the *T. gondii* infection probably occurred very recently with the low shedding representing the terminal stage due to an immune 'arrest'. This speculation is based on the earlier report of DUBEY and BEATTIE (1988), WILLS and WOLF (1993) that oocyst shedding last 1 to 3 weeks in primary infections, and that its termination is due usually to the arrest by the host immune responses. By our speculative hypothesis, the groups B and C cats should also be shedding by day 2, but they were not. This could either be attributed to some functional age resistance earlier reported by DUBEY et al. (1977), FRENKEL and SMITH (1982a) or the possible earlier infection of the groups with their shedding being completed before day 2.

Other differences in the group A and groups B and C cats included the consistently lower haemogram and the correspondingly lower levels of both the IgM and IgG classes of antibody in the groups B and C which probably related more to the retroviral co-infection than to the tachyzoites and brachyzoites antigenicities. According to LAPPIN (1992a), the effect of FIV infection on T. gondii-specific infection humoral responses may vary depending on the duration of T. gondii infection, the strains of T. gondii and FIV, as well as the degree of immuno-suppression induced by the FIV. As in the human acquired immune deficiency syndrome (AIDS) infection. both the IgM and IgG are usually low or irresponsive to reactivation of latent toxoplasmosis by the intercurrent HIV immunosuppression (LUFT et al., 1984). A similar reactivation of latent toxoplasmosis might be prevailing in the FeLV and FIV co-infected groups B and C cats; with the initially high IgG titre on day 2 representing a specific B-cell response to the depressed cellular immunity by the FIV induced immunosuppression. Such development have been documented to develop as a consequence of FeLVinduced immunosuppression (WITT et al., 1989) and corticosterid administration in the toxoplasmal-infected cats (DUBEY and FRENKEL, 1974). According to HARDY (1980a), FeLV immunosuppresses and lowers the IgG serum level in particular by two methods viz: formation of FeLV-IgG complexes and other fixed immune complexes on the glomeruli of cats and also by the exfoliation of PI5E from the viral envelop to abrogate

lymphocyte functions, thus compromising the cats immune responses. This might be applicable to the FeLV-infected group B cats. Similarly, BOYCE et al. (1981) reported that experimental infection of kittens with the FeLV induced rapid and selective suppression of erythroid progenitor cells. This red-cell aplasia was characterized by a non-regenerative anaemia, lymphopenia and a severe depletion of marrow erythroid precursors, but no evidence of immune-mediated red cell destruction was found. The non-regenerative anaemia and lymphopenia observed in the FeLV infected group B cats might be due to a similar suppression of the erythroid progenitor cells rather than an increased destruction of red cells.

The higher sero-prevalence of FIV in older cats with toxoplasmosis might relate to increased chances of exposure, predatory behaviour (*T. gondii* cysts), aggression, fights and bites over queens, since most of them were mature males. These are all consistent with the earlier reports of YAMAMOTO et al. (1989), WITT et al. (1989) and LAPPIN et al. (1992a) on the epidemiology of FIV.

Despite the apparent effectiveness of the anti-toxoplasmal treatment protocols, as evidenced by the marginal improvement in the haematological and live mass changes in the 3 groups, the IgG titre appreciated only in the group A cats while it actually depreciated in the groups B and C. These observations again corroborated the fact of different ages of the infection in the groups (being earlier in the groups B and C) and the other fact that toxoplamal drugs (Clindamycin phosphate and other) merely slow down the replication of, rather than eliminate *T. gondii* stages in cats (DUBEY and BEATTIE, 1988; DUBEY and THULLIEZ, 1989). Such uneliminated stages continue to sustain the chronic low-level IgG titre for as long as 6 years after the IgM titre had faded out at end of its transient rise (DUBEY, 1995).

The marginal improvement in the haematocrit values of these anaemic cats might ordinarily be creditable to the testosterone therapy that stimulate erythropoiesis (PERMAN and SCHALL, 1983) but the secondary nature of the anaemia itself to other treatable and stressful conditions might equally be worthy of consideration. Treatment of the latter (with Clindamycin in the case) might have resolved the secondary anaemia also.

E. A. Okewole and M. O. Akpan: Clinical feline toxoplasmosis: parasitological, haematological and serological findings in retroviral infected and uninfected cats

Acknowledgments

The authors wish to thank A. Mariya and Y. Raji for their care of the cats while on admission at the VTH, and Prof. F.D. Adu for the virological screening of the cats.

References

- BOYCE, J. T., E. A. HOOVER, G. J. KOCIBA, R. G. OLSEN (1981): Feline leukemia virus-induced erythroid aplasia: in vitro hemopoietic culture studies. Exptal. Haematol. 9, 990-1001.
- DUBEY, J. B. (1986): Toxoplasmosis in cats. Feline Pract. 16, 12-26.
- DUBEY, J. B., C. P. BEATTIE, (1988): Toxoplasmosis of animals and man. Baca Raton, Fla, CRC Press. pp. 1-220.
- DUBEY, J. B., J. K. FRENKEL (1974): Immunity to feline toxoplasmosis: modification by administration of corticosteroids. Vet. Pathol. 11, 350-379.
- DUBEY, J. B., P. H. THULLIEZ (1989): Serological diagnosis of toxoplasmosis in cats fed *T. gondii* cysts. J. Am. Vet. Med. Assoc. 194, 1297-1299.
- DUBEY, J. B. (1995): Duration of immunity to shedding *Toxoplasma gondii* cysts by cats. J. Parasitol. 81, 410-415.
- DUBEY, J. B., E. A. HOOVER, K. W. WALL (1977): Effects of age and sex on the acquisition of toxoplasmosis. J. Protozool. 21, 184-186.
- FENNER, F. (1976): The classification and nomenclature of viruses. Virology 71, 371-378
- FRENKEL, J. K. (1973): Toxoplamosis in and around us. Bioscience 23, 343-352.
- FRENKEL, J. K., D. D. SMITH (1982a): Immunization of cats against shedding of toxoplasma oocyst by cats. J. Parasitol. 68, 744-748
- FRENKEL, J. K., J. HOLZWORTH (1987): The coccidia. In: Disease of the cat. (Holzworth J., Ed.). Philadelphia, W. B. Saunders Co. pp. 359-405.
- FRENKEL, J. K., J. B. DUBEY, N. L. MILLER, (1969): *Toxoplasma gondii* faecal forms separated from eggs of the nematode, *Toxocara cati*. Science 164, 432-433.
- HARDY, D. Jr. (1980a): Feline leukamia virus disease. In: Feline leukamia virus. (HARDY, W. D. Jr., M. ESSEX, A. J. MCDELLAND, Eds.), New York, Elsevier (North Holland). pp. 3-31.
- HARDY, W. D. Jr. (1982): Immunopathology induced by the feline leukemia virus. In: Springer semin. Immunopathol. (KLEIN, G., Ed.). 1982, New York, Springer-verlag. pp. 75-105.
- HOSIE, M., C. ROBERTSON, O. JARRET (1989): Prevalence of feline leukamia virus and antibodies to feline immunodeficiency virus in cats in the United Kingdom. Vet. Rec. 125, 293-297.
- LAPPIN, M. R. (1989): Feline toxoplasmosis. In: Current Veterinary Therapy X. (KIRK, R. W., Ed.) Philadelphia, W. B. Saunders Co. pp. 1112-1115.

- E. A. Okewole and M. O. Akpan: Clinical feline toxoplasmosis: parasitological, haematological and serological findings in retroviral infected and uninfected cats
- LAPPIN, M. R. (1996): Feline toxoplasmosis: interpretation of diagnostic results. Seminar in Veterinary Medicine and Surgery 11, 154-160.
- LAPPIN, M. R. (1999): Feline toxoplasmosis. In Practice, Nov. Dec., 578-589.
- LAPPIN, M. R., D. L. DAWE, P. A. WINDL, C. E. GREENE, A. K. PRESTWOOD (1992a): The effect of glucocorticoid administration on oocyst shedding, serology and cell-mediated immune responses of cats with recent/chronic toxoplasmosis. J. Am. An. Hosp. Ass. 27, 625-632.
- LAPPIN, M. R., C. E. GREENE, A. K. PRESTWOOD (1989b): Diagnosis of *Toxoplasma gondii* infection utilizing an enzyme-linked immunosorbent assay for immunoglobulin M. Am. J. Vet. Res. 50, 1580-1585.
- LAPPIN, M. R., C. E. GREENE, S. WINSTON, S. L. TOLL, M. E. EPISTEIN (1989a): Clinical feline toxoplasmosis. J. Vet. Intern. Med. 13, 139-143.
- LAPPIN, M. R., S. M. ROBERTS, M. G. DAVIDSON, C. C. POWELL, J. S. REIF (1992b): Enzyme-linked immunosorbent assay for the detection of *Toxoplasma gondii* specific antibodies and antigens in the aqueous humour of cats. J. Am Vet. Med. Assoc. 201, 1010-1016.
- LUFT, B. J., R. G. BROOKS, F. K. CONLEY, R. E. McCABE, J. S. REMINGTON (1984): Toxoplasmic encephalitis in patients with acquired immune deficiency syndrome. J. Am. Med. Assoc. 252, 913-917.
- PERMAN, C., W. D. SCHALL (1983): In: Textbook of Veterinary Internal Medicine, 2nd ed. (ETTINGER, S. J. Ed.). Philadelphia, W. B. Saunders Co. pp. 1938-2000.
- PERRYMAN, L. E., E. A. HOOVER, O. S. YOHN (1972): Immunological reactivity of the cat: Immunosuppression in experimental feline leukemia. J. Natl. Cancer Inst. 49, 1357-1365.
- SCHALM, O. W., N. C. JAIN, E. T. CARROL (1975): Veterinary Haematology, 3rd ed. Philadelphia, Lea and Febiger.
- SPARKES, A. H., A. WOLF, J. WILLS (1993): Infectious diseases. In: Handbook of feline medicine (WILLS, J. and A. WOLF, Eds.). Oxford, Pergamon Press. pp. 351-354.
- WILLS, J., A. WOLF (1993): Infectious diseases. In: Handbook of feline medicine (WILLS, J. A. WOLF. Eds.). Oxford, Pergamon Press. pp. 381-382.
- WITT, C. J., T. R. MOENCH, A. M. GITTELSOHN, B. D. BISHOP, J. E. CHILD (1989): Epidemiological observations in feline immunodeficiency virus and *Toxoplasma gondii* coinfection in cats in Baltimore, Md. J. Am. Vet. Med. Assoc. 194, 229-233.
- YAMAMOTO, J. K., H. HANSEN, E. W. HO, T. Y. MORISHITA, T. OKUDA, T. SAWA. R. M. NEKUMURA, N. C. PEDERSEN (1989): Epidemiological 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. 194, 213-220.

Received: 11 January 2002 Accepted: 25 April 2002

OKEWOLE, E. A., M. O. AKPAN: Klinička toksoplazmoza mačaka: parazitološki, hematološki i serološki nalazi u neinficiranih i retrovirusom inficiranih mačaka. Vet. arhiv 72, 67-79, 2002.

SAŽETAK

Opisani su nalazi parazitološke, hematološke i serološke dijagnostike u 10 sijamskih mačaka oboljelih od toksoplazmoze s posebnim osvrtom na usklađivanje nalaza u postupku postavljanja konačne dijagnoze. Tri pretražene mačke bile su imunokompetentne, a sedam je bilo s oslabljenim imunskim sustavom zbog retrovirusne infekcije. Glavni klinički znakovi bili su anoreksija, vrućica (>39,8 °C), hiperpneja, dispneja, pneumonija, oslabljenost, letargija, ikterus, regenerativna anemija i limfopenija, dok je u životinja s oslabljenim imunosnim sustavom opisana neregenerativna anemija i očni poremećaji. Imunokompetentne mačke su izliječene četverotjednom primjenom lijekova dok imunokompromitirane nisu odgovorile na liječenje te nisu dalje korištene u istraživanju. Mačke unutar imunokompetentne skupine imale su prosječno veću tjelesnu masu 1,8 kg tijekom 55 dana te istovremeno normalan hemogram. Usprkos ozdravljenju mačke nisu bile uzete u daljnja istraživanja s obzirom da je poznato da i slaba imunosupresija može imati za posljedicu reaktivaciju bolesti s izlučivanjem oocisti u izmetinama. Raspravlja se o higijeni držanja takvih mačaka s obzirom da je toksoplazmoza zoonoza.

Ključne riječi: toksoplazmoza, hematologija, serologija, retrovirusne infekcije, imunodeficijencija, mačka