Jl S.Afr. vet. Ass. 46(3) 295-297 1975

CASE REPORT GEVALVERSLAG

ULCERATIVE GLOSSITIS — A FACET OF FELINE PANLEUKOPENIA

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SUMMARY

The clinical findings and the macroscopic and microscopic lesions in a kitten which died during an outbreak of ulcerative glossitis in a cattery are described. A brief review of the literature is given to support the theory that the virus of feline panleukopenia may well be the aetiological agent in this outbreak.

OWNER HISTORY

On 15.1.1974 two fortnight old orphan kittens from Cattery A were introduced into Cattery B, which was occupied by a number of adults together with 28 healthy kittens, in order that they might suckle off a queen with a litter of the same age. Approximately 10 days after the two kittens had been introduced, the breeder found that they were lethargic and salivating and on examination "red spots" were found on the dorsal aspect of the tips of the tongues. The two kittens were thereupon returned to Cattery A and subsequently died.

Two weeks later, a similar syndrome appeared in kittens present in Cattery B and within 3 weeks nine kittens had developed tongue lesions. The lesions developed on the tip of the tongue dorsum and were at first of approximately match head size. Subsequently they spread around the sides of the tongue, until finally after approximately 10 days the lingual edges appeared swollen and "waxy-white". This waxiness eventually sloughed off to leave a raw tender area. The lesions then regressed and healed within 21 days of their first appearance. At no time was any nasal or ocular discharge or diarrhoea observed amongst the affected kittens.

Other signs of illness reported were lassitude and salivation. The breeder reported feeling hard "lumps" in the abdomens of the affected kittens and dosed them with cod liver oil and manually extracted the faeces. During the period of illness, a commercial invalid food, Complan* mixed with milk and water was force fed. In addition one or a combination of the following drugs was administered to the affected kittens: lincomycin, chloramphenicol and ampicillin by parenteral injection, ampicillin, sulphadimidine, proteplex**, vitamin C and vitamin B per os and the topical application of a solution of gentian violet to the ulcerative lesions.

Three of the nine affected kittens died; only one of these kittens was available for autopsy.

It is interesting that in Cattery B the condition occurred in two kittens as early as 2 weeks of age, whereas the remainder of the affected kittens were 6-8 weeks old. In some cases whole litters were affected, in others only part of the litter. All queens were apparently healthy throughout the duration of the enzootic.

It is perhaps significant to record that the affected kittens were the progeny of queens whose ages varied from 4 - 6 years and which had been inoculated against feline panleukopenia (FPL) 1-3 years previously. It was the breeder's policy to inoculate all breeding stock annually with an inactivated vaccine until they reached the age of 3 years.

CLINICAL HISTORY

Three weeks after the commencement of the outbreak, four of the affected kittens were brought to the Department of Medicine, Faculty of Veterinary Science, University of Pretoria.

During examination, one male 9 weeks of age, died. The cadaver was frozen until autopsy 24 hours later, when the anterior two-thirds of the tongue was excised and stored at -20°C for virological examination. The other three kittens were taken home. Before death the salient clinical findings were salivation, lassitude, ulcerative glossitis, dehydration and constipation.

LABORATORY FINDINGS

Bacterial cultures made from the tongue yielded a rough strain of *Escherichia coli*.

PATHOLOGICAL FINDINGS

GROSS

The only lesions of significance were ulcerative glossitis particularly of the tip and sides of the anterior two-thirds of the tongue which resembled that described above, generalized atrophy of lymph nodes and oedema and congestion of the lungs. Specimens

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* Glaxo-Allenburys (S.A.)(Pty.) Ltd.

** Burns (S.A. Cyanamid(Pty.)Ltd.)

from all lesions and major organs were taken and fixed in 10% formalin for histopathological examination

HISTOPATHOLOGY

The tongue lesion, examined microscopically, consisted of a focal area of necrosis of the epithelium which in its centre involved cells of all layers and was manifested chiefly by karyorrhexis or karyolysis and cytoplasmic eosinophilia although in some areas cell detail was absent and superficial desquamation had occurred. There was no associated inflammatory reaction but several nuclei of epithelial cells bordering the necrotic area contained single small eosinophilic inclusion bodies.

The lungs showed mild inflammation, chiefly interstitial in location. Infiltrating cells were mainly mononuclear. The pneumonia was accompanied by rather severe oedema.

As it is considered that the freezing and subsequent thawing of the carcase might have influenced possible pathological changes in the other organs examined these are not included in this discussion.

DISCUSSION

Ulcerative glossitis in cats has been known for some time. The condition was first described by Smythe [11] in 1934. Subsequently Kirk [7], Weipers [12], Joshua [6] and Wilkinson [13] mentioned the condition and the disease. All five of the above authors suggested a relationship between this condition and FPL, an assumption which was borne out by the fact that cats which had recovered from ulcerative glossitis were apparently immune to FPL. According to Joshua (personal communication) the condition is most frequently seen in cats between the age of 18 months and 3 years.

Both Joshua [6] and Wilkinson [13] reported that the disease is rarely fatal. In the outbreak under discussion only three of the nine kittens died. The dehydration and constipation of the autopsied kitten can be attributed to the tongue lesions which made lapping painful.

Rohovsky and Fowler [10] in a study of FPL in germ-free cats reported that none of the inoculated cats died and that enteritis did not occur. Thus it would appear that enteritis in FPL is not due primarily to the virus but due to bacteria present in the gastro-intestinal tract. The variety of antibiotics used by the breeder would certainly have depressed, if not modified, the intestinal flora in the affected kittens. The absence of lesions in the small intestine in this case is in marked contradistinction to classical FPL but nevertheless does not preclude a diagnosis of FPL.

In a survey [1], it was reported that the characteristic sternal recumbency or "praying position" and the hanging posture of the head over the water bowl was considered to be a significant diagnostic sign of FPL. It was further reported that mouth and tongue ulcers sometimes accompanied FPL. Carpenter [3] reported that a variety of lesions, including necrotic gingivitis and ulcerative glossitis, may occur in cats affected with FPL.

Langheinrich and Nielsen [8] commenting on the significance of intranuclear inclusion bodies in FPL considered their presence to be diagnostic, especially when correlated with the other histological lesions, but stated that they may be absent. The associated histopathological changes which they described included reticuloendothelial hyperplasia, lymphocytic depletion, loss of follicular architecture, haemorrhage, congestion, reticuloendothelial hypoplasia and plasma cell infiltration in the spleen. There is in addition a severe depletion of neutrophils, while the occurrence of erythrophagocytosis in lymph nodes and the spleen in FPL is common knowledge.

Glossitis has been reported in a number of other feline viral diseases. Wilkinson [13] reported ulceration of the tongue in feline viral rhinotracheitis (FVR). No lesions of the upper respiratory system were noticed in the cat reported on here. Hoover and Griesemer [4] working with germ-free cats reported that severe upper respiratory symptoms were a constant finding in FVR-infected cats.

Hoover and Kahn [5] investigated the lesions produced by feline picornaviruses of high and low virulence in pathogen-free cats and reported that ulcerative glossitis occurred in both cases. Furthermore, the highly-virulent virus always produced pneumonia, whereas the virus of low virulence produced mainly a lingual ulceration. They did not mention any pathological changes in the liver or spleen.

O'Reilly, Paterson and Harris [9] in an experiment to determine the persistence of maternal antibody to FPL in kittens found that the queens' antibody titres declined in the absence of exposure to natural infection. Furthermore, they found that six out of 10 kittens born to queens with an antibody titre of 1:32 had FPL antibodies 4 weeks after birth, but that by 6 weeks of age only three out of 16 kittens had a detectable antibody titre. After 11 weeks no antibodies were detectable in any of the kittens. In their study the antibody titre levels in the kittens varied from 1:32 to less than 1:8, thus only 28% of kittens from queens with low antibody titres had detectable antibody levels at 6 to 7 weeks of age.

No reference can be found concerning the variation in antibody titres within the same litter of kittens; however, in a study undertaken on piglets, Nordbring and Olsson (cited by Brambell [2], 1970) showed that at 6 weeks of age titres from colostral antibody to a paratyphoid vaccine in a litter of piglets varied between 1:160 to 1:320. Since both these species are polytocous, an analogous situation may well exist in cats.

The demonstration of eosinophilic intranuclear inclusion bodies in the lingual epithelium supports the theory that FPL virus, or a strain thereof, may be the aetiological agent in this outbreak of ulcerative glossitis.

In any attempt to explain the pathogenesis of ulcerative glossitis and its relationship to classical FPL there would appear to be an interesting interrelationship between the waning antibody levels of the affected kittens, the predilection of the virus for rapidly multiplying cells and the particular location of the lesions, which in this situation appears to be confined to the tongue – an organ whose epithelial surface is subject to considerable cellular replacement and possibly a mild degree of trauma in the suckling kitten.

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BOOK REVIEW

RESENSIE

ECOLOGY AND CONTROL OF RODENTS OF PUBLIC HEALTH IMPORTANCE

TECHNICAL REPORT SERIES NO. 553 W.H.O. 1974

pp. 42, Annex 1, Tables 1, Price SW fr. 5. -

This Report of a WHO Scientific Group which met in 1973 deals in a brief concise and systematic way with the importance of rodents in public health as vectors of diseases transmissible to man, the ecology of these rodents, control and management of rodents, and recommendations. It includes a chapter on Information and Training of the public and rodent control personnel, and an Annexure with a table providing detailed information on the relevant rodent species and the associated human disease. A list of 78 references is provided for persons who require further detailed information.

The report is a valuable document for all who are interested in Zoonoses, Environmental Health and Sanitation

L.W. v.d. H.

Chimpanzee-Associated Hepatitis

Between May 22 and May 28, 1974, 5 persons in Cumberland County, Pennsylvania, developed jaundice, and diagnoses of hepatitis were made. Serum specimens obtained from these individuals and tested for hepatitis B surface antigen (HBsAg) were negative.

None of the patients had been exposed to known cases of hepatitis, raw shell fish or contaminated food or water. However, all 5 patients had had contact with a young, newly imported chimpanzee. The 12-month-old chimpanzee had arrived at a privately owned zoo on April 10, 1974, thin and highly nervous with dry, scaly skin. Also, she had a poor appetite and persistent diarrhea.

On May 1 the chimp was treated by a local veterinarian and cared for by the assistant of another veterinarian in her home. Subsequently, over a 7-day period in May, the chimpanzee owner (age 53) and his wife (55), a part-time employee (17), the veterianarian's assistant (20) and her boyfriend (24), all of whom had frequent contact with the chimpanzee, developed acute HBsAg-negative hepatitis.

Fifty-two contacts of the 5 patients received immune serum globulin (ISG). No additional cases of hepatitis occured.

A blood specimen obtained from the implicated chimpanzee revealed an SGOT of 85 IU (chimpanzee normal 0-15 IU) and a bilirubin of 2.0 mg% (chimpanzee normal 0.1-0.5%). A cage-mate of the implicated chimpanzee appeared healthy, but blood tests revealed a normal bilirubin with an SGOT of 81 IU.

Since the first reports of nonhuman primate-associated hepatitis in the early 1960s, over 200 cases in humans have been reported, and the frequency of such reports seems to be increasing. In 1974, 8 outbreaks in 7 states were reported to CDC. The disease is usually mild, of brief duration, and clinically indistinguishable from hepatitis A. Various nonhuman primates have been associated with cases of hepatitis in humans, but the most frequently implicated have been chimpanzees that were newly imported and appeared well or had nonspecific clinical illness.

Persons who must work with newly imported chimps are advised to maintain scrupulous personal hygiene, and since immune serum globulin seems to protect animal handlers against clinical hepatitis, they should receive ISG routinely.

Source: Center for Disease Control (U.S. Dept. of Health, Education and Welfare): Morbidity and Mortality Weekly Report 24(12):22 March 1975