

Chorioretinitis And Detached Retina As Post-Distemper Lesions in the Canine

by

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Summary

In the canine species there are many causes of chorioretinitis and detachment of the retina. Among these are: fungal infections such as Blastomycosis and Coccidioidomycosis; invasion by Toxoplasma organisms; trauma to structures of the eye with subsequent hemorrhage, infection or rupture of the vitreous; tumors of the choroid or retina; parasitic larval migrans such as Toxacara canis; progressive hereditary diseases; post-systemic diseases such as distemper; and those of unknown etiology.¹ The ophthalmoscopic lesions of the case presented were those of a bilateral, chronic chorioretinitis with a unilateral detachment of the retina suspected of being due to a delayed post-distemper virus infection reaction.

Introduction

Due to the juxtaposition of the retina to the choroid, both structures usually are involved in an inflammatory process. Chorioretinitis by definition means primary inflammation of the choroid with subsequent spread of the inflammatory process to the retina. The retina is attached to the choroid only at the optic disk and at the ora serrata. It is held against the choroid due to the pressure of the vitreous.⁹ Thus the retina can be easily detached due to loss of vitreous pressure, presence of inflammatory exudates, or hemorrhage between the choroid and the retina. Consequently, chorioretinitis often leads to a partial or complete detachment of the retina with resultant impairment or complete loss of vision.

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Case Report #63037

On December 2, 1974 a 3 year old male Blue Coonhound was admitted to the Stange Memorial Clinic. The dog exhibited an ataxia with the toes spread wide in splay fashion as he walked. He also appeared weak and was showing some dysmetria of the front legs. The first signs of front leg lameness had appeared ten days following hunting for coons thus a Coondog paralysis was suspected. The dog had been given butazolidine tablets at that time. Upon close examination it was noted that the pupils were dilated and by ophthalmoscopic exam a severe retinitis was discovered in both eyes. The right eye showed chronic scarring lesions and the left eye had a detached retina. At this stage chorioretinitis and detached retina were diagnosed with a possible cerebellar lesion as a cause of the dysmetria. Since the dog was not vaccinated for canine distemper a post-distemper demyelination was also suspected. Further examination revealed anisocoria with the left pupil dilated more than the right one. The left eye was not responding to light with direct or consensual pupillary response from the left to the right eye. Direct and consensual pupillary response from the right to the left eye was normal.

Differential diagnosis for the dysmetria of the forelimbs included a space occupying lesion of the cerebellum, Coondog paralysis, distemper, tick paralysis and rabies. The dilatation of the left pupil with no light response could have been due to a lesion in the area of cranial nerves II and/or III. Some of the differentials considered as possible causes of the eye lesions were distemper, toxoplasmosis, progressive retinal atrophy, or detached retina

due to trauma or some unknown etiology.

Blood taken for clinical pathology analysis on Dec. 3rd revealed no abnormalities. Serum for the toxoplasmosis titer was found to be negative. On Dec. 5th a cerebrospinal fluid tap was taken and the results are as follows:

Pandy test—negative

Glucose—72

Blood glucose—113

Cells—0

Culture—negative

A complete ophthalmoscopic exam of the left eye revealed several superficial lines across the cornea, cataract formation on both the anterior and posterior capsules of the lens and a detached retina. The right eye showed multifocal areas of chorioretinitis suspected of being old distemper lesions.

The dog was released on Dec 13, 1974 after close observation and no treatment. His condition appeared to be getting better. He was less ataxic now and his toes were less splayed than when admitted. The damage to the retina was thought to be permanent, although the dog had some vision in the right eye, he was completely blind in the left eye.

Discussion

Canine distemper is the most common cause of chorioretinitis in the dog.³ According to experimental research reported by Parry¹² in 1953, twenty-five dogs were proven to have canine distemper by viral inclusion body isolation from the renal pelvis, urinary bladder, bronchial epithelium or the brain. All but two of these dogs showed degeneration of the retinal ganglion cells upon histopathological examination, although these dogs did not necessarily exhibit signs of blindness. These lesions were thought to be due to primary localization of the virus in retinal tissues or due to secondary extension of viral infection from the brain through the optic nerve to the retina. Thus it is important to perform an ophthalmoscopic exam on any animal suspected of having canine distemper in order to determine the extent of chorioretinal involvement

and prognosis for vision of the animal.

Ophthalmoscopically, chorioretinitis lesions have been divided into two forms: (1) active chorioretinitis and (2) inactive or chronic chorioretinitis, which is the form most recognized by clinicians due to the fact that active inflammation often obscures good observation of the retina. Active chorioretinitis can be characterized ophthalmoscopically by cellular infiltration, which appears as dull grey areas with indistinct borders in the tapetal region and white or grey areas in the non-tapetal region, usually accompanied by retinal exudates, edema and perivascular cuffing. Bullous retinal detachment can occur and appear as well delineated cystic spaces between the retina and the pigment epithelium.¹ Inactive or chronic chorioretinitis, on the other hand, is characterized by retinal atrophy and scarring either focal or diffuse. This appears as bright, hyperreflective areas in the tapetal region and pale areas in the non-tapetal region where the pigment epithelium has atrophied. In addition, pigment epithelial proliferation can also occur in which the lesions themselves are invaded by focal, multifocal or diffuse pigmentation. The decreased caliber of the retinal vessels indicate thinning of the retina.

In many cases chorioretinitis progresses causing a partial or complete separation of the retina from the overlying pigment epithelium. Ophthalmoscopically, retinal detachment appears as if the retina has been folded over maybe even obscuring the optic disk. The retina is a silver grey color and it undulates with movement of the eye as if it were floating free in the vitreous. The retinal vessels are tortuous and appear elevated whereas the choroid vessels are hard to envision. A sharp line appears at the junction of the attached and unattached portions and the detachment usually continues to spread to the rest of the retina resulting in complete loss of vision.

The histological lesions caused by infection with canine distemper virus with respect to the eye are as follows: necrosis and degeneration of retinal ganglion cells with accompanying edema and perivascu-

lar cuffing, swelling and proliferation of retinal pigment epithelial cells, inclusion bodies found in the retinal and optic nerve glial cells, local and diffuse atrophy of the rods and cones, sometimes with a loss of retinal layer organization, and CNS inflammation and demyelination of the optic nerve and its tracts.¹⁰

Treatment

The treatment of chorioretinitis medically has had questionable results due to the fact that many of the cases are not recognized until they have reached the chronic stages and retinal detachment with permanent scarring has already occurred resulting in clinical symptoms of blindness. If diagnosed early, however, medical treatment of the inflammatory, exudative type of chorioretinitis with retinal detachment has had some good results. The therapeutic regimen of steroids, Diuril^a, and chloramphenicol when indicated has resulted in absorption of subretinal fluid with reattachment of the retina and return of vision.¹¹ Possible use of systemic enzymes such as Kymar^b may be helpful. This treatment is recommended for at least two weeks before a hopeless prognosis is given.¹¹ It should be noted, however, that the condition must be recognized in its very early stages and therapy initiated immediately in order to obtain any response to medical treatments.

In man there are various techniques used to correct a detached retina such as perforating diathermy, electrolysis, scleral resection, surface coagulation, photocoagulation, cryo freezing technique¹¹ and the laser beam method. Few of these methods have been used in the canine species, however a surgical technique has been used by Rubin.^{13,14} In this method a lateral canthotomy is performed and the conjunctiva is incised 6 mm. from the limbus in an 80° arc laterally. A stay suture is placed in the lateral rectus muscle, it is transected and freed laterally. A 1.5 mm. electrode needle is used to burn a series of holes 1.5 to 2.0 mm. apart in

the sclera overlying the retinal detachment in a triangular pattern 6 mm. from the limbus to 1 mm. from the optic nerve. The muscle and conjunctiva are closed separately with #0000 catgut and the canthotomy incision closed with #0000 silk. Post-operative treatment includes atropine to maintain dilation of the eye and 2.5 mg. prednisolone given subconjunctivally. Dexamethasone drops 0.1% are given q.i.d. for six days and 5 mg. prednisolone orally for three days. Benzathine penicillin, 400,000 units, has also been given at the conclusion of the surgery. This procedure caused destruction and retinal atrophy in the area burned but prevented the rest of the retina from detaching thus resulting in only a partial loss of vision.

Conclusion

In conclusion, the canine distemper virus can often lead to chorioretinitis and more serious retinal detachment with resultant loss of vision. This can occur either during acute infection with the virus even a few years post-infection. Thus, it is important to perform an ophthalmoscopic exam on dogs suspected of having canine distemper in order to determine the extent of ocular lesions and to initiate therapy early. History of an unvaccinated dog, past systemic disease with or without accompanying nervous signs, and sudden onset of blindness can help differentiate this consequence of distemper from progressive retinal atrophy or other causes of chorioretinal lesions and detached retina.

Bibliography

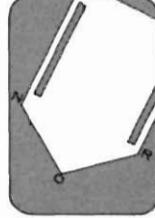
1. Aguirre, Gustavo V.M.D. *The Retina*, AAHA Continuing Education Series, 1974.
2. Aguirre, Gustavo V.M.D. "Retinopathies", *Kirk's Current Veterinary Therapy V*, Philadelphia: W. B. Saunders Co., 1974, pp. 501-5.
3. Albert, R. A. "Lesions of the ocular fundus associated with systemic disease", *JAVMA* Vol. 157 No.11, Dev. 1, 1970, pp. 1635-9.
4. Appel, M. J. G., "Distemper pathogenesis in dogs" *JAVMA* Vol. 156, June 15, 1970, pp. 1681-3.
5. Barnett, K. C. *Variations of the Normal Ocular Fundus of the Dog*, AAHA publication 1972.
6. Bistner, Stephen I. "Examination of the eye", *Kirk's Current Veterinary Therapy V*, Philadelphia: W. B. Saunders Co., 1974, pp. 467-8.
7. Bistner, Stephen I. "Ocular manifestation of systemic disease", *Vet. Clinics of N.A.* Vol. 3 No. 3, Sept., 1973.
8. Fischer, C. A. "Retinal and retinochoroidal lesions in early neuropathic canine distemper", *JAVMA* Vol. 158, 1971, p. 740.

a. Diuril, chlorothiazide, Merck, Sharpe & Dohme.

b. Kymar, Chymotrypsin, Armour.

9. Jenson, H. E. *Stereoscopic Atlas of Clinical Ophthalmology of Domestic Animals*, St. Louis: C. V. Mosby Co., 1971.
10. Jubb, K. V., Saunders, L. Z. and Coates, H. V., "The Intraocular lesions of Canine distemper", *J. Comp. Path & Therap.* Vol. 67, 1957, pp. 21-9.
11. Magrane, Wm. G., *Canine Ophthalmology*, Philadelphia: Lea and Febiger, 1971.
12. Parry, H. *Brit. Journal Ophthal.* Vol. 37, 1953 p. 37, Vol.38, p. 295.
13. Paton, H. J. et al. *Atlas of Eye Surgery* 2nd ed. New York: McGraw-Hill Book Co., 1962, pp. 162-5
14. Rubin, L. F. *Atlas of Veterinary Ophthalmology*, Philadelphia: Lea and Febiger, 1973.
15. Rubin, L. F. "Correction of retinal detachment in a dog", *JAVMA* Vol. 157, Aug. 15, 1970, pp. 461-6.
16. Vainisi, S. J. *The Canine and Feline Ocular Fundus* AAHA publication, 1965.

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Will the Real Feline Respiratory Disease Please Stand Up?

by
Char Slindee*

Feline respiratory diseases are a serious and frustrating problem to cat owners and veterinarians alike. They are highly contagious and difficult to treat. In recent years, a number of agents have been recognized as causing or contributing to different forms of respiratory disease. Complicating the problem, the clinical signs of each are very similar, requiring laboratory facilities for specific diagnosis. The vet-

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erinarian must be aware of the organisms most commonly encountered in order to gain the most value from a preventative program of vaccination.

In the past, "pneumonitis" was thought to be almost synonymous with feline respiratory disease. Recent extensive research indicates this disease, caused by a chlamydia, actually plays a much less significant role. Numerous agents producing indistinguishable clinical signs have been rec-