



CASE REPORT

Progressive retinal atrophy in Turkish Shepherd Dog; A case report

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Türk Çoban Köpeğinde progresif retinal atrofi; Bir vaka raporu

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Öz

Progresif retinal atrofi (PRA), köpeklerde yaygın görülen retinanın kalıtsal dejeneratif bozukluğudur. Sunulan vaka 9 yaşlı, dişi Türk Çoban Köpeği, Küçük hayvan hastanesine poliüri ve stranguri şikayetleri ile getirildi. Muayene sonrası akut hepatit tanısı konuldu. Medikal tedavide (ursofalk, deve dikenini özü, prednisolone ve assist) uygulandıktan üç gün sonra görme kaybı oluştu. Oftalmolojik muayeneler, slit lamb, Schirmer göz yaşı testi, aplanasyon tonometresi, floresein boyası ve ultrason ile yapıldı. Oftalmoskopik ve fundoskopik muayeneler sonucunda, tapetal bölgesinde hiperreflektivite ve renk değişikliği, retina damarlarında belirgin zayıflama, non-tapetal bölgede depigmentasyon ve sınırlı optik disk atrofi gözlemlendi. Fundoskopik muayene, her iki gözde de retina dejenerasyonunu gösterdi. Bu olgu sunumunun amacı Türk Çoban Köpeklerinde PRA teşhisi ve sonuçlarını klinisyenler ile paylaşmaktır.

Anahtar kelimeler: Funduskopi, Köpek, Progresif retinal atrofi

Abstract

Progressive retinal atrophy (PRA) is described as an inherited degenerative disease of the retina that is common in dogs. The case was a 9-year-old female Turkish shepherd dog. It was presented to the Small Animal Hospital with complaints of polyuria and stranguria. After examination, the diagnosis was acute hepatitis. Medical treatment (ursofalk, milk thistle, prednisolone and N acetyl cystein) was given. The case lost vision after three days of treatment. The ophthalmological examinations were performed as included slit lamp, Schirmer's tear test, applanation tonometry, fluorescein staining and ultrasound. Ophthalmoscopic and fundoscopic examinations revealed hyperreflexivity and colour change in the tapetal region, marked attenuation of retinal vessels, depigmentation in the non-tapetal region, and optic disc atrophy with scalloped borders. Fundoscopic examination revealed retinal degeneration in both eyes. The aim of this case report is to diagnose PRA in Turkish shepherd dogs, to highlight its significance and to share the results with clinicians.

Keywords: Dog, Funduscopy, Progressive retinal atrophy



Introduction

Progressive retinal atrophy (PRA) describes hereditary/genetic degenerative retinal diseases in dogs and cats. The term is used to describe a range of hereditary neuroretinal degenerations that occur with different mutations. There is genetic heterogeneity affecting over 100 dog breeds (Bedford 2006, Kelawala et al 2017). The retina contains photoreceptors that convert light into electrical nerve signals. Several different inherited types of photoreceptors are affected. This interaction leads to loss of night vision. As the disease progresses, cone vision deteriorates, leading to visual impairment in daylight and eventually to complete blindness (Narfström et al 2002). The first problems may be seen in puppies at 12 weeks of age. Clinically, the dog may go blind at 1-2 years of age. Various degenerative forms may begin in early adulthood, as the dog matures, or between 9 and 11 years of age. Degenerative changes are most common in purebred dogs, but can also be seen in crossbreeds. The breeds most commonly affected include Miniature Poodles, Tibetan Terriers, American and English Cocker Spaniels, Papillions, Samoyeds, Longhaired Dachshunds, Labrador Retrievers, Akitas, Tibetan Spaniels and many more. The disease is not painful, so it is rarely noticed in the early stages of its development. Night blindness is often the first sign noticed in a dog with PRA. Diagnosis is based on clinical history, ophthalmology, funduscopy, electroretinography (ERG) and genetic testing (Gomes et al 2013).

The aim of this case report is to diagnose PRA in Turkish shepherd dogs, to highlight its significance and to share the results with clinicians.

Case Presentation

The case involved a nine-year-old female Turkish shepherd. It was presented to the small animal hospital with complaints of polyuria and stranguria. A clinical examination and laboratory tests (blood, faeces and urine) were carried out. Acute hepatitis was diagnosed (ALT 332.52, AST 505.21, CPK 3209.66, total bilirubin 1.79, direct bilirubin 0.71, LDH 387.78). The dog was positive for *Giardia* spp. Urinalysis values were within normal reference range, but medical treatment was suggested as follows: ursodeoxycholic acid 10 days (10 mg/kg, PO, Ursafalk Kapsul, Dr. Falk Pharma GmbH, Germany), milk thistle 10 days, PO, prednisolone 3 days (2.2 mg/kg, im, Prednol-L 250 mg, Mustafa Nevzat İlaç San. A.Ş., Turkey), N-acetylsystein for 10 days (70 mg/kg, im, Asist 300 mg/3 ml, İdol İlaç Dolum Tic. A.Ş., Turkey). At the beginning of treatment, 3 days later, blood values were reassessed (ALT 126.55, AST 43.64, CPK 237.98, total bilirubin 5.17, direct bilirubin 1.93, LDH 43.23). On the third day after the treatment, the patient was examined for loss of vision. Both eyes were found to become blind but progressed slowly. The dog's history was of loss of vision at night. After that, the dog

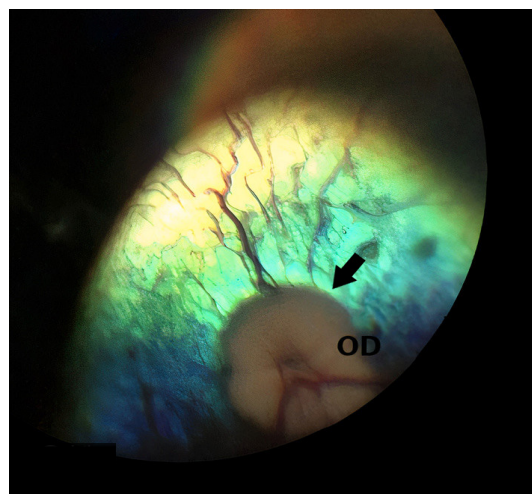


Figure 1. The retinal examination was started with funduscopy of the optic disc. OD (optic disc).

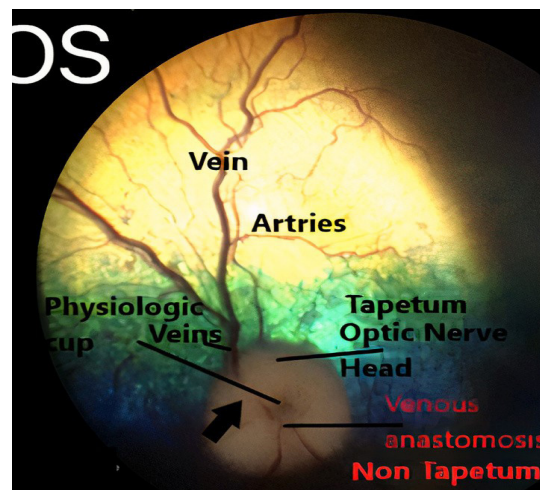


Figure 2. Examination showed loss of the physiological cup and peripapillary oedema (black arrow).

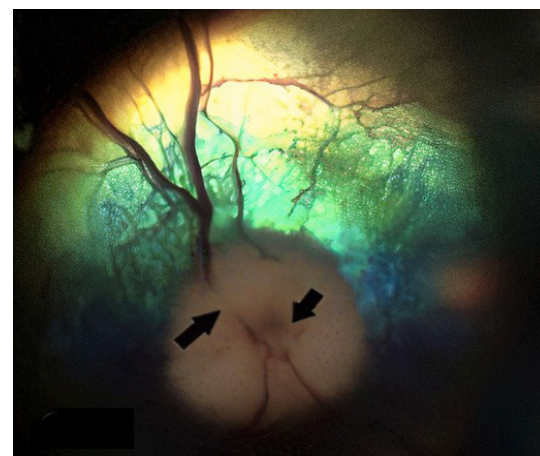


Figure 3. Fundoscopic examination showed early progressive retinal atrophy. Some of the blood vessels were completely attenuated (black arrows). Hyperreflectivity of the tapetum.



lost vision in the light. The owners' history indicated that the dog would be unwilling to go out at night, enter dark rooms, or go up or down stairs.

A threat response was used to start the ophthalmological examination. Pupillary reflexes were assessed with a focal light source. The results of the Schirmer tear test were recorded for both eyes at 10 mm. Intraocular pressure (IOP) was measured with a tonometer (Tonovet plus®, iCare, USA). The pressures in the left and right eyes were recorded as 16 mmHg and 20 mmHg, respectively. The anterior segments were examined with a slit lamp (HSL-150+X-99-105, Heine, Germany). The fundus was examined with an indirect ophthalmoscope (ClearView® 2 retinal camera, Eickemeyer, Germany). 20 min after tropicamide (Mydriacyl®, Alcon Inc., Puurs, Belgium) instillation, fundus photographs were taken with a fundus camera (iPod®, Apple, USA). No retinal detachment was observed by ultrasonography. Only fibrin formation was observed in the vitreous cavity. Hyperreflectivity and discolouration started in the tapetal area, the discolouration of the tapetal area was not clear (Figure 1). However, depigmentation had started in the non-tapetal area. There was clear evidence of marked attenuation of the retinal vessels, depigmentation in the non-tapetal area and optic disc atrophy with scalloped margins (Figure 2-3). Multivitamins (Dr. Nature's Dog Immune, Turkey) and NSAID (0.2 mg/kg, sc, Meloxicam, Bavet Meloksikam, Bavet, Turkey) were administered. No change was observed after 10 days. The case was followed up for 8 months. The liver problems were cured, but the vision in both eyes did not return.

There is no cure for inherited retinal diseases in humans and dogs. It is one of the leading causes of vision loss. For dogs to have PRA, they must be born with a defective gene (Reddy et al 2015). There are at least 24 mutations in 18 genes that cause retinal disease in dogs. The DNA-based blood test that helps confirm the presence of the faulty gene. It can be congenital or can develop later (Clements et al 1996, Miyadera et al 2012). In this case, the visual impairment developed after a metabolic disease. The affected gene was not analysed. There is no information on when the disease process started. As complete loss of vision does not occur at the onset of the disease, it takes time. For this reason, early diagnosis of the disease is difficult without routine screening. There are some cases with retinal examination in Turkish shepherd dogs (Şaroğlu et al 2005). There is currently no suitable treatment for PRA. There is a lot of research going on to help both pets and people affected by these conditions (Cooper et al 2014, Reddy et al 2015, Svensson et al 2016). There are a number of vitamin therapies. However, there is no evidence that vitamins have any therapeutic effect. In this case, vitamin A and multivitamins have been used. The aim is to help the affected dog adapt and maintain a high quality of life. ERG showed abnormal rod and cone responses

in affected dogs at the ages studied (Narfström et al 2002, Sandmeyer et al 2006). The ERG test measures the electrical response of the retina to light. This test is particularly useful in assessing retinal disease. An ERG was not performed in this case report. If it had been, the diagnosis would have been more accurate.

In conclusion, PRA is a common genetic problem in dogs. Frequent eye examinations are necessary in certain breeds. ERG examination is required in addition to fundoscopic examination for diagnosis. Gene banks could be established for Turkish shepherd dogs for early diagnosis of the disease. Early eradication of these cases would be possible. The case presented is intended to raise awareness among clinicians.

Conflict of Interest

The authors did not report any conflict of interest or financial support.

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Author Contributions

Motivation/Concept: MA; Design: MA; Control/Supervision: MA; Data Collection and/or Processing: MA, EOU, YT, HC, IS; Analysis and/or Interpretation: MA, EOU, YT, HC, IS; Literature Review: MA; Writing the Article: MA; Critical Review: MA

Ethical Approval

Selcuk University Faculty of Veterinary Medicine Experimental Animal Production and Research Center Ethics Committee (SÜVDAMEK) decided that "There is no need for an Ethics Committee Decision" (Decision No: 2023/583149). The authors confirm that written informed consent was obtained from the patient owner.