



RETINAL DISEASES OF SENIOR DOGS

Balicka, A., Lapsanska, M., Trbolova, A.

Small Animals Clinic
University of Veterinary Medicine and Pharmacy in Košice, Komenského 73, 041 81 Košice
Slovakia

agnieszka.a.balicka@gmail.com

ABSTRACT

Aging consists of a physiological decline of an organism's functional activity. During the aging process, the structural and functional changes of the retina can be observed. In most cases, progressive vision loss occurs due to the age related changes of the anterior segment. Retinal diseases, characteristic for senior dogs are: retinal detachment, hypertensive chorioretinopathy, sudden acquired retinal degeneration syndrome (SARDS), progressive retinal atrophy (PRA), glaucoma, retinopathy, cystoid degeneration and neoplasms. The examination of the retina in senior dogs is based on: ophthalmoscopic examination, electroretinography, spectral-domain optical coherence tomography (AD-OCT) and if necessary, histopathological examinations. Comprehensive knowledge regarding the senior dog's health, significantly increases their quality of life.

Key words: senior dogs; retina; vision loss; retinopathy

INTRODUCTION

An animal's life can be divided into four stages: paediatric, adult, senior (mature, middle age) and geriatric (called senior or super senior) [14]. The understanding of ageing varies between different authors [9, 14, 34]. The progress of ageing in the individual species depends upon its size, breed, genetics and conditions under which they exist. In 2012, Fortney published "The current human pet analogy chart", that helps to assess an animal's life stage, based on its weight and age. This chart has been widely used in many studies, which required assigning patients to different age groups [14].

The main reasons for vision loss of geriatric dogs are not only the commonly occurring cataracts or corneal diseases, but also retinal disorders. Retinal diseases in aging dogs are usually secondary. In many dogs, vision loss is the consequences of systemic diseases or other ophthalmic problems like retinal degeneration due to high intraocular pressure (IOP). The primary problem is typically cases affected with hereditary or congenital eye diseases with a progressive character.

The process of aging of the retina varies between species. Generally, during aging, the retinal inner limiting membrane becomes thick and vacuolated. In the inner and outer plexiform layers, cystoid spaces may be observed. The outer nuclear layer of the peripheral retina progressively thins [15]. According to a study performed in dogs using SD-OCT, the whole retinal thickness in geriatric dogs is thinner than in the middle aged dogs [6]. The understanding of the aging retina develops with every performed study, and requires further analysis [46].

The changes in the senses, including vision, hearing, and olfaction are common problems in ageing dogs causing a decrease ability to react to stimuli [9]. There are several “normal” aging changes which occur in dog’s and cat’s eyes like: lenticular sclerosis, iris atrophy, asteroid hyalosis, or endothelial degeneration of the cornea [11]. In many cases, the main reason for the ophthalmic consultations are problems with the vision caused by age-related diseases.

This report reviews some of the most common retinal problems observed in geriatric dogs.

Retinal detachment

Systemic hypertension is a common problem in ageing dogs and cats and are often the primary cause of retinal detachments. Although the prevalence of hypertension in dogs and cats is not known, an annual screening of cats and dogs older than 9 years old, is recommended [1]. Retinal detachments due to systemic hypertension, often occurs in patients with heart or kidney failure. The main sign of retinal detachment is partial or complete vision loss depending on the extent of the detachment. It can be diagnosed during ophthalmic examination using behavioural testing (menace response) and ophthalmoscopy. In electroretinography, the results may be normal, partially attenuated, or attenuated depending on the severity of the condition. Additionally, an ultrasound examination (USG) of the posterior eye segment can demonstrate the typical appearance of the condition called “seagull wings”. During ultrasound, it can be visible that a detached retina remains attached in the area of the optic nerve head and *ora serrata* [43]. In some cases, retinal detachments cannot be seen with the ophthalmoscope or on USG. In these cases, optical coherence tomography (OCT) is required to scan the whole retinal thickness and detect the origins of the flat focal retinal separations [33]. A typical detachment occurs between the retinal pigmented epithelium and the photoreceptor layer

due to its embryological development. In early eye development, the retina is made with two layers of epithelium of neuroectoderm origin. The outer epithelial layer differentiates into the retinal pigment epithelium, while the inner epithelial layer differentiates into the sensory neuroretina. These two layers may be separate due to accumulation of fluids in the subretinal space [35, 43]. Patients with retinal detachments require complete ophthalmic and physical examinations. The condition is not painful, but it may cause pain due to its primary cause like: trauma, inflammation, etc. The treatment of retinal detachments require emergency service as a detached retina needs metabolites and diffusion of oxygen from the separated choroid. The medical treatment is mainly based on the treatment of the primary cause. Progression of a partial detachment may be prevented by laser retinopexy. It is important to remember that a blind eye is not painful, does not require enucleation, and blindness is not a reason for euthanasia [43, 48].

Hypertensive chorioretinopathy

Ocular lesions are common in dogs with systemic hypertension [28]. Retinal and choroidal vascular diseases may be caused by systemic hypertension secondary to renal or endocrine diseases [47], e.g. *diabetes mellitus* which is a common problem in geriatric dogs [43]. The typical fundus lesions that can be observed in dogs with hypertensive chorioretinopathy are: papilledema, narrowing of the retinal vessels, retinal oedema, serous detachment, retinal and vitreal haemorrhages [54]. Systemic hypertension can be the main cause, not only of retinal detachment but also of retinal haemorrhages which usually develop as the disease progresses. It is possible to classify the depth of haemorrhages based on the ophthalmoscopy appearance: subretinal (bleeding from the choroidal vessels between the retina and choroid), intraretinal (usually manifest as focal petechiae bleeding from the deep capillaries of the retinal vessels), or preretinal (bleeding between the retina and vitreous from retinal vessels). Subretinal haemorrhage can lead to retinal detachments [43]. An analysis of 83 dogs diagnosed with punctate retinal haemorrhage revealed a mean age of the patients of 10 years $\pm$ 3.8 [55]. A study performed on 42 hypertensive dogs revealed that 40 % of them had retinal haemorrhages [28]. In dogs affected with acute blindness due to initial clinical signs, such as intraocular haemorrhage, retinal detachment, or secondary glaucoma, hypertensive chorioretinopathy, should be taken under consideration [30].

Diabetic retinopathy

Diabetic retinopathy is a less common manifestation of systemic diseases. The typical ocular manifestation of *diabetes mellitus* (DM) in dogs are cataracts. In one study, 80 % of the dogs affected with DM developed cataracts within 16 months of the diagnosis [7]. According to H e r r i n g et al. [20], about 20 % of the diabetic dogs had retinal petechiae. Retinal haemorrhages involve: retinal capillary degeneration, micro aneurysms, or even can cause small retinal detachments that can be observed in the ophthalmoscopic examinations. These changes may lead to vision impairment, which often occurs in humans affected with *diabetes mellitus* [43]. DM tends to occur in middle aged to older dogs (5—12 years of age) [12]. Retinopathies in dogs occur less often than in humans [20].

Sudden acquired retinal degeneration syndrome (SARDS)

Sudden acquired retinal degeneration syndrome has only been diagnosed in dogs. The typical reported sign of the disease is an acute blindness with a normal-looking fundus [43]. SARDS affects middle-aged to elderly dogs and often moderately overweight, aged between 7—10 years [2, 3, 51, 53]. According to the studies, the majority of the affected dogs are spayed females [5, 24]. Until now, genetic predisposition has not been documented. An analysis of breed predisposition revealed that SARDS occurs most commonly in mixed-breed dogs. It has also been reported that SARDS occurs more often in small breed dog (Dachshund, Miniature Schnauzer, Pug, Brittany, Bichon Frise, Beagle, Maltese, American Cocker Spaniel, Pomeranian, and possibly Shihtzu) [5, 10, 19, 51, 53].

Typical for SARDS is that a rapid vision loss develops over a period of days to weeks; this has been reported in the USA since the 1980s. Previously, the condition had been known as “toxic metabolic retinopathy” or “silent retina syndrome”. SARDS can be diagnosed using electroretinography (ERG). This examination helps to distinguish SARDS from central blindness, in which the manifestation of the disease is similar—acute blindness, but a normal looking fundus can be seen. In dogs with central blindness, the ERG is normal, while in SARDS cases, it is distinguished due to the loss of photoreceptor outer segments and the numbers of apoptotic nuclei in the outer nuclear layer can be seen in the histopathology examinations [43]. Similar findings were revealed after analyses of 10 dogs affected

with SARDS using OCT [44]. Although the primary cause of the disease is still not known, endocrine disorders have been suspected due to the common history of SARDS patients, i. e. polyuria, polydipsia, polyphagia, lethargy, and obesity. Currently, autoimmune inflammatory processes and toxicity have been taken under consideration as a primal reason in case of sudden acquired retinal degeneration syndrome. Even today, there is no proven treatment for SARDS, so affected dogs remain blind, although systemic signs usually decrease over time [43].

Progressive retinal atrophy (PRA)

Progressive retinal atrophy is a broad general term that describes a number of inherited neuroretinal degenerations [8]. Although PRA can affect even young animals, very often it is a terminal stage, when the owner recognise eyesight problems are occurring in advanced age. The analysis of 31 dogs with PRA revealed that 48 % were diagnosed between 6—10 years and 26 % were diagnosed between 11—15 years of age [22]. PRA is a hereditary disease that initially affect rods, leading to loss of night-vision. Complete blindness occurs when the disease develops and cones starts to be affected [42]. Because the disease is incurable, it is important to determine the diagnosis before breeding [39]. It has been found that PRA is an autosomal recessive inherited disease, but X-linked forms and one autosomal dominant form has also been reported [40]. The diagnosis is based upon the clinical history and complete ophthalmic examinations with fundoscopy and electroretinography (ERG) [17]. During ophthalmoscopic examination hyperreflectivity that develops as a result of thinning neurosensory retina and the thinning of blood vessels can be seen as a characteristic for PRA. In terminal stages, the complete atrophy of the retina occurs. Abnormalities, like vitreous liquefaction (syneresis), asteroid hyalosis and cataracts are also observed in the later stages of PRA [18, 50].

Glaucoma related fundus changes

Glaucoma has many definitions. Generally, it is a group of diseases united by increased intraocular pressure (IOP). According to M a g g i o, it is a neurodegenerative disorder of the retinal ganglion cells and the optic nerve, causing blindness [31]. Glaucoma is a common ocular emergency in veterinary practice. The value of IOP is a consequence of balance between aqueous humour production and outflow

[31]; both decline with age [36]. Primary glaucoma is associated with increasing age [4, 25, 26], e.g. primary close angle glaucoma can occur in any age but most affected dogs are middle aged to older [16]. The value of intraocular pressure for certain species varies between different sources and different devices. According to Slatter's Ophthalmology, the normal range of IOP in dogs is 8–25 mmHg [37, 38] or 19.2 ± 5.5 mmHg or 12.9 ± 2.7 mmHg using applanation tonometry and 10.8 ± 3.1 mmHg or 9.1 ± 3.4 mmHg using rebound tonometry [31, 36]. In the case of glaucoma, an unresponsive dilated pupil can often be present [38]. Mydriasis or a poorly responsive pupil is a consequence of increased IOP that is causing ischemia, pressure induced paresis, or paralysis of the iris sphincter muscles [32]. Highly increased IOP for longer than 24 to 72 hours leads to irreversible blindness [36]. Vision loss occurs due to the damage of ganglion cell axons in the optic disc region. The damage is caused by increased IOP that is causing negative effects on the vascular supply of the optic nerve and axonal transport in optic nerve axons [32]. During the fundus examination, the optic nerve cupping and retinal and optic nerve atrophy can be observed. Cupping of the optic disc is due to the bowed outward optic nerve head. It is caused by an increased IOP on the *lamina cribrosa*. Another fundoscopic finding is the common hyperreflectivity due to retinal thinning and attenuation of the retinal vessels [32]. There are various types of glaucoma that should be managed according to aetiology, presence or absence of vision, and the stage of the disease [31].

Primary neoplasms of the retina—canine retinal gliomas (astrocytoma)

Although retinal glioma rarely occurs in canine patients, Glial Fibrillary Acid Protein-positive gliomas are the most frequently encountered primary retinal tumours in dogs. Gliomas most often arise in the central retina, near or continuous with the optic nerve. It can have a bland or an anaplastic cellular profile and can be extensively necrotic [13]. Gliomas in the central nervous systems usually occur in brachycephalic dog breeds [23, 29, 49]. According to studies, the affected dogs are middle-aged to old with the mean age of 9 years in which enucleation is performed, similarly to previous descriptions of gliomas of the central nervous system [23, 49, 52]. The diagnosis of glioma is reached based on a signalment and ocular examination with ultrasound examination of the eye globe, supported

with other ocular imaging tests. A histopathology examination and eventually immunohistochemistry is necessary in order to confirm the diagnosis. In the literature there is an interesting clinical case which describes a 9.5 year old spayed female miniature Schnauzer with the history of a unilateral 1-week old ocular problem. Employing a B-mode ultrasonographic examination using 10 MHz, the authors demonstrated a mushroom-shaped, relatively homogenous hyperechoic mass arising from the mid-dorsal chorioretinal region. The histopathology examination of the mass revealed a retinal glioma [41]. Other investigators have described ocular changes of retinal gliomas such as: intra ocular haemorrhage, glaucoma, and retinal detachment with its typical clinical signs [27, 41]. The metastatic potential of canine retinal glioma (astrocytoma) is low, but ascending invasion into the ventral aspect of the brain should be considered [41].

Cystoid degeneration of the retina

The other rarely diagnosed ocular disease that occurs in senior dogs is cystoid degeneration [45]. However, an analysis performed on age-related changes in the eyes of 86 Beagle dogs revealed that 85 % of the animals were affected with cystoid degeneration of the retina. The cysts were single or multiple at or near the ora ciliaris retinae [21]. With age, the inner limiting membrane becomes thickened and increasingly vacuolated. The inner and outer plexiform layers can have cystoid spaces, which are formed within Mullerian cells [46]. Peripherally, the retina is adjacent to the ora ciliaris retinae; this region undergoes cystic changes with multiple cystic structures that protrude into the vitreous. These changes may lead to retinal detachment. Because the changes occur peripherally, they tend not to affect the vision [42].

ACKNOWLEDGEMENT

The study was supported by the project VEGA No. 1/0479/18.

REFERENCES

1. **Acierno, M. J., Brown, S., Coleman, A. E., et al., 2018:** ACVIM consensus statement: Guidelines for the identification, evaluation, and management of systemic hypertension in dogs and cats. *J. Vet. Intern. Med.*, 32, 6, 1803—1822. DOI: 10.1111/jvim.15331.
2. **Acland, G. M., Aguirre, G. D., 1986:** Sudden acquired retinal degeneration: clinical signs and diagnosis. *Trans. Am. Coll. Vet. Ophthalmol.*, 17, 58—63.
3. **Acland, G. M., Irby, N. L., Aguirre, G. D., et al., 1984:** Sudden acquired retinal degeneration in the dog: clinical and morphologic characterization of the “silent retina” syndrome. *Trans. Am. Coll. Vet. Ophthalmol.*, 15, 86—104.
4. **Ahonen, S. J., Pietilä, E., Mellersh, C. S., Tiira, K., Hansen, L., Johnson, G. S., Lohi, H., 2013:** Genome-wide association study identifies a novel canine glaucoma locus. *PLOS ONE*, 8, e70903. DOI: 10.1371/journal.pone.0070903.
5. **Auten, C. R., Thomasy, S. M., Kass, P. H., Good, K. L., Hollingsworth, S. R., Maggs, D. J., 2018:** Cofactors associated with Sudden Acquired Retinal Degeneration Syndrome: 151 dogs within a reference population. *Vet. Ophthalmol.*, 21, 3, 264—272. DOI: 10.1111/vop.12504.
6. **Balicki, I., Balicka, A., Szadkowski, M., Trbolova, A., 2019:** Assessment of the geriatric retina in mixed breed dogs using spectral domain optical coherence tomography (SD-OCT). In Proc. Ann. Sci. Meet. Europ. Coll. Vet. Ophthalmol., Antwerp, Belgium, May 23—26. *Vet. Ophthalmol.*, 22, E1—E27.
7. **Beam, S., Correa, M. T., Davidson, M. G., 1999:** A retrospective-cohort study on the development of cataracts in dogs with *diabetes mellitus*: 200 cases. *Vet. Ophthalmol.*, 2, 3, 169—172. DOI: 10.1046/j.1463-5224.1999.00073.x.
8. **Bedford, P., 2006:** Hereditary retinal diseases. In *Proceedings of World Congress WSAVA/FECAVA/CSAVA*, Prague, Czech Republic, October 11—14, 609—610.
9. **Bellows, J., Colitz, C. M. H., Daristotle, L., et al., 2015:** Common physical and functional changes associated with aging in dogs. *J. Am. Vet. Med. Assoc.*, 246, 1, 67—75. DOI: 10.2460/javma.246.1.67.
10. **Braus, B. K., Hauck, S. M., Amann, B., Heinrich, C., Fritsche, J., Köstlin, R., Deeg, C. A., 2008:** Neuron-specific enolase antibodies in patients with sudden acquired retinal degeneration syndrome. *Vet. Immunol. Immunopathol.*, 124, 1—2, 177—183. DOI: 10.1016/j.vetimm.2008.02.020.
11. **Bromberg, N. M., 2013:** *Your Dog Needs Bifocals—Geriatric Changes in the Eyes of Dogs and Cats*. https://www.sagecenters.com/wp-content/uploads/sites/4/Brochure_Geriatric_6-10.pdf.
12. **Catchpole, B., Ristic, J. M., Fleeman, L. M., Davison, L. J., 2005:** Canine *diabetes mellitus*: can old dogs teach us new tricks? *Diabetologia*, 48, 10, 1948—1956. DOI: 10.1007/s00125-005-1921-1.
13. **Dubielzig, R. R., Ketrung, K., McLellan, G. J., Albert, D. M., 2010:** *Veterinary Ocular Pathology*. Elsevier, Philadelphia, 456 pp.
14. **Fortney, W. D., 2012:** Implementing a successful senior/geriatric health care program for veterinarians, veterinary technicians, and office managers. *Vet. Clin. North. Am. Small Anim. Pract.*, 42, 4, 823—834. DOI: 10.1016/j.cvsm.2012.04.011.
15. **Gao, H., Hollyfield, J. G., 1992:** Ageing of the human retina. Differential loss of neurons and retinal pigment epithelial cells. *Invest. Ophthalmol. Vis. Sci.*, 33, 1, 1—17.
16. **Gelatt, K. N., MacKay, E. O., 2004:** Prevalence of the breed-related glaucomas in pure-bred dogs in North America. *Vet. Ophthalmol.*, 7, 2, 97—111. DOI: 10.1111/j.1463-5224.2004.04006.x.
17. **Gomes, D., Otsuki, D. A., Lisaki, R., De Mendonça, Safatle Vaz, A., 2013:** Generalized progressive retinal atrophy in Cocker Spaniel dogs. *Cienc. Rural.*, 43, 1405—1414.
18. **Grahn, B. H., Peiffer, R. L., 2013:** Veterinary ophthalmic pathology. In **Gelatt, K. N., Gilger, B. C., Kern, T. J.:** *Veterinary Ophthalmology*. 5th edn., Wiley-Blackwell, New Jersey, 435—523.
19. **Grozdanic, S. D., Lazic, T., 2013:** Early detection of autoimmune retinopathies (SARD and IMR) in dogs with normal day vision (Abstract). In Proc. 44th Ann. Meet. Am. Coll. Vet. Ophthalmol., Puerto Rico, USA, November 4—9. *Vet. Ophthalmol.*, 16, 6, E26—E50.
20. **Herring, I. P., Panciera, D. L., Werre, S. R., 2014:** Longitudinal prevalence of hypertension, proteinuria, and retinopathy in dogs with spontaneous *diabetes mellitus*. *J. Vet. Intern. Med.*, 28, 2, 488—495. DOI: 10.1111/jvim.12286.
21. **Heywood, R., Hepworth, P. L., Abbe, N. J., 1976:** Age changes in the eyes of the Beagle dog. *J. Small Anim. Pract.*, 17, 3, 171—177. DOI: 10.1111/j.1748-5827.1976.tb06588.x.
22. **Kelawala, D. N., Patil, D. B., Parikh, P. V., Sheth, M. J., Joshi, C. G., Reddy, B., 2017:** Clinical studies on progressive retinal atrophy in 31 dogs. *Iran J. Vet. Res.*, 18, 2, 119—123.
23. **Koestner, A., Higgins, R. J., 2002:** Tumours of the nervous system. In **Meuten, D. J.:** *Tumours in Domestic Animals*, 4th edn., Iowa State Press, Ames, 697—738.
24. **Komáromy, A. M., Abrams, K. L., Heckenlively, J. R., Lun-**

- dy, S. K., Maggs, D. J., Leeth, C. M., et al., 2016: Sudden acquired retinal degeneration syndrome (SARDS)—a review and proposed strategies toward a better understanding of pathogenesis, early diagnosis, and therapy. *Vet. Ophthalmol.*, 19, 4, 319—331. DOI: 10.1111/vop.12291.
25. Kuchtey, J., Kunkel, J., Esson, D., Sapienza, J. S., Ward, D. A., Plummer, C. E., et al., 2013: Screening ADAMTS10 in dog populations supports Gly661Arg as the glaucoma-causing variant in beagles. *Invest. Ophthalmol. Vis. Sci.*, 54, 3, 1881—1886. DOI: 10.1167/iovs.12-10796.
26. Kuchtey, J., Olson, L. M., Rinkoski, T., Mackay, E. O., Iverson, T. M., Gelatt, K. N., et al., 2011: Mapping of the disease locus and identification of ADAMTS10 as a candidate gene in a canine model of primary open angle glaucoma. *PLOS Genetics*, 7, 2, e1001306.
27. Kuroki, K., Kice, N., Ota-Kuroki, J., 2017: Retinal astrocytoma in a dog. *Can. Vet. J.*, 58, 9, 919—922.
28. Leblanc, N. L., Stepien, R. L., Bentley, E., 2011: Ocular lesions associated with systemic hypertension in dogs: 65 cases (2005—2007). *J. Am. Vet. Med. Assoc.*, 238, 7, 915—921. DOI: 10.2460/javma.238.7.915.
29. LeCoteur, R. A., Withrow, S. J., 2007: Tumors of the nervous system. In Withrow, S., Vail, D.: *Withrow and Mac Ewen's Small Animal Clinical Oncology*. 4th edn., Saunders Elsevier, St. Louis, 659—685.
30. Littman, M. P., Robertson, J. L., Bovée, K. C., 1988: Spontaneous systemic hypertension in dogs: five cases (1981—1983). *J. Am. Vet. Med. Assoc.*, 193, 4, 486—494.
31. Maggio, F., 2015: Glaucomas. *Top. Companion Anim. Med.*, 30, 3, 86—96. DOI: 10.1053/j.tcam.2015.07.011.
32. McLellan, G. J., Narfstrom, K., 2014: The fundus. In Gould, D., McLellan, G. J.: *BSAVA Manual of Canine and Feline Ophthalmology*. 3rd edn., BSAVA, Gloucester, 322—356.
33. McLellan, G. J., Rasmussen, C. A., 2012: Optical coherence tomography for the evaluation of retinal and optic nerve morphology in animal subjects: practical considerations. *Vet. Ophthalmol.*, 15, 2, 13—28. DOI: 10.1111/j.1463-5224.2012.01045.x.
34. Metzger, F. L., 2005: Senior and geriatric care programs for veterinarians. *Vet. Clin. North Am. Small Anim. Pract.*, 35, 3, 743—753. DOI: 10.1016/j.cvsm.2004.12.005.
35. Miller, P. E., 2017: The eye and the vision. In Maggs, D. J., Miller, P. E., Ofri, R.: *Slatter's Fundamentals of Veterinary Ophthalmology*. 6th edn., Saunders Elsevier, St Louis, 1—17.
36. Miller, P. E., 2017: The glaucomas. In Maggs, D. J., Miller, P. E., Ofri, R.: *Slatter's Fundamentals of Veterinary Ophthalmology*. 6th edn., Saunders Elsevier, St Louis, 279—305.
37. Miller, P. E., Pickett, J. P., Majors, L. J., Kurzman, I. D., 1991: Evaluation of two applanation tonometers in cats. *Am. J. Vet. Res.*, 52, 1, 1917—1921.
38. Miller, P. E., et al, 1991: Clinical comparison of the Mackay Marg and Tono Pen applanation tonometers in the dogs. *Prog. Vet. Compar. Ophthalmol.*, 1, 171—176.
39. Millichamp, N. J., Curtis, R., Barnett, K. C., 1988: Progressive retinal atrophy in Tibetan terriers. *J. Am. Vet. Med. Assoc.*, 192, 6, 769—776.
40. Miyadera, K., Acland, G. M., Aguirre, G. D., 2012: Genetic and phenotypic variations of inherited retinal diseases in dogs: the power of within- and across-breed studies. *Mamm. Genome*, 23, 1—2, 40—6. DOI: 10.1007/s00335-011-9361-3.
41. Naranjo, C., Schobert, C., Dubielzig, R. R., 2008: Canine ocular gliomas: A retrospective study. *Vet. Ophthalmol.*, 11, 6, 356—362. DOI: 10.1111/j.1463-5224.2008.00658.x.
42. Narfstrom, K., Petersen-Jones, S., 2013: Diseases of the canine ocular fundus. In Gelatt, K. N., Gilger, B. C., Kern, T. J.: *Veterinary Ophthalmology*. 5th edn., Wiley-Blackwell, New Jersey, 1303—1392.
43. Ofri, R., 2017: Disease of retina. In Maggs, D. J., Miller, P. E., Ofri, R.: *Slatter's Fundamentals of Veterinary Ophthalmology*. 6th edn., Saunders Elsevier, St Louis, 347—389.
44. Osinchuk, S. C., Leis, M. L., Salpeter, E. M., Sandmeyer, L. S., Grahn, B. H., 2019: Evaluation of retinal morphology of canine sudden acquired retinal degeneration syndrome using optical coherence tomography and fluorescein angiography. *Vet. Ophthalmol.*, 22, 4, 398—406. DOI: 10.1111/vop.12602.
45. Rubin, L. F., 1974: *Atlas of Veterinary Ophthalmoscopy*. Lea and Febiger, Philadelphia, 83 pp.
46. Samuelson, D. A., 2013: Ophthalmic anatomy. In Gelatt, K. N., Gilger, B. C., Kern, T. J.: *Veterinary Ophthalmology*. 5th edn., Wiley-Blackwell, New Jersey, 39-170.
47. Sansom, J., Bodey, A., 1997: Ocular signs in four dogs with hypertension. *Vet. Rec.*, 140, 23, 593—598. DOI: 10.1136/vr.140.23.593.
48. Schmidt, G. M., Vainisi, S. J., 2004: Retrospective study of prophylactic random trans scleral retinopexy in the Bichon Frise with cataract. *Vet. Ophthalmol.*, 7, 5, 307—310. DOI: 10.1111/j.1463-5224.2004.04046.x.
49. Snyder, J. M., Shofer, F. S., Van Winkle, T. J., et al., 2006: Canine intracranial primary neoplasia: 173 cases (1986—2003). *J. Vet. Intern. Med.*, 20, 3, 669—675. DOI: 10.1892/0891-6640(2006)20[669:cipnc]2.0.co;2.

50. Stades, F. C., Wyman, M., Boeve, M., Neumann, W., Spiess, B., 2007: *Ophthalmology for the Veterinary Practitioner*. 2nd edn., Schlutersche, Hannover, 257 pp.
51. Stuckey, J. A., Pearce, J. W., Giuliano, E. A., Cohn, L. A., Bentley, E., Rankin, A. J., et al., 2013: Long-term outcome of sudden acquired retinal degeneration syndrome in dogs. *J. Am. Vet. Med. Assoc.*, 243, 10, 1425—1431. DOI: 10.2460/javma.243.10.1426.
52. Summers, B. A., Cummings. J. F., De Lahunta, A., 1995: *Veterinary Neuropathology*. Mosby, St. Louis, 527 pp.
53. Van der Woerd, A., Nasisse, M. P., Davidson, M. G., 1991: Sudden acquired retinal degeneration in the dog: clinical and laboratory findings in 36 cases. *PVCO*, 1, 11—18.
54. Villagrasa, M., Cascales, M. J., 2000: Arterial hypertension: angiographic aspects of the ocular fundus in dogs. A study of 24 cases. *J. Am. Vet. Med. Assoc.*, 238, 7, 915—921.
55. Violette, N. P, Ledbetter, E. C., 2018: Punctate retinal haemorrhage and its relation to ocular and systemic disease in dogs: 83 cases. *Vet. Ophthalmol.*, 21, 3, 233—239. DOI: 10.1111/vop.12496.

Received September 24, 2020

Accepted November 2, 2020