

## Combined iridencleisis and posterior sclerectomy in surgical treatment of glaucoma secondary to uveitis in a dog: a case report

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**ABSTRACT:** In early diagnosed and causally treated cases of secondary glaucoma, it is possible to save the sight in a glaucomatous eye and to preserve the sight in a healthy eye. The most common causes of secondary glaucoma include anterior uveitis, in which diverse pathological processes can lead to an increase in IOP. In cases of treating glaucoma secondary to uveitis with annular posterior synechiae in connection with peripheral anterior synechiae, iridencleisis is the method of choice. This article presents the results of the eight-month treatment of such a case with iridencleisis combined with posterior sclerectomy. This method makes it possible to increase the area of aqueous humour outflow to the subconjunctival space. The treatment resulted in the reduction of IOP in the left eye from the preoperative value of 72 mmHg to 15 mmHg, which was maintained at a constant level during the eight-month period following the surgery. Sight assessment in the operated eye, carried out on the basis of PLR, menace and dazzle response, brought a moderately positive result.

**Keywords:** iridencleisis; sclerectomy; secondary glaucoma; dog

Secondary glaucoma is the most common form of glaucoma in dogs (Smith et al., 1993; Barnett et al., 2002; Barnett, 2006). Unlike primary glaucoma, it is not a hereditary disease, but its causes may be (Curtis and Barnett, 1980; Gelatt and Brooks, 1999; Johnsen et al., 2006). In contrast to primary glaucoma, secondary glaucoma may involve only one eye; therefore, if the primary disease is diagnosed and the treatment is started early enough, the patient may not lose vision (Johnsen et al., 2006).

Among the most common causes of secondary glaucoma in dogs are, anterior uveitis (Crispin, 1988; Abrams, 2001; Johnsen et al., 2006), lens displacement (Curtis and Barnett, 1980; Abrams, 2001; Gelatt and Gelatt, 2001; Gelatt and MacKay, 2004; Morris and Dubielzig, 2005; Johnsen et al., 2006), intumescent cataract (phacomorphic glaucoma) (Gelatt and Brooks, 1999; Gelatt and MacKay, 2004), intraocular cysts (Deehr and Dubielzig, 1998; Spiess et al., 1998; Sapienza et al., 2000), hyphema (Nelms et al., 1993; Sansom et al., 1994; Barnett et

al., 2002), intraocular neoplasia (Dubielzig, 1990), ocular melanosis (pigmentary glaucoma) (Petersen Jones, 1991; van de Sandt et al., 2003).

An accurate estimate of the most common direct cause of secondary glaucoma in dogs is possible with retrospective statistical studies. Therefore, it is obvious that the longer the period under study, the more representative the results will be. The results of a retrospective study by Johnsen et al. (2006) of a 5-year period indicate that the direct cause of over three quarters of all the cases of secondary glaucoma in dogs are anterior uveitis and lens dislocation. With the estimation criteria narrowed down, nonsurgical anterior uveitis alone accounted for nearly 45% of the group of dogs.

Different data have been reported by Gelatt and MacKay (2004), who presented the results of a retrospective study of a 39-year period. According to the results of the study, over 80% of secondary glaucoma cases in dogs are caused by cataract formation and only 7.1% are related to uveitis of

an unknown cause. However, uveitis, secondary to cataract surgery, lens displacement or hyphema have not been included in the group, which would make it much larger.

Differences in the size of the groups may result from the differences between a classification of direct causes of secondary glaucoma and the seamless transformation of one disease stage into another. Several different pathological processes may be distinguished, which result in an identical clinical picture, i.e., elevated IOP and secondary glaucoma (Abrams, 2001; Gelatt and Gelatt, 2001; Renwick and Petersen Jones, 2001). For example, anterior lens luxation may cause anterior iris displacement, resulting in narrowing or closure of the iridocorneal angle, iritis, or iridocyclitis, peripheral anterior synechiae and, consequently, narrowing or occlusion of the entrance to the ciliary cleft. The process may also run along a different pathway, i.e., anterior lens luxation may cause pupillary block secondary to annular posterior synechiae (*seclusio pupillae*), resulting in iris bombe, iritis and peripheral anterior synechiae and the closure of the entrance to the ciliary cleft. Slight anterior lens luxation may cause anterior iris displacement, similarly to cases of intumescent cataract, where increased lens causes anterior iris displacement and its resulting inflammation. Anterior lens luxation may ultimately fail to cause a significant iris displacement, but it may block the outflow of aqueous humour on the trabecular meshwork level through inflammatory cells, melanocytes, erythrocytes from hyphaema, lymphocytes from hypopyon and other cellular debris released due to iritis (Renwick and Petersen Jones, 2001; Barnett et al., 2002). Iritis itself may create pre-iridal fibrovascular membranes, which may stretch across the pupil, thereby increasing pupillary resistance or a total pupillary blockage. It may also migrate radially, covering the entrance to the ciliary cleft (Peiffer et al., 1990; Smith et al., 1993; Deehr and Dubielzig, 1998; Gelatt and Gelatt, 2001; Renwick and Petersen Jones, 2001; Barnett et al., 2002).

Although different, all the disease pathways presented above, result in a slow or rapid increase in IOP and secondary glaucoma. A reverse process to the ones described above may also occur, namely, chronic uveitis may weaken lens zonules and result in lens luxation (Renwick and Petersen Jones, 2001). Therefore, diseases thought to be causes of secondary glaucoma may in some cases actually be symptoms (Johnsen et al., 2006).

Veterinary ophthalmologists agree that there is no method of glaucoma treatment with long-term effectiveness. However, the most commonly recommended surgical procedure in glaucoma secondary to uveitis with annular posterior synechiae, associated with peripheral anterior synechiae, is iridencleisis (Gelatt and Brooks, 1999; Gelatt and Gelatt, 2001; Strubbe, 2002). It is a procedure which combines the features of filtration and drainage and consists in incarceration of a portion of the iris in corneoscleral incision, thereby creating a filtering cicatrix. Various modifications of the procedure are known: Holth's or Wecker's techniques (Gupta et al., 1966), one or two-limb iridencleisis, or iridencleisis with sclerectomy (Nirankari and Malhotra, 1965).

This paper presents a case of glaucoma secondary to uveitis, treated by surgery employing iridencleisis combined with posterior sclerectomy.

### Case description

A 4-year-old Central Asian shepherd dog was referred to the Surgery Clinic with an eye disease. It was established in an anamnesis that for over three weeks the patient had been treated for idiopathic uveitis of the left eye and that steroid medicines had been administered systemically and topically. An initial improvement was followed by a rapid (three day) increase in the intraocular pressure (IOP) and corneal opacity, which had been the reason for the referral of the patient to the centre.

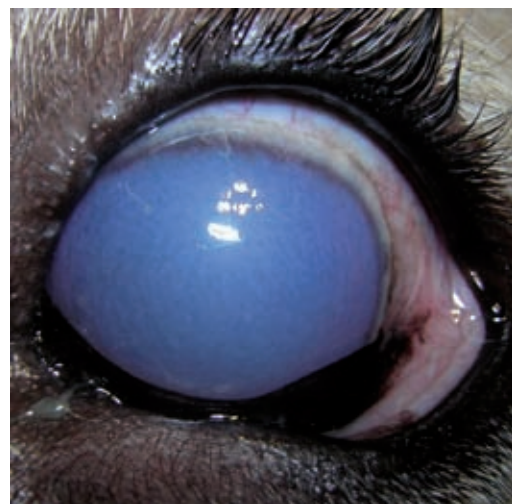


Figure 1. Dense corneal oedema with vascular fringe along the limbus

A clinical examination of the left eye revealed dense corneal oedema with characteristic vascular fringe (Figure 1). IOP measured with an applanation tonometer (TonoPen XL, Reichert, USA) reached 72 mmHg for the left eye and 20 mmHg for the right eye. Gonioscopic and fundus examination was impossible to perform due to lack of transparency of the cornea. Consensual pupillary light reflex (PLR) in the right eye was not significant. Menace response, dazzle response and maze testing in the left eye gave negative results. Routine, general clinical and laboratory examinations performed on the patient did not demonstrate any deviations from the reference values. A hemogram, as well as a serum chemistry profile were normal.

Anterior uveitis and a rapid increase in the IOP raised the concern that annular posterior synechiae had been created leading to pupillary obstruction. A differential diagnosis was made, glaucoma secondary to anterior uveitis.

Due to the extremely high IOP, the case was considered an emergency and as such qualified for immediate surgery.

Immediately before the surgery, osmotic reduction of IOP was applied with a Mannitol 20% solution (Mannitol, Polfa Lublin, Poland) administered intravenously at 2 g/kg of body weight.

The patient was anaesthetized according to the following anaesthetic protocol: pre-medication with Atropine sulphate (Atropinum sulfuricum, Polfa, Poland) at a dose of 0.05 mg/kg of body weight and Acepromazine maleate (Calmivet, Vetoquinol, France) at a dose of 0.5 mg/kg of body weight, administered *i.m.* Anaesthesia was induced by a combination of xylazine (Rometa, SPOFA, Czech Republic) at a dose of 1 mg/kg of body weight, administered *i.m.* and, after inserting a vascular cannula with ketamine (Bioketan, Biovet, Poland), at a dose of 5 mg/kg of body weight, administered *i.v.* to the cephalic vein. The dog was then intubated. The anaesthesia was maintained with isoflurane (Isoflurane, Abbott, Great Britain) at a concentration of 1–1.5%.

Before the surgery, general anaesthesia was completed with a 3-fold local anaesthesia of the conjunctival sac using Proxymetacaine hydrochloride (Alcaine, Alcon Couvreur, Belgium) at a dose of two drops at 5-min intervals. 1% tropicamide (Mydriacyl, Alcon Couvreur, Belgium) and 10% Phenylephrine hydrochloride (Neo-Synephrine, Sanofi-Winthrop, USA) were also administered twice at 10-minute intervals, half an hour before the surgery.

Analgesic treatment was conducted with Tramadol hydrochloride (Tramal 100, Polpharma S.A., Poland) at a dose of 5 mg/kg of body weight *i.m.* The treatments were continued until day 2 after the surgery.

Following the surgical preparation of the surgery field, the eyelids were fixed with blepharostat. For atraumatic detachment of ocular conjunctiva from Tenon's capsule and sclera, subconjunctival injection of 1 ml of balanced salt solution was made at the 12 o'clock position. A Fornix-based conjunctival flap was made at a distance of 2 mm away from the limbus with a base of 12 mm and length of approximately 14 mm. After the removal of the Tenon's capsule, a full-thickness 3 × 6 mm block of sclera was cut out, 4 mm away from the limbus. Bleeding from the intrascleral plexus venosus of Hovius was stopped by monopolar microdiathermia. The edges of the window cut out in the sclera were coagulated with extreme caution.

An iridial hook was introduced between the ciliary body and the iris root into the anterior chamber parallel to the sclera. After an iris fragment was transposed to the window cut out in the sclera, it was grasped with a pair of iris forceps and torn towards its base, the operation being controlled by diathermia. Each of the created pillars was sutured at two ends of the window to the sclera with a simple interrupted 6–0 absorbable suture (Figure 2). Subsequently, the anterior chamber was rinsed with a balanced Ringer lactate solution heated to body temperature. The window in the sclera remained unclosed. The wound in the conjunctiva was closed with a 6–0 simple continuous absorbable suture (Figure 3).

Postoperative management included local treatment according to the following procedure: Dexamethasone (Dexamethason 0.1%, Polfa Warsaw, Poland) QID topical, and Gentamicin sulfate (Gentamicin 0.3%, Polfa Warsaw, Poland) QID topical, in both eyes. Mydriasis and cycloplegia was reached by administration of 1% tropicamide (Mydriacyl, Alcon Couvreur, Belgium) and 10% Phenylephrine hydrochloride (Neo-Synephrine, Sanofi-Winthrop, USA) BID topical in left eye.

The treatment procedure described above was followed for four weeks. To assure low IOP, the application of Dorzolamide hydrochloride (Trusopt, Merck Sharp&Dohme, France) BID topical was continued until the 6<sup>th</sup> week. Solcoseryl (Solcoseryl, Solco Basel, Switzerland) QID topical was administered additionally.

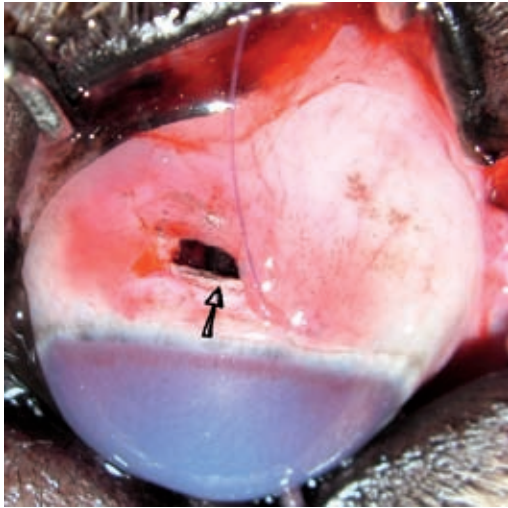


Figure 2. Posterior sclerectomy with two iris pillars sutured at two ends of the window

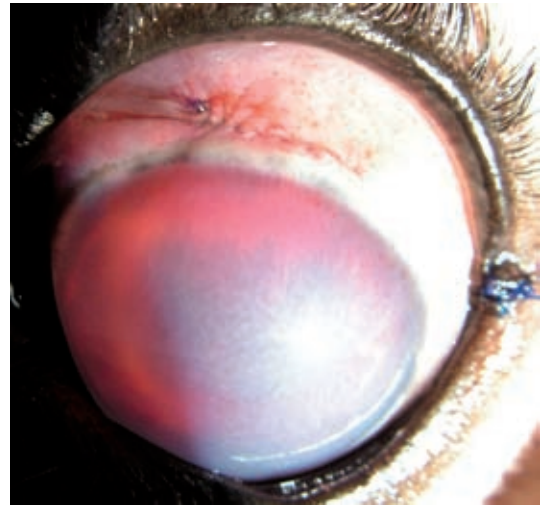


Figure 3. Conjunctiva – closed with simple continuous suture

A post-surgery IOP measurement revealed 8 mmHg in the left eye and 20 mmHg in the right eye. On day 3 after the surgery, IOP was 12 mmHg in the left eye and was unchanged in the right eye. Corneal oedema subsided during the next 15 days, making ophthalmologic examination possible. A gonioscopic examination revealed a narrow angle and local peripheral anterior synechiae.

Local posterior synechiae and iris pigment deposition on the lens capsule were found and at the site where irydenclieisis had been performed, an enlarged dorsal pupil was observed (Figure 4). The anterior lens capsule was slightly opaque, which impeded any fundus examination. An ophthalmo-



Figure 4. Appearance of the left eye four weeks after operation – local posterior synechiae, iris pigment deposition on the lens capsule and enlarged dorsal pupil at the site of irydenclieisis

scopic examination of the left fundus showed slight attenuation of the retinal vessels and very slight optic nerve head cupping. Ophthalmoscopy of the right fundus did not demonstrate any deviations from the norm.

Additional assessment of the vision in the eye that had been operated on and in the healthy one was based on PLR, menace and dazzle response, and maze testing on days 1, 3, 7 and 15 following the surgery. The first three tests, i.e., conducted on days 1, 3 and 7 following the surgery, were negative for the left eye. The vision in the right eye remained unchanged. On day 15 after the surgery, direct PLR in the left eye was not significant, but consensual PLR in the right eye was distinct. An additional assessment of the vision in the eye that had been operated on, based on maze testing, was negative, but menace response and dazzle response yielded positive results. The vision tests described above were conducted once a month, until the 8<sup>th</sup> month following the surgery, and similar results were obtained.

Weekly IOP measurements in the left eye, conducted until month 8 following the surgery, yielded a constant value of 15 mmHg. It was recommended that the dog's guardian should perform massages of the filtration bleb several times each day.

## RESULTS AND DISCUSSION

In cases of anterior uveitis, IOP is initially low, but it is indisputably numbered among the poten-

tial causes of secondary glaucoma (Abrams, 2001; Johnsen et al., 2006). Miosis, plication, atonia and oedema of the iris may rapidly create synechiae. Focal, and then annular posterior synechiae, cause pupillary block, iris bombe and, as a consequence, cause secondary glaucoma (Crispin, 1988; Bedford and Jones, 2001; Gelatt and Gelatt, 2001; Strubbe, 2002). Additional factors which favour pupillary occlusion are fibrin clots and deposition of the inflammatory cell and small keratic precipitates (Crispin, 1988). Iris bombe and resulting shallowing of the anterior chamber favour the creation of peripheral anterior synechiae, narrowing, followed by occlusion of the entrance to the ciliary cleft, which, when combined with pupillary seclusion, may result in a rapid and considerable increase in IOP (Bedford and Jones, 2001). Mechanical resistance of aqueous humour circulation is accompanied by filtration resistance caused by the blocking of the trabecular meshwork with inflammatory cells and migrating melanophores, released from the iris and ciliary body (Crispin, 1988; Girkin et al., 2005; Reilly et al., 2005; Johnsen et al., 2006).

In such cases, IOP may remain extremely high despite pharmacological treatment (Crispin, 1988). Therefore, despite the absence of full ophthalmologic examination, i.e., the absence of fundus examination and gonioscopy, the patient qualified as emergency and a decision was taken to perform surgery (Abrams, 2001; Johnsen et al., 2006). At this stage of treatment, the main purpose was to normalise the IOP and protect the optic nerve (Abrams, 2001; Grozdanic et al., 2007; Ofri and Narfstrom, 2007).

Surgeries which restore the circulation of the aqueous humour in pupillary seclusion include basal or radial iridectomy and iridencleisis (Strubbe, 2002). The first two become useless in cases where posterior synechiae are accompanied by peripheral anterior synechiae. The method of choice in surgical treatment of pupillary block glaucoma is iridencleisis (Gelatt and Gelatt, 2001; Strubbe, 2002). It helps restore the flow of aqueous humour from the posterior chamber to the anterior chamber and further to the subconjunctival space by a window cut out in the sclera (Nirankari and Malhotra, 1965; Cook, 1997; Gelatt and Brooks, 1999; Gelatt and Gelatt, 2001; Strubbe, 2002). In the classic understanding of Weeker's or Holth's iridencleisis, this surgery involves making a small window in the sclera in the shape of a narrow fissure – anterior sclerectomy (Nirankari and Malhotra, 1965; Gupta et al., 1966).

In the case in question, trans-scleral iridencleisis was performed with posterior sclerectomy, with the dimensions of 3 × 6 mm, which hindered the transposition and anchoring of the iris pillars on the one hand, but enlarged the drainage area to the subconjunctival space on the other. The effect of IOP reduction was increased by cyclodialysis performed in the section connecting the iris root with the ciliary body under the window in the sclera.

Additional coagulation of the edge of the window in the sclera with monopolar diatermia was to performed to reduce fibrosis and obliteration of the wound, thereby retaining the patency of the fistula.

Minimising the fibrosis of the scleral fistula is crucial in the action of the filtration bleb (Gelatt and Gelatt, 2001; Strubbe, 2002).

Early diagnosis and aggressive treatment are of key importance in preventing the development of glaucoma secondary to uveitis (Johnsen et al., 2006). The decision to perform an immediate surgery and the post-surgery pharmacological therapy were aimed at reducing the progressive loss of vision in the left eye and of preventing uveitis in the right eye, respectively. The preventive actions against iridocyclitis may stop secondary glaucoma in the contralateral eye and help avoid a total loss of vision (Bedford, 1985; Johnsen et al., 2006). For this reason, the anti-inflammatory steroid therapy of the right eye was implemented.

The pharmacological treatment implemented was similar to that applied by other authors and was aimed not only at reducing IOP, but also targeted the primary cause of the disease (Crispin, 1988; Abrams, 2001; Strubbe, 2002; Johnsen et al., 2006).

The techniques of surgical treatment of glaucoma have been developed based on human anatomy. The existing differences between the anatomical structure of human and dog eyes, especially filtration angle, trabecular meshwork and vascular supply, hinder, or even make impossible, application of identical procedures in each case of treating glaucoma of the same aetiology (Calkins, 1960; Gelatt and Gelatt, 2001). Iridotomy, or tearing the iris, when performed during the iridencleisis procedure in humans, and sclerotomy or sclerectomy, do not pose a serious problem. In the case of dogs, the presence in about 50% of the animals of an iridial vascular ring and intrascleral plexus venosus of Hovius instead of Schlemm's canal in humans, makes the surgery in dogs both more difficult tech-

nically and bears a more serious risk of intraocular haemorrhage and resulting hyphema.

The most common complications following iridencleisis include intraocular hemorrhage, posterior synechiae, iris pigment deposition on the lens capsule, cataract formation, intraocular infection, postoperative iridocyclitis, and closure of the iridencleisis (Nirankari and Malhotra, 1965; Gupta et al., 1966; Nagpaul et al., 1966; Gelatt and Brooks, 1999; Gelatt and Gelatt 2001; Strubbe, 2002). Of the complications mentioned above, only local posterior synechiae and iris pigment deposition on the lens capsule occurred during the eight months follow-up observations; these may well have been caused by uveitis from the pre-surgery period.

The non-significant PLR in the left eye, observed on day 15 after the surgery, and distinct consensual PLR in the right eye may have been caused by local posterior synechiae and may have indicated a certain vision in the left eye. The supposition may be confirmed by positive results of menace response and dazzle response.

The IOP of 15 mm Hg, which remained constant throughout the entire 8-month post-surgical period, indicates the effectiveness of this method which combines iridencleisis and posterior sclerectomy in the surgical treatment of glaucoma secondary to uveitis in dog. However, pre-surgery IOP at 72 mmHg and the optic disc cupping found in the ophthalmologic examination are an unfavourable prognosis for the long-term retention of vision in the left eye.

## REFERENCES

- Abrams K.L. (2001): Medical and surgical management of the glaucoma patient. *Clinical Techniques in Small Animal Practice*, 16, 71–76.
- Barnett K.C. (ed.) (2006): *Diagnostic Atlas of Veterinary Ophthalmology*. Mosby Elsevier, London. 89–96.
- Barnett K.C., Sansom J., Heinrich C. (2002): Glaucoma. 99–107. In: Barnett K.C., Sansom J., Heinrich C. (eds.): *Canine Ophthalmoscopy an Atlas and Text*. W.B. Saunders, Toronto. 213 pp.
- Bedford P.G.C. (1985): A simple method of gonioscopy for the dog and cat. *Journal of Small Animal Practice*, 26, 407–410.
- Bedford P.G.C., Jones R.G. (2001): Abnormal appearance. 94–95. In: Peiffer Jr R.L., Petersen Jones S.M. (eds.): *Small Animal Ophthalmology: a Problem-Oriented Approach*. WB Saunders, London. 266 pp.
- Calkins L.L. (1960): The aqueous filtration angle: a phylogenetic and ontogenetic comparative histo-anatomic study of mammalian eyes. *Transactions of the American Ophthalmological Society*, 58, 364–391.
- Cook C.S. (1997): Surgery for glaucoma. *Veterinary Clinics of North America: Small Animal Practice*, 27, 1109–1129.
- Crispin S.M. (1988): Uveitis in the dog and cat. *Journal of Small Animal Practice*, 29, 429–447.
- Curtis R., Barnett K.C. (1980): Primary lens luxation in the dog. *Journal of Small Animal Practice*, 21, 657–668.
- Deehr A.J., Dubielzig R.R. (1998): A histopathological study of iridociliary cysts and glaucoma in Golden Retrievers. *Veterinary Ophthalmology*, 1, 153–158.
- Dubielzig R.R. (1990): Ocular neoplasia in small animals. *Veterinary Clinics of North America: Small Animal Practice*, 20, 837–848.
- Gelatt K.N., Brooks D.E. (1999): The canine glaucomas. 701–754. In: Gelatt K.N. (ed.): *Veterinary Ophthalmology*. Lippincott Williams & Wilkins, Philadelphia. 1544 pp.
- Gelatt K.N., Gelatt J.P. (2001): Surgical procedures for treatment of the glaucomas. 244–283. In: Gelatt K.N., Gelatt J.P. (ed.): *Small Animal Ophthalmologic Surgery*. Butterworth-Heinemann, Oxford. 381 pp.
- Gelatt K.N., MacKay E.O. (2004): Secondary glaucomas in the dog in North America. *Veterinary Ophthalmology*, 7, 245–259.
- Girkin C., McGwin G., Long C., Morris R., Kuhn F. (2005): Glaucoma after ocular contusion: a cohort study of the United States Eye Injury Registry. *Journal of Glaucoma*, 14, 470–473.
- Grozdanic S.D., Matic M., Betts D.M., Sakaguchi D.S., Kardon R.H., (2007): Recovery of canine retina and optic nerve function after acute elevation of intraocular pressure: implications for canine glaucoma treatment. *Veterinary Ophthalmology*, 10, 101–107.
- Gupta S.P., Garg K.C., Bisaria K.K. (1966): Different techniques of iridencleisis and their value in glaucoma – a study of 60 cases. *Indian Journal of Ophthalmology*, 14, 36–39.
- Johnsen D.A.J., Maggs D.J., Kass P.H. (2006): Evaluation of risk factors for development of secondary glaucoma in dogs: 156 cases (1999–2004). *Journal of American Veterinary Medicine Association*, 229, 1270–1274.
- Morris R.A., Dubielzig R.R. (2005): Light-microscopy evaluation of zonular fiber morphology in dogs with glaucoma: secondary to lens displacement. *Veterinary Ophthalmology*, 8, 81–84.
- Nagpaul P.N., Charan H., Sarda R.P. (1966): Cataract and iridencleisis-one stage operation. *Indian Journal of Ophthalmology*, 14, 31–35.

- Nelms S.R., Nasisse M.P., Davidson M.G., Kirschner S.E. (1993): Hyphema associated with retinal disease in dogs: 17 cases (1986–1991). *Journal of American Veterinary Medicine Association*, 202, 1289–1292.
- Nirankari M.S., Malhotra G.S. (1965): Evaluation of modified techniques of iridencleisis. *British Journal of Ophthalmology*, 49, 646–659.
- Ofri R., Narfstrom K. (2007): Light at the end of the tunnel? Advances in the understanding and treatment of glaucoma and inherited retinal degeneration. *Veterinary Journal*, 174, 10–22.
- Peiffer R.L. Jr, Wilcock B.P., Yin H. (1990): The pathogenesis and significance of preiridial fibrovascular membrane in domestic animals. *Veterinary Pathology*, 27, 41–45.
- Petersen Jones S.M. (1991): Abnormal ocular pigment deposition associated with glaucoma in the cairn terrier. *Journal of Small Animal Practice*, 32, 19–22.
- Reilly C.M., Morris R., Dubielzig R.R. (2005): Canine goniodysgenesis-related glaucoma: a morphologic review of 100 cases looking at inflammation and pigment dispersion. *Veterinary Ophthalmology*, 8, 253–258.
- Renwick P.W., Petersen Jones S.M. (2001): Orbital and ocular pain. 177–218. In: Peiffer Jr R.L., Petersen Jones S.M. (eds.): *Small Animal Ophthalmology: a Problem-Oriented Approach*. WB Saunders, London. 266 pp.
- Sansom J., Barnett K.C., Dunn K.A., Smith K.C., Dennis R. (1994): Ocular disease associated with hypertension in 16 cats. *Journal of Small Animal Practice*, 35, 604–611.
- Sapienza J.S., Simo F.J., Prades Sapienza A. (2000): Golden Retriever uveitis: 75 cases (1994–1999). *Veterinary Ophthalmology*, 3, 241–246.
- Smith R.I.E., Peiffer R.L., Wilcock B.P. (1993): Some aspects of the pathology of canine glaucoma. *Progress in Veterinary and Comparative Ophthalmology*, 3, 16–28.
- Spiess B.M., Bolliger J.O., Guscetti F., Haessig M., Lackner P.A., Ruehli M.B. (1998): Multiple ciliary body cysts and secondary glaucoma in the Great Dane: a report of nine cases. *Veterinary Ophthalmology*, 1, 41–45.
- Strubbe T. (2002): Uveitis and pupillary block glaucoma in an aphakic dog. *Veterinary Ophthalmology*, 5, 3–7.
- van de Sandt R.R., Boeve M.H., Stades F.C., Kik M.J.L. (2003): Abnormal ocular pigment deposition and glaucoma in the dog. *Veterinary Ophthalmology*, 6, 273–278.

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