**Sudden acquired retinal degeneration in the dog: report of seventeen cases and literature review**

F. GOULLE

Clinique Vétérinaire du Haut-Brion, 1 avenue Nancel Penard, 33600 Pessac, FRANCE.

*Corresponding author: f.goulle@aquivet.fr

**SUMMARY**

Seventeen cases of canine sudden acquired retinal degeneration syndrome (SARDS) diagnosed over a four years period are reported in this study. Seven of dogs of the series were Brittany Spaniels. Bilateral fundus examination by indirect ophthalmoscopy was normal or quasi-normal, while electroretinogram recordings were flat or barely detectable for both eyes in all cases. SARDS is an acute degenerative retinopathy which specifically affects the canine species. Its pathogenesis has not been clearly established yet. The diagnosis of SARDS relies on the association of rapid loss of vision and electroretinographic changes despite a normal ophthalmoscopic aspect of the fundus at the early stage of the disease. In the absence of an efficient treatment to date, the SARDS leads to irreversible blindness.

**Keywords:** Retinal degeneration, dog, blindness, electroretinography, Brittany spaniel.

**Introduction**

The canine sudden acquired retinal degeneration syndrome (SARDS) is well described in the literature, but its pathogenesis is poorly understood [4,9,12,15]. It is characterised by a fast and irreversible loss of vision which occurs over a very short period of time (in general a few days, sometimes a few weeks) [9,10,12,15]. A series of seventeen SARDS cases diagnosed by the author over a four years period between January 2005 and April 2009, is described.

**Case reports**

**INCLUSION CRITERIA**

Medical records of dogs referred during the period January 2005 to April 2009 with a primary problem of sudden blindness were reviewed. In every case, the owner reported that the dog had lost all sense of orientation and constantly bumped into objects, even among daily surroundings.

**ANCILLARY DIAGNOSIS**

The vision was first evaluated from a distance: an obstacle run was therefore systematically performed, in order to evaluate the visual behaviour of the animal. A close examination of each eye was then performed to evaluate the menace response. The pupil size was observed at rest and under light stimulus. Any detected mydriasis was either partial or total. Pupillary light reflexes were either present, decreased or absent. In all cases, the intraocular pressure was checked by tonometry (Tono-Pen™ XL, Medtronic). The thirty-four eyes were examined by slit-lamp biomicroscopy (Hawk Eye™, Dioptrix). Bilateral examination by indirect ophthalmoscopy (binocular indirect ophthalmoscope Omega 500™, Heine) with a planoconvex + 20-D lens (Heine) was performed, either without chemical dilation for the dogs having total unresponsive mydriasis, or after chemical dilation with topical tropicamide (Mydriaticum™ 0.5%, Thea, one drop every 5 minutes for 30 minutes) for the remaining dogs. A standardised electroretinography (Visiosystem™, Dioptrix) was performed under general anaesthesia in order to evaluate retinal function of each eye. If pupils had not been dilated already for indirect ophthalmoscopy, the eyes were then submitted to tropicamide dilation for the electroretinographic examination according to the same procedure. The anaesthesia was performed by intramuscular injection of 8 mg/kg of ketamine (Ketamine Virbac™, Virbac) and of 1 mg/kg of xylazine (Paxman™, Virbac). An achromatic stimulation in
photopic atmosphere was followed by a first instantaneous blue stimulation in scotopic atmosphere, and another one after eight minutes of dark adaptation. An ERG flicker was finally conducted by stationary achromatic stimulation (30 Hz).

Results

Seventeen dogs met the criteria for inclusion in this study. Each dog had a history of vision loss over a period ranging from several days to a few weeks (13 days on average). The average dog age in the investigated series was 8.9 years, ranging from 3.5 to 11.5 years. There were 13 spayed females (76.5%) and 4 males. Ten breeds of dogs were represented, including Brittany Spaniel (n = 7), English Setter (n = 2), Miniature Pinscher (n = 1), West Highland White Terrier (n = 1), Pekingese (n = 1), Shar Pei (n = 1), Poodle (n = 1), German Shepherd (n = 1), Short-haired Dachshund (n = 1) and mixed-breed dog (n = 1). Five dogs became noticeably overweight shortly before the blindness developed, and eight dogs had polyuria-polydipsia. All the animals lived in their owners’ house and had a garden run. They were fed with commercial dry food and were correctly vaccinated. Some of them were occasionally used for hunting.

The obstacle run revealed that every dog bumped into all obstacles. The menace response was absent in either eye in all seventeen cases. Bilateral mydriasis was detected in fourteen cases (total for eight dogs, partial for the other six). Direct and indirect pupillary light reflexes were absent in both eyes of seven dogs and decreased in the eyes of six other dogs. There was no visible symptom of ocular inflammation in any eye of the seventeen animals.

Tonometry examination showed that in all cases values obtained were consistently below the usual upper limit (<25 mm Hg) [5]. Examination of the thirty-four eyes by slit-lamp microscopy did not reveal any anomaly: lens and vitreous had normal aspect. Bilateral examination by indirect ophthalmoscopy revealed for fifteen dogs that optic discs were normal, as well as retinal vessels (with respect to aspect, amount and diameter) and that tapetal and non-tapetal zones had a normal aspect (absence of hyperreflectivity) (Fig 1). In the case of two dogs, a slight tapetal hyperreflectivity was detected, without modification of retinal vessel diameter. All electroretinograms obtained were either flat or with barely detectable morphology in both eyes.

All collected examinations and data enabled the diagnosis of sudden acquired retinal degeneration syndrome, also referred as silent retina syndrome. Its diagnosis relies on the association of rapid loss of vision, non recordable electroretinogram but normal ophthalmoscopic aspect of the fundus at the early stages of the disease.

Discussion

The sudden acquired retinal degeneration syndrome is characterised by a rapid and irreversible loss of vision over a short period of time (in general few days, sometimes few weeks) [4,12]. This retinopathy can affect a number of breeds including mixed breeds with a possible predisposition of the Dachshund, the Miniature Schnauzer and the Brittany Spaniel [3,10,15,16]. In the present study, whereas only one Dachshund is afflicted with SARDS (5.9%), as many as seven Brittany Spaniels are reported (41.2%). The other breeds covered in the study do not reveal any predominance.

The affected dogs are usually middle-aged to old (average age of appearance of the disease is 8.5 to 10 years old), either male or female (although females seem to be predisposed) [3,4,6,9,12,15]. The results of this study (average age 8.9 years with 76.5% of females) are in line with published data. The affected dogs are usually in good health and often experience polyuria, polydipsia, polyphagia and a significant gain in weight [4,6,9]. The same features are observed in this study: five dogs became noticeably overweight shortly before their blindness developed and polyuria-polydipsia was detected in seven cases.

Clinically, the dogs experience a sudden blindness and are therefore frequently disoriented. Some cases of transient night blindness have been reported in the literature, however not in this study [3,15,16]. Bilateral mydriasis either reactive or not reactive is often detected [3,10]. Generally, there is no sign of ocular inflammation associated with SARDS [10]. In our study, bilateral mydriasis was present in fourteen cases (82.4%); pupillary light reflexes were absent in both eyes of seven dogs and decreased in the eyes of six other dogs. There was no visible symptom of ocular inflammation in any eye of the seventeen animals. A report nevertheless refers to the presence of conjunctival hyperemia [16].

In the acute phase of the appearance of the disease, examination of the fundus by indirect ophthalmoscopy does not reveal any anomaly in most cases (normal optical disc, normal retinal vessels, absence of retinal hyperreflectivity) [4,9,12,15]. In some cases however, a slight tapetal hyperreflectivity can be observed a few days after blindness has been declared [3,9]. In the three to six following weeks, the tapetal hyperreflectivity is known to be more pronounced and coupled with a decrease of the vessel diameter [3]. In the pre-
sent study though, only two dogs experienced a slight tapetal hyperreflectivity, without modification of retinal vessel diameter. A possible rationale is that most of the dogs were referred rather early, less than three weeks after blindness had appeared (13 days in average). After a few months, ophthalmoscopic examination of the fundus shows signs of retinal degeneration [4,15]. In these advanced stages, the fundus of dogs affected by SARDS may present either only slight tapetal hyperreflectivity with modification of the retinal arteriolar diameter or an appearance similar to that of dogs suffering from a genetically transmitted progressive retinal atrophy (pronounced tapetal hyperreflectivity, significant diminution of retinal vessels diameter, optical disc atrophy) [3,4,9,12]. In some cases, discrimination between these two retinal degenerations is not possible solely by fundus examination [4,9,12]. In this study, the dogs were not further examined after diagnosis was established. No data could be collected on the long term aspect of the fundus.

Electrophysiological examination of retinal function usually reveals in SARDS cases either a flat or barely detectable electroretinogram: the electrical activity of cones and rods is strongly decreased in all cases, despite normal ophthalmoscopic appearance of the fundus in the early phase of the disease [4,6,12,14]. These findings are in line with the electroretinograms recorded in the present study: ten flat electroretinograms (Fig 2) and seven electroretinograms with barely detectable morphology (Fig 3).

The origin of SARDS is unknown [4,13,15]. Although several possible causes have been investigated, no specific etiology has yet been proven. An association between hyperadrenocorticism and the appearance of SARDS has been suggested, but the link between both conditions is not clearly established [4,9,12,14,15]. It has been suggested that substances having glucocorticoid-like activity might be secreted by the adrenal or pituitary gland of dogs affected with SARDS, thereby explaining clinical symptoms and biochemical profiles similar to those of Cushing’s disease [14].

A further study investigated the role of apoptosis in SARDS [10]. This form of cell death may occur as a result of exposure to multiple stimuli, such as for example steroid

**Figure 2:** Bilateral electroretinogram of the 8-year-old male Brittany Spaniel.
hormones, cytokines or toxins. Apoptosis could be one mechanism through which photoreceptors die. It is nevertheless also possible that cell death is initiated by other mechanisms leading to retinal degeneration at a later stage [10]. Photoreceptors (rods and cones) degenerate and lose their outer segments. As the disease progresses, the other cell layers degenerate (amacrine, bipolar and ganglion cells) eventually leading to complete retinal degeneration [13,15].

The triggering mechanism of apoptosis is unknown; it might be caused by an excitotoxin. Glutamate is sometimes incremented in apoptosis of the internal retinal layer, but in the case of SARDS where apoptosis involves the external retinal layer, the implication of glutamate is probably not relevant [10].

An alternative hypothesis would be an autoimmune mechanism suggested by the presence of circulating antiretinal antibodies in the serum of dogs affected by SARDS [1,6,8,12]. The role of these antibodies is not clearly defined: rather than being responsible for photoreceptor death, they may result from a sudden and massive exposure of the immune system to antigens from degenerated photoreceptors [10].

A recent study of SARDS affected dogs shows the presence in their blood of self-antibodies targeting the recently discovered retinal self-antigen NSE (neuron-specific enolase) [2].

**Conclusion**

Former studies reported a possible predisposition of Dachshund, Miniature Schnauzer and Brittany Spaniel breeds for SARDS [3,10,15], including a predominance of Brittany Spaniel (19%) in a recent report [16]. The present study reveals a significant occurrence of Brittany Spaniel breed (41.2%) affected by SARDS over the other breeds, which confirms the hypothesis of a predisposition of that breed for this acute retinopathy. To date, the origin of SARDS is not elucidated and there is no treatment for this retinal degeneration which irreversibly leads to rapid blindness. Alternatively, the recent use of intravenous immunoglobulins in the treatment of SARDS affected dogs enables an improvement of the coarse vision of some dogs, without nevertheless restoring the menace response [7].

**FIGURE 3**: Bilateral electroretinogram of the 11.5-year-old female Pekingese.
References


