



NOTE

Pathology

Synchysis scintillans of the anterior chamber in a dog

Yuto SANO^{1)*}, Minoru OKAMOTO¹⁾, Miri HAYASHI²⁾, Takashi SATO²⁾,
Seiya MAEHARA²⁾ and Kazuya MATSUDA¹⁾¹⁾Department of Veterinary Pathology, School of Veterinary Medicine, Rakuno Gakuen University, Ebetsu, Hokkaido 069-8501, Japan²⁾Department of Small Animal Clinical Sciences, School of Veterinary Medicine, Rakuno Gakuen University, Ebetsu, Hokkaido 069-8501, Japan*J. Vet. Med. Sci.*

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ABSTRACT. Intraocular cholesterol granuloma (CG) associated with synchysis scintillans (SS) was diagnosed in a 5-year-old spayed Shetland sheepdog. During the initial clinical examination, the patient exhibited SS in the anterior chamber. Canine SS is usually found in the vitreous cavity, and SS in the anterior chamber has not been described. Since canine SS has been reported to be a non-progressive condition, and its long-term clinical course has not been adequately documented. The present case report describes the long-term clinical course of a case of canine SS, in which SS occurred in the anterior chamber, leading to intraocular CG formation, and eventually glaucoma.

KEY WORDS: anterior chamber, dog, intraocular cholesterol granuloma, synchysis scintillans

In human ophthalmology, synchysis scintillans (SS) is known as *cholesterolosis bulbi* [17, 20]. This condition refers to a state in which floating cholesterol particles form within the vitreous cavity. Clinically, the particles usually settle in the inferior region when eyes/head are kept still, but ocular movement results in a glittering shower of particles spreading throughout the vitreous cavity. The cholesterol particles usually form within the vitreous cavity, but cases in which cholesterol particles have formed in the anterior chamber have occasionally been reported [1, 2, 6, 9, 10, 12, 13, 16, 18–20]. SS can arise in the anterior chamber as a result of long-term SS.

In veterinary ophthalmology, SS is an uncommon condition, but canine SS has been reported to be similar to human SS [5, 11, 14]. In dogs, SS is recognized as an acquired degenerative condition and has been reported to be related to retinal atrophy [3, 4]. Since canine SS was reported to be as a non-progressive condition [7], its long-term clinical course has not been adequately documented, such as SS in anterior chamber. In this report, we describe the long-term clinical course of a case of canine SS of the anterior chamber, in which the SS led to intraocular cholesterol granuloma followed by secondary glaucoma.

A 5-year-old spayed female Shetland sheepdog visited the Department of Ophthalmology of Rakuno Gakuen University Animal Medical Center with a month-long history of visual impairment. The dog did not have a relevant medical history, and no abnormalities were detected during physical examinations, including of complete blood cell counts, blood biochemical examinations, and blood pressure. Ophthalmic examinations were performed, and neuro-ophthalmic examination revealed the absence of the menace response and dazzle reflex in both eyes (OU), and the presence of the weak direct and indirect pupillary light reflexes in OU. Normal intraocular pressure (IOP) values were detected in OU by applanation tonometer (Tono-Pen XL applanation tonometer; Medtronic Solan, Jacksonville, FL, U.S.A.), and slit-lamp microscopy (SL-D7; Topcon, Tokyo, Japan) detected findings that were suggestive of conjunctival hyperemia and severe flaring of the anterior chamber in OU. Retinal detachment and vitreous hemorrhaging were detected in the right eye (OD) during funduscopy (TRC-50IX; Topcon). It was not possible to examine the left eye (OS), as it was obscured by severe vitreous hemorrhaging. An ocular ultrasound examination confirmed that there were no ocular abnormalities other than retinal detachment of the OU. Based on the patient's clinical findings, a diagnosis of pan-uveitis associated with complete retinal detachment and vitreous hemorrhaging of the OU was made. The exact cause of the bilateral pan-uveitis remains unclear, but non-specific causes or conditions, such as a trauma or idiopathy, were suspected as a cause of the uveitis in accordance with the results of the clinical examinations. The patient's owner chose maintenance drug therapy, and so the OU was treated with 15 mg/kg intravenous (IV) methylprednisolone sodium succinate (Pridol for injection; Sankyo, Tokyo, Japan) quaque (q) 24 hr for 2 days and one drop of betametasone (Rinderon; Shionogi, Osaka, Japan) q6hr. Then, the OU was treated with 1.0 mg/kg per oral (PO) prednisolone (Predonine; Shionogi) q24hr for 7

*Correspondence to: Sano, Y.: y-sano@rakuno.ac.jp

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days, followed by 0.5 mg/kg PO prednisolone q24hr for a further 7 days and then one drop of betametasone q6hr. Both eyes were re-evaluated on day 13, and it was demonstrated that the uveitis and anterior chamber flaring in the OU had been ameliorated, but retinal detachment was still noted. Retinal detachment continued to be seen until the 98th day, and but no physical abnormalities or recurrent uveitis were observed.

On day 203, the patient was brought to our clinic again, as she had been suffering from blurriness in the OD for a few days. Slit-lamp microscopy suggested that the patient was suffering from severe blepharospasm and conjunctival hyperemia, and floating shiny particles with severe flaring were seen in the anterior chamber (Fig. 1) and vitreous cavity. These particles usually fell to the bottom of the eye when the eyes/head were kept still, but eye and head movements tended to cause the particles to float throughout, which has been known as typical floating patterns of intraocular particles in SS [5, 20]. An ultrasound examination revealed the presence of highly echoic materials in the anterior chamber and vitreous cavity (Fig. 2), and no rupturing of the lens capsule or lens displacement was observed. The axial length of OD which measured by the ultrasound was 19.1 mm. According to these clinical findings, the patient was diagnosed with synchysis scintillans of the anterior chamber and vitreous cavity combined with uveitis, all of which affected the OD. The other physical examinations obtained normal results. The patient was treated by 1.0 mg/kg PO prednisolone q24hr for a further 7 days, followed by one drop of betametasone q6hr.

Subsequently, an increase in the volume of intraocular particles was seen in the anterior chamber on day 215 (Fig. 3), and slit-lamp microscopy revealed semi-lustrous tissue, which had formed a bridge across the pupil and covered most of the anterior surface of the iris, on day 265 (Fig. 4). The IOP of the OD was 21 mmHg, and an ocular ultrasound examination demonstrated the presence of highly echoic non-floating tissue in the anterior chamber and on the anterior iridal surface of the OD. The axial length of the OD was 21.6 mm, and it revealed that the OD enlarged 2.5 mm in the axial length of eyeball comparing to the day 203. The patient was diagnosed with secondary glaucoma of the OD, associated with semi-lustrous tissue formation in the anterior chamber and anterior uveitis. For therapeutic and diagnostic purposes, the intraocular tissue was removed, and a silicone intrascleral prosthesis was implanted on day 275. The removed intraocular tissue was submitted for histopathological examination. On day 344, the OD did not present with any postoperative abnormalities, and no similar condition was observed in the OS.

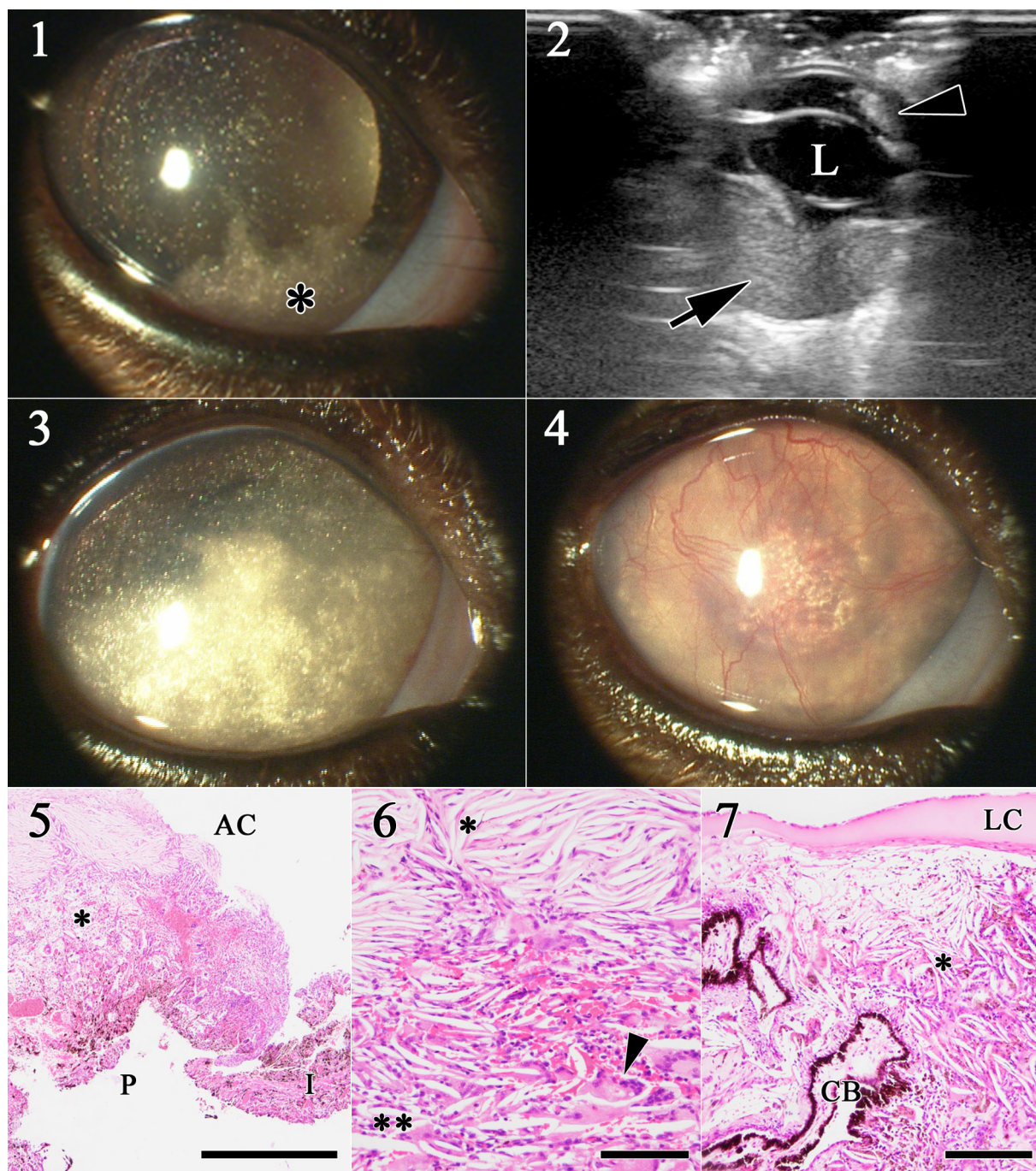
The tissue was fixed in 10% neutral buffered formalin, processed routinely, and embedded in paraffin wax. Sections (4 μ m) were stained with hematoxylin and eosin (HE), Berlin blue (BB), gram and periodic-acid Schiff (PAS). Histologically, the semi-lustrous tissue, which had formed a bridge across the pupil, was diagnosed as a CG (Fig. 5). It was composed of closely packed slit-like spaces, which were indicative of cholesterol clefts, and exhibited granulomatous inflammation. The cholesterol clefts were surrounded by macrophages, foreign body multinucleated giant cells, and small numbers of lymphocytes and plasma cells (Fig. 6). The CG had also covered the anterior surface of the iris, and it had partially adhered to Descemet's membrane. A pre-iridal fibrovascular membrane was observed in the residual iridal tissue. In the posterior ocular tissue, a CG had formed between the ciliary body and lens capsule, and it had adhered to both tissues (Fig. 7). Scattered and follicular lymphocyte-associated inflammation were observed in the residual iris and anterior choroid. BB-positive deposits (hemosiderin) and hemosiderin-laden macrophages were scattered throughout the CG and hemorrhagic intraocular tissue. No gram and PAS react pathogenic organisms were detected. According to these histological findings, a diagnosis of intraocular CG combined with intraocular hemorrhaging and chronic anterior uveitis was made.

In human ophthalmology, SS in the anterior chamber is a rare ocular condition, but it has been reported sporadically [1, 2, 6, 9, 10, 12, 13, 16, 18–20]. It usually occurs with prolonged visual impairment and is related to antecedent ocular insults or diseases. Among such antecedent ocular insults, the most common is a trauma that was severe enough to result in a traumatic cataract, lens subluxation, retinal detachment, or vitreous hemorrhaging [20]. SS has also been reported to occur following ocular conditions, including hypermature cataracts, chronic uveitis, long-standing retinal detachment, and vascular disorders without any systemic metabolic abnormalities [1, 10, 20]. In veterinary ophthalmology, SS is described as an uncommon ocular condition [11], and it has only been reported in dogs in association with retinal atrophy [3, 4]; however, no cases of SS in anterior chamber have been documented. The present case, in which the patient suffered from long-term visual impairment accompanied by retinal detachment, is the first to be reported in veterinary ophthalmology.

Different processes have been proposed to explain the appearance of cholesterol particles in human SS of the anterior chamber. One of these processes involves intravitreal cholesterol particles directly entering the anterior chamber from the vitreous cavity via the pupillary space. In addition, the particles pass through the atrophic iris and degenerated suspensory ligament, and this condition is accompanied by lens abnormalities, such as aphakia and lens (sub) luxation [2, 9, 13, 19]. Another possible process is that the proteins and fats released from the posterior segment of the eye enter the anterior chamber with the flow of intraocular fluid, and cholesterol particles first form in the anterior chamber [9, 16, 18, 20]. In the present case, the ocular conditions experienced by the patient were not accompanied by lens anomalies; thus, particles in the anterior chamber seemed to have formed via the latter process rather than the former process.

The floating cholesterol particles have been found to be associated with cholesterol deposition in intraocular tissues in human SS [9, 20]. Although the exact nature of floating particles could not investigate in the present case, the histopathological findings suggested that those were composed of cholesterol, and that the cholesterol particles that had continuously accumulated in the dog's intraocular tissues had acted as foreign bodies [15]. Finally, the accumulation brought about the formation of granulomatous lesions. The histopathological findings of the current case were considered to be indicative of long-term canine SS, and the obstacle to the drainage of the aqueous humor effluent by the intraocular CG formations were concluded to be a cause of the glaucoma.

In human SS, cholesterol particles are considered to form after the release of cholesterol molecules due to the lysis of intraocular red blood cells [9, 13, 20]. However, SS has also been reported to occur in patients with retinal detachment or hypermature cataract



- Fig. 1.** Gross photographs of the right eye obtained on day 203. Minute glittering particles are present in the anterior chamber. They usually fall to the bottom of the eye (*), but eye and head movements result in the particles being distributed throughout the anterior chamber and vitreous cavity.
- Fig. 2.** Ultrasound image of the right eye obtained on day 203. Highly echogenic materials are present in the anterior chamber (arrowhead) and vitreous cavity (arrow). The lens (L) is normally positioned, and the lens subluxation or luxation is not observed.
- Fig. 3.** Gross photographs of the right eye obtained on day 215. The volume of the particles in the anterior chamber has markedly increased.
- Fig. 4.** Gross photographs of the right eye obtained on day 265. Semi-lustrous tissue is seen in the pupil. It has also covered the iridal surface.
- Fig. 5.** Histological photograph of the semi-lustrous tissue (*) which had formed a bridge across the pupil of the right eye. The tissue is formed on the pupil (P) with attachment of the pupillary margin of the iris (I). AC: anterior chamber. HE. Bar=500 μ m.
- Fig. 6.** A higher magnification of the image of the semi-lustrous tissue shown in Fig. 5. The tissue is composed of aggregates of typical cholesterol clefts (*) and infiltrating inflammatory cells (**). Macrophages, foreign body multinucleated giant cells (arrowhead), and small numbers of lymphocytes and plasma cells are infiltrated in the regions around the cholesterol clefts. Red blood cells are also observed. HE. Bar=100 μ m.
- Fig. 7.** Histological photograph of the posterior ocular tissue. Cholesterol granuloma (*) forms between the ciliary body (CB) and lens capsule (LC). HE. Bar=100 μ m.

alone [6, 13, 16]. The former cases suggested the leakage of cholesterol-rich subretinal fluid without hemorrhaging resulting in the formation of intraocular cholesterol particles [13, 16], and the latter case indicated that such particles can originate from exudative lens material [6]. In the present case, the finding of the ophthalmic examinations suggested that a protein-rich exudate that had been released from the subretinal space had participated in the formation of the cholesterol particles. In addition, the extracted intraocular tissue revealed the presence of old hemorrhages, which were indicative of the lysis of red blood cells, and it was also revealed that new blood had been supplied. It was assumed that large proportion of the cholesterol particles had formed due to the presence of abundant raw materials.

In veterinary ophthalmology, since canine SS has been reported to be a non-progressive ocular disease, treatment is generally considered to be unnecessary [7]. Although SS has been reported to be a cause of intraocular CG [8, 11], the mechanism responsible for this has not been adequately documented. The clinical and histological findings of the present case showed that canine SS brought about SS in the anterior chamber, which led to the formation of an intraocular CG over the long term. Therefore, canine SS requires further clinical attention, as it may have the potential to exhibit a progressive clinical course.

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