

Case Report—

## Fatal Verminous Pharyngitis and Esophagitis Caused by *Streptocara incognita* in Mute Swans (*Cygnus olor*)

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**SUMMARY.** *Streptocara* spp. infections are reported to cause gastritis, proventriculitis, esophagitis, and pharyngitis in various waterfowls, especially diving ducks. In the present paper, we describe severe fatal diphtheritic pharyngitis and esophagitis caused by *Streptocara incognita* in three female mute swans (*Cygnus olor*) in Bosnia and Herzegovina. Prior to death, the swans were showing signs of lethargy, anorexia, and reluctance to move. At necropsy, in all swans severe diphtheritic pharyngitis and esophagitis with deep, dark red hemorrhagic ulcerations were observed. Numerous thin, white, up to 1-cm-long nematodes, identified as *S. incognita*, were observed embedded in the pharyngeal and esophageal mucosa under the diphtheritic membranes. Histopathology revealed severe fibrinonecrotic inflammation with numerous cross-sections of the parasites. To the authors' knowledge, this is the first report of severe, fatal streptocariasis in mute swans.

**RESUMEN.** *Reporte de Caso*—Faringitis y esofagitis verminosas fatales causadas por *Streptocara incognita* en cisnes vulgares (*Cygnus olor*).

Se ha reportado que las infecciones por *Streptocara* spp. son causantes de gastritis, proventriculitis, esofagitis y faringitis en aves acuáticas, sobre todo en diversos patos buceadores. En el presente trabajo se describe un caso severo y fatal de faringitis y esofagitis diftéricas causado por *Streptocara incognita* en tres cisnes vulgares (*Cygnus olor*) hembras en Bosnia y Herzegovina. Antes de la muerte, los cisnes mostraron signos de letargia, anorexia y renuencia a moverse. Durante la necropsia, se observó en todos los cisnes faringitis y esofagitis diftéricas severas y con profundas ulceraciones de color rojo oscuro y hemorrágicas. Numerosos nemátodos delgados de color blanco y de hasta un centímetro de largo, identificados como *S. incognita*, se observaron incrustados en la mucosa de la faringe y del esófago por debajo de las membranas diftéricas. La histopatología reveló inflamación fibrinonecrotica severa con numerosas secciones transversales de parásitos. De acuerdo al conocimiento de los autores, este es el primer reporte de streptocariasis grave y mortal en cisnes vulgares.

**Key words:** *Cygnus olor*, esophagitis, fibrinonecrotic, mute swan, pharyngitis, *Streptocara incognita*

Streptocariasis is a parasitic disease caused by several species of *Streptocara* genus, a cosmopolitan nematode that mostly inhabits the gizzard, the proventriculus, and the mucosa of the upper digestive tract of various wild waterfowls worldwide (5,13,14,17). *Streptocara crassicauda* Creplin, 1829, and *Streptocara incognita* Gibson, 1968, are the two most commonly involved species (1,13). *Streptocara* spp. is a well-known cause of severe, fatal pharyngitis, esophagitis, and ventriculitis, particularly in diving ducks. Cases of streptocariasis are recorded in variety of wild ducks in Australia (12), Canada (5,11,14), the United States (16), the United Kingdom (2,3), and Italy (1). In the United States, ulcerative gastritis and proventriculitis caused by *S. incognita* has been reported in two adult Chilean flamingos (*Phoenicopterus chilensis*) (4). Severe esophagitis caused by *S. incognita* (formerly *pectinifera*) has been reported in domestic Japanese geese (*Sygnopsis sygnoides* L.) in former Yugoslav republics (present Croatia) (5,8). However, infections of domestic birds are rare because the life cycle of the parasite requires an intermediate host, an amphipod crustacean (*Gammarus lacustris*, *Gammarus locusta*, *Gammarus maeoticus*, *Gammarus triacanthus*, and *Hyalella azteca*) from water sources that are usually preyed by the waterfowl (10). Parasites' larvae develop in the hemocoel of the intermediate

host, and reach their infectivity in 19–25 days. In the definitive host, parasites develop rapidly and females start to lay eggs in only 1–2 wk (13).

Swans are recognized as a host species for *S. crassicauda*, which is recorded in low prevalence and low intensity in the gizzard of tundra swans (*Cygnus columbianus*), whooper swans (*Cygnus cygnus*) (13,17), and trumpeter swans (*Cygnus buccinator*) (13). However, no lesions caused by *Streptocara* spp. in swans have been reported previously.

In the present paper, we describe an outbreak of severe, fatal fibrinonecrotic pharyngitis and esophagitis caused by *S. incognita* in three mute swans (*Cygnus olor*) in Bosnia and Herzegovina.

### CASE HISTORY

Three 1- to 2-yr-old dead female mute swans (*C. olor*) were submitted for necropsy to the Department of Pathology at the Veterinary Faculty of Sarajevo. These birds were from a pond in the Bosnia River protected area located in the vicinity of Sarajevo, Bosnia-Herzegovina. The dead swans had been housed within a fenced pond together with six other swans and mallard ducks (*Anas platyrhynchos*). Animals were not vaccinated and no drugs were administered. Clinically, animals were showing signs of anorexia,

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Fig. 1. Mucosa of the pharynx and the beginning of the esophagus of the mute swan 1 covered with multifocal to coalescing, thick, granular, tan to brown plaque-like fibrinonecrotic masses (indicated by \*).

lethargy, inappetence, and reluctance to move. Remaining swans (six birds) were translocated to another pond due to a lack of water. No other death cases were recorded in swans or ducks.

**Necropsy.** At necropsy, carcasses (swans nos. 1–3) were in poor body condition, and pectoral muscle atrophy was evident. Multifocal to coalescing, up to 1-cm-thick, granular, tan to brown, firmly attached fibrinonecrotic masses were present on the mucosa of the pharynx, hard palate, caudal to choanal opening, laryngeal opening, and upper third of the esophagus (Fig. 1). Multiple small foci of similar appearance were also observed on the mucosa of the laryngeal cavity. In the lower esophagus, lesions were smaller, mostly longitudinal, parallel, and multifocal in distribution. Upon removal of the necrotic mass, deep, dark red hemorrhagic ulcerations of the mucosa were observed. In swan no. 3, focally extensive, thin, fibrinonecrotic membranes were observed on the mucosa of the proventriculus and joining esophagus, but ulcerations were not present. In the pharynx, obstructing laryngeal and esophageal openings and extending down the esophagus, there were stratified masses of impacted water grass and algae mixed with food particles and sloughed fibrinonecrotic membranes. Intestines were devoid of content in all carcasses. Livers were mottled, dark red, and slightly enlarged. In two animals (swans no. 1 and no. 2), multifocal petechial to ecchymotic hemorrhages were observed on the epicardium.

**Histopathologic examination.** For histopathology, samples of pharynx, esophagus, larynx, liver, intestine, pancreas, kidneys, and heart were fixed in 10% buffered formalin, routinely processed, embedded in paraffin, sectioned at 5  $\mu$ m and stained with hematoxylin and eosin.

Histologic examination revealed severe multifocal to coalescing diphtheritic pharyngitis and esophagitis. Multifocal to coalescing deep ulcerations covered with thick membranes composed of fibrin, necrotic tissue, and degenerated inflammatory cells, mostly heterophils, were observed in the mucosa of the pharynx and esophagus (Fig. 2). Numerous cross-sections of adult parasites were present under the fibrinonecrotic membranes, embedded into the necrotic tissue and surrounded with numerous heterophils, lymphocytes, plasma cells, and macrophages. Parasites were round to oval, 90 to 200  $\mu$ m wide, with a thin cuticle (external ridges visible at longitudinal sections), coelomyarian musculature, and prominent lateral cords (Fig. 2, insert). In the coelom, the intestine and uterus were filled with numerous oval-to-ellipsoid, thick-walled embryonated eggs. Throughout the fibrinonecrotic membrane, there

were basophilic bacterial colonies (Fig. 2). The blood vessels of the mucosa were hyperemic and there were perivascular infiltrates of numerous heterophils. In the larynx, there were multiple small foci of mucosal ulceration covered with moderate fibrinonecrotic membranes, but no parasites were observed. In the liver, multifocal mild to moderate foci of lytic necrosis infiltrated with moderate numbers of mostly degenerate heterophils and lesser numbers of lymphocytes and macrophages were observed throughout the parenchyma. In a few foci, there was accumulation of homogenous eosinophilic material (fibrinous exudate). Hepatic sinusoids were hyperemic and multifocally occluded with homogenous eosinophilic material (microthrombi). In the heart, multiple small foci of myocardial necrosis infiltrated with small numbers of heterophils and rare macrophages and lymphocytes were observed. The blood vessels were hyperemic, and small numbers of heterophils and lymphocytes were present in the lumen and in the perivascular space. The heart interstitium was edematous. In the kidneys there were multifocal to coalescing foci of the vacuolar degeneration and necrosis of the epithelial cells of the proximal and the distal tubules. Occasionally the tubules were distended with accumulated compact eosinophilic material (protein). Throughout the kidney parenchyma, there were aggregates of globular basophilic casts. In the glomeruli, the mesangium was slightly proliferated. No lesions were observed in the pancreas and in the intestine.

**Parasitology.** Membranous lesions from pharynx and esophagus of all three animals (swans 1–3) and of proventricular membranes from swan 3 were scraped, placed in 5% potassium hydroxide, and scanned for parasites. Recovered parasites were preserved in 70% ethyl alcohol, cleared, and observed in lactophenol. Based on the data from previous reports (4,5,13,16), specimens were identified as *Streptocara* spp. Later, according to Gibson (5), specimens were identified to a species level.

Numerous thin, white, up to 1-cm-long parasites of both sexes were identified as *S. incognita* (Nematoda: Acuariidae) from the samples of pharynx and esophagus of all three animals (swans 1–3). No parasites were recovered from the proventricular sample (swan 3). In both sexes, the collarette was slightly dorsoventrally indented. They had a short, poorly striated vestibule. The cervical papillae were sharp pointed, deeply divided longitudinally, and located along the full vestibular length down the vestibule (Fig. 3a). The cuticle above the papilla was flat without annuli. In males, at the rear end there were two spicules, four pairs of, and six pairs of postanal papillae



Fig. 2. Tissue section of the esophagus of the mute swan 1, with severe necrosis and inflammation of the mucosa covered with thick fibrinonecrotic membrane (\*). Cross-section of the parasite (arrow) and bacterial colonies (arrowheads) are visible in the fibrinonecrotic mass. Insert shows the higher magnification of the parasite surrounded by inflammatory cells. H&E. Bar = 200 μm.

(Fig. 3b). In females, the uterus was filled with numerous oval-to-ellipsoid, thick-walled, embryonated eggs, 19 to 30 μm wide and 35 to 40 μm long.

**Bacteriology.** Samples of pharyngeal mucosa (swans 1 and 3) and liver from swan 1 were subjected to bacteriology. The samples were inoculated on McConkey agar, Chapman agar, and blood agar

supplemented with 10% defibrinated sheep blood; all were incubated at 37 C for 24 hr. Isolated bacteria were determined according to *Bergey's Manual of Determinative Bacteriology* (7).

Hemolytic *Escherichia coli*, *Aeromonas hydrophyla*, and *Staphylococcus* spp. were isolated from samples of pharyngeal tissue from swans 1 and 3, and from the liver of the swan 1.

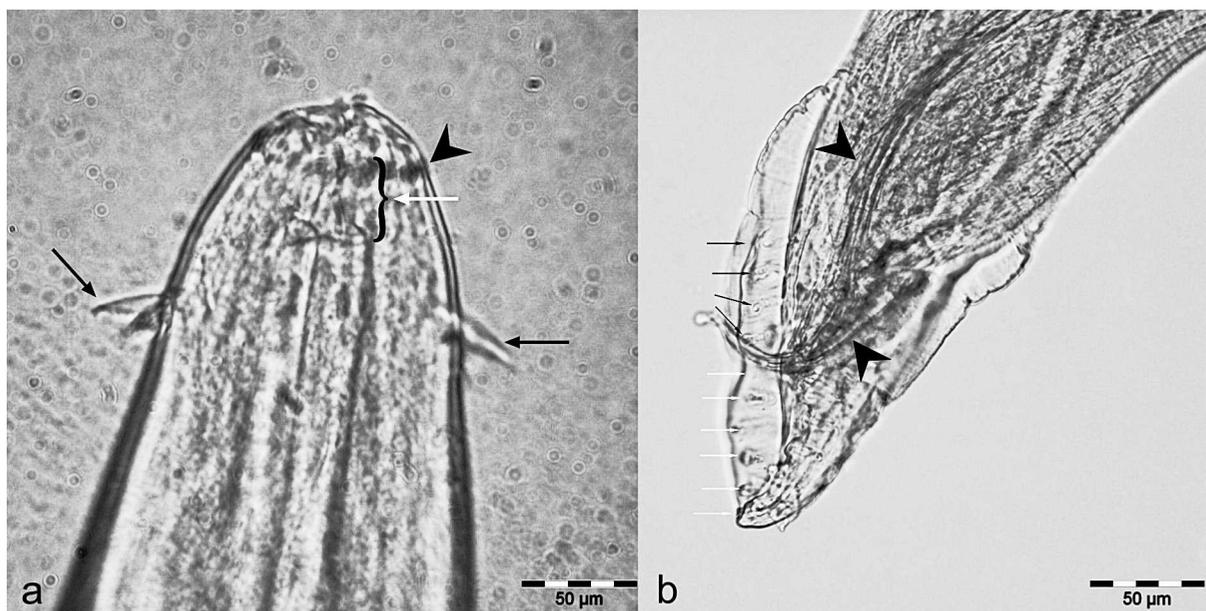


Fig. 3. *Streptocara incognita* observed in lactophenol. (a) Head of a female *S. incognita*. The collarette (arrowhead), poorly striated vestibule (white arrow), and sharp pointed cervical papillae (black arrows). Bar = 50 μm. (b) Rear end of a male *S. incognita*. Two spicules (arrowheads), four pairs of preanal papillae (black arrows), and six pairs of postanal papillae (white arrows). Bar = 50 μm.

## DISCUSSION

*Streptocara* spp. are thin cosmopolitan nematodes parasitizing the mucosa of the esophagus, crop, proventriculus, and gizzard, and are capable of causing severe disease and death in a variety of waterfowl (12).

In the present paper, severe diphtheritic pharyngitis and esophagitis in swans associated with *S. incognita* was described. Swans are recognized as a host species for *Streptocara* spp. (13,17); however, in these reports *Streptocara* spp. have been recovered from the gizzard only and no significant lesions were observed. To the best of our knowledge, this is the first report of severe, fatal streptocariasis in mute swans.

*Streptocara incognita*, one of the two most commonly implicated *Streptocara* species, has been previously described in Croatia (8), England (2), the United States (4), and, recently, Italy (1). Unlike *S. crassicauda*, *S. incognita* was recovered from the esophagus and proventriculus, in addition to the gizzard (1,8). Based on the experimental investigations in three species of ducks, *S. incognita*, rather than *S. crassicauda*, is more likely to be responsible for lesions occurring outside the gizzard (11). Since all parasites recovered from the lesions in swans reported here were identified as *S. incognita*, this appear to be pure infection that further supports the pathogenic influence of this species.

The remarkable severity of the lesions in this report is similar to those observed in previous investigations (1,2,4,5,8,12,16). In swans reported here, lesions were mostly located in the pharynx and the beginning of the esophagus, and were similar to lesions reported by Boughton (2) and Mason (12) in ducks. The presence of thick necrotic, diphtheritic, plaque-like masses on the pharyngeal mucosa, and impacted masses of food and necrotic material was completely obstructing the laryngeal opening leading to death by asphyxiation. As opposed to previous investigations (4,5,16), no lesions (apart from thin fibrinonecrotic membranes in the proventriculus in swan 3), or *Streptocara* spp. were observed in the proventriculus and gizzard in the present report. Lesions at the laryngeal opening and on the mucosa of the laryngeal cavity were probably the result of bacterial, rather than parasitic, infection because no parasites were recovered from these areas. In addition, severe inflammatory response in the surrounding tissue might have contributed to the development of the lesions in these locations.

The amphipods *Gammarus* spp. and *H. azteca* are common intermediate hosts of *Streptocara* spp. (2,10). *Hyalella azteca* is very important in permanent shallow waters where *Gammarus* spp. are absent or in lower numbers due to warm water (10). Various ducks are commonly infected with *S. incognita*, which is found in the gizzard and proventriculus, but also in the upper digestive tract (1,5,12,16). Infected ducks release the eggs that are eaten by amphipod crustaceans as a part of their life cycle. Spatial overlap and use of areas contaminated by species harboring *Streptocara* spp. makes other birds vulnerable to the infection (14). Furthermore, even though duck species may play negligible role on the wider scale, heavily infected individuals could significantly contribute to a local infective pool (14). In cases reported here, the source of infection could have been eggs of parasites released by mallard ducks held together with the swans in an enclosure with a shallow water pond. Thus, in shallow water, amphipod crustaceans bearing parasitic larvae could have been more readily available; however, their presence was not determined. In addition, in our case no death cases in mallards were reported.

On differential diagnosis, other parasites such as *Echinuria* (6), *Capillaria*, and *Tetrameres* spp. should be considered. Furthermore, bacterial and mycotic pharyngitis and esophagitis, and corrosive chemicals, should be considered as a differential diagnosis (1).

Bacteriologic examination of the lesions caused by *S. crassicauda* in ducks in Tasmania revealed the presence of *Moraxella* sp., *Aerococcus* sp.,  $\alpha$ -hemolytic streptococci, *E. coli*, *Pasteurella haemolytica* var. *ureae*, and *Alcaligenes* sp. (12). In the present report *E. coli*, *Aeromonas hydrophyla*, and *Staphylococcus* spp. were isolated from pharyngeal tissue and from the liver. These bacteria could cause pharyngitis, esophagitis, and hepatitis. Lesions observed in the liver (heterophilic infiltrates and microthrombi) and the heart (myocardial necrosis) are consistent with systemic bacterial infections (15). Although nonspecific, observed lesions were most likely caused by isolated bacteria, in particular *E. coli* that have reached the circulation in the pharynx and lodged in the liver and the heart. Most probably, these were secondary infections that further contributed to development of the lesions caused by *S. incognita* in the pharynx and the esophagus, and to the death of the birds.

Several viruses, including anatis herpes virus, are known to cause severe diphtheritic pharyngitis and esophagitis in swans, accompanied with necrotic lesions in the liver, intestine, spleen, and kidneys (9). Viral inclusion bodies are usually found in the necrotic lesions in the liver and intestine, and in the epithelial cells of the pharyngeal and esophageal mucosa, as well as in sloughed cells in the vicinity of the lesions (9). In swans reported here, however, liver and intestinal lesions, and characteristic inclusion bodies, were not observed.

In this report, the cases occurred in the late winter, in the period of February to March. This is in accordance with seasonality reported for cases in ducks in England and Australia (2,12).

Remaining swans and other birds were relocated and no other deaths were recorded. As an effective anthelmintic treatment against streptocariasis, levamisole, tetramisole, and mebendazole were suggested (1).

The present report indicates that *Streptocara* spp. could cause fatal disease in swans and, as such, should be included in differential diagnosis of fibrinonecrotic pharyngitis and esophagitis.

## REFERENCES

- Bano, L., A. Natale, M. Vascellary, D. Comin, F. Mutinelli, and F. Agnoletti. First report of parasitic esophagitis by *Streptocara incognita* in Muscovy ducks (*Cairina moschata domestica*) in Italy. *Avian Dis.* 49:298–300. 2005.
- Boughton, E. On the occurrence of oesophageal worms, *Streptocara crassicauda*, in ornamental ducks in Hampshire. *J. Helminthol.* 43:273–280. 1969.
- Dalton, P. J. *Streptocara* infestation of ducks. *Vet. Rec.* 107:384. 1980.
- Fox, J. G., S. B. Snyder, G. D. Schmidt, and L. H. Campbell. Infection with the nematode *Streptocara incognita* in the Chilean flamingo. *J. Wildl. Dis.* 10:66–69. 1974.
- Gibson, G. G. Species composition of the genus *Streptocara* Railliet et al., 1912 and the occurrence of these avian nematodes (Acuariidae) on the Canadian Pacific coast. *Can. J. Zool.* 46:629–645. 1968.
- Griffiths, G. L., D. Hopkins, R. H. Wroth, and W. Gaynor. *Echinura uncinata* infestation in mute swan cygnets (*Cygnus olor*). *Aust. Vet. J.* 62:132. 1985.
- Hunt, G. J., R. N. Krieg, H. A. P. Sneath, T. J. Staley, and T. S. Williams, eds. *Bergey's manual of determinative bacteriology*, 9th ed. Lippincott Williams and Wilkins, Baltimore. pp. 175–289, 527–558. 1994.
- Karlović, M., S. Richter, and Z. Aleraj. Streptocariasis in the Japanese goose (*Sygnopsis sygnoides* L.). *Vet. Arh.* 30:7–12. 1960.
- Keymer, I. F., and R. E. Gough. Duck virus enteritis (anatis herpes virus infection) in mute swans (*Cygnus olor*). *Avian Pathol.* 15:161–170. 1986.
- Laberge, R. J. A., and J. D. McLaughlin. *Hyalella azteca* (Amphipoda) as an intermediate host of the nematode *Streptocara crassicauda*. *Can. J. Zool.* 67:2335–2340. 1989.

11. Laberge, R. J. A., and J. D. McLaughlin. Susceptibility of blue-winged teal, gadwall, and lesser scaup ducklings to experimental infection with *Streptocara crassicauda*. *Can. J. Zool.* 69:1512–1515. 1991.
12. Mason, R. W. Laryngeal streptocariasis causing death from asphyxiation in ducks. *Aust. Vet. J.* 65:335–336. 1988.
13. McDonald, M. E. Catalogue of helminths of waterfowls (Anatidae). In: Bureau of Sport Fisheries and Wildlife, Washington, DC. Special Scientific Report—Wildlife, No. 126. pp. 640–645. 1969.
14. McLaughlin, J. D., and B. P. McGurk. An analysis of gizzard worm infections in fall migrant ducks at Delta, Manitoba, Canada. *Can. J. Zool.* 65:1470–1477. 1987.
15. Randall, J. C., and L. R. Reece. Color atlas of avian histopathology. Mosby-Wolfe, London. 1996.
16. Sterner, M. C., and L. Stackhouse. Parasitic ulcerative ventriculitis in mallards (*Anas platyrhynchos*). *J. Wildl. Dis.* 23:680–682. 1987.
17. Yoshino, T., J. Uemura, D. Endoh, M. Kaneko, Y. Osa, and M. Asakawa. Parasitic nematodes of anseriform birds in Hokkaido, Japan. *Helminthologia* 46:117–122. 2009.

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