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Histopathological Changes in the Gills of Common Carp (*Cyprinus carpio* L.) Infected with the Myxosporean Parasite *Myxobolus koi* Kudo, 1920

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Abstract

Histopathological changes in the gill filaments of common carp (*Cyprinus carpio* L.) infected with the myxosporean parasite *Myxobolus koi* Kudo, 1920 were studied. Infections are confined to the gill lamellae and are characterized by intralamellar plasmodial development. Histological lesions in the gill filaments due to the unilocular "cysts" of the parasite infection were not prominent, but those resulting from multilocular infections were severe. The primary histopathological changes observed in the gill lamellae of infected carp were fusion, inflammation, congestion and necrosis.

Introduction

Species of the genus *Myxobolus* are common parasites of freshwater fishes. In Indonesia, members of this genus are reported to cause serious problems in the culture of common carp *(Cyprinus carpio),* with mortality rates of 60-90% occurring in young carp (Rukyani 1978; Djajadiredja et al. 1983).

A species of *Myxobolus* which produces "cysts" in the gill filaments of common carp has been identified as *Myxobolus koi* Kudo, 1920 (see Rukyani 1978). It is believed to have been accidentally introduced into Indonesia with imported carp (See Djajadiredja et al. 1983), and has since caused considerable losses of common carp in hatcheries.

Great losses to fish culture inflicted by myxosporeans have been widely reported (see, for example, Sachlan 1952; Yasutake and Wood 1957; Hoffman 1963; Sanaullah and Ahmed 1980). However, little is

known about the pathogenic potential of many species (see Mitchel 1977). How the parasite affects the host has often not been determined (Dykova and Lom 1978). The present study was initiated to record the histopathological lesions associated with *Myxobolus koi* infection in common carp.

Materials and Methods

Gill tissue containing cysts of *Myxobolus koi* were removed and fixed in Bouin's fixative and 10% formalin for histological examination. After embedding in paraffin, tissues were sectioned at 6-7 μm thickness and stained with hematoxylin and eosin. Additional staining with Giemsa's stain, and Gomori's One Step Trichrome for collagen, were also done.

Fresh mounts and fixed impression smears of the organs were also made. The tissue sections and the smears were examined with light microscopy for the presence and distribution of spores and for histopathological changes.

Results

Lesions were limited to the gills. Inflammation, congestion and hyperplasia were the most common changes observed in the gill epithelial tissue. In multilocular infections, hyperplasia of the gill epithelial cells caused fusion of the lamellae. In many cases, dilation of the blood vessels with the accumulation of blood occurred. Blood accumulation was often extensive, so that it appeared to occupy most of the area of the cyst. A chronic inflammatory response was observed within the epithelial tissue surrounding the multilocular cyst. Inflammatory cells, lymphocytes, macrophages and a few fibrocytes were the most prominent components forming the layer surrounding the multilocular cyst. The inflammatory cells were covered by an outermost and innermost layer two to three epithelial cells thick. In multilocular cysts, blood capillaries were found between the plasmodia.

Mature plasmodia released spores into the water through a rupture of the plasmodial membrane, causing destruction of the adjacent blood sinusoids. Advanced stages of destruction in the unilocular plasmodia were not observed. In multilocular cysts, the

spores were gradually released from the outer, then from the inner plasmodia. Mature plasmodia eventually assumed an irregular shape due to infiltration with fibrocytes and phagocytic cells.

Following cyst rupture an inflammatory reaction occurred, indicated primarily by the infiltration of lymphocytes and macrophages. Cyst regression then occurred as an advanced stage of the inflammatory reaction. Macrophages removed remaining spores by phagocytosis while granulation tissue formed as a late stage of repair at the site of infection. In contrast, some cysts had markedly dilated and congested capillaries, and most of the areas of the multilocular cysts were occupied by masses of blood. The rupture of the cysts caused hemorrhage and necrosis of gill tissue. Most of the gill filaments were damaged as a consequence, leaving only their cartilagenous rods.

Discussion

Lesions were confined to the gill lamellae. The presence of plasmodia exerted pressure on adjacent gill tissue and reduced the respiratory surface, particularly in cases of heavy infection with macroscopic cysts, or those with microscopic plasmodia in the lamellae. Diseased fish exhibited clinical signs similar to those described by Hoshina (1952) in common carp, and by Sanaullah and Ahmed (1980) in major Indian carps. These included stunted growth, lethargy, increased susceptiblity to low dissolved oxygen concentration, crowding near the water surface and weakness. Infected fish often died, particularly during transportation or when kept in holding tanks for marketing.

The lesions associated with infection of *Myxobolus koi* were generally similar to those described for other myxosporean infections in gill tissue (see McCraren et al. 1975; Minchew 1977; Dykova and Lom 1978). The inter- and intralamellar forms of *Henneguya* spp. have been extensively described. However, in the present study, intralamellar plasmodia were the only form found in the gills; the characteristics of this form are only recognized in the unilocular plasmodia. The intralamellar form as the only form found in the gills was also reported by Sanaullah and Ahmed (1980) in the gills of carp and by Crawshaw and Sweeting (1986) in gills of koi.

Intralamellar plasmodia are known to be less pathogenic than interlamellar plasmodia (McCraren et al. 1975). However, in the

present study this form is also potentially pathogenic to common carp fingerlings, particularly when a large number of plasmodia are present in the gills. Rupture of plasmodia may also cause damage to the blood sinuses and capillaries, eventually impairing the respiratory function of the gills.

Unilocular cysts in the gill filaments may occur in 100% of the infected fish population without noticeable external symptoms. The destruction of the gill filaments caused by the unilocular cysts is also harmful, while the multilocular form is undoubtedly a major cause of mortality. Fish infected with multilocular cysts usually exhibit hypoxia by swimming at the surface of the pond with flared opercula. Histologically, dilation appeared to be the most prominent lesion that occurred in the capillaries surrounding the plasmodia of the multilocular cysts. Therefore, the visible pinkish color of the cysts may be due to the accumulation of blood. The normal respiratory function of the gills may be impaired as most of the gill lamellar surface for gas exchange was covered by cysts. The condition of the gills became more serious after the cysts ruptured, leaving necrotic tissue, and the fish died due to the nearly complete loss of respiratory function.

After releasing mature spores from the ruptured plasmodia, the cysts gradually became smaller. After about one month, they usually disappeared, leaving only damaged gill tissue. In the sections, the cysts appeared to be in the process of destruction due to inflammation. The cysts were infiltrated with connective tissue and macrophages which formed granulation tissue. In the recovery process (unpublished data) about 50% of infected fish with visible cysts died, and after one to two months, only 8% showed visible cysts. Histologically, the host tissue reaction in the healing process was demonstrated. However, the subsequent pathological effects are not known. Reinfection may occur but did not produce visible cysts on the gills of larger fish.

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References

Crawshaw, M.T. and R.A. Sweeting. 1986. *Myxobolus koi* Kudo, 1919: a new record for Britain. J. Fish Dis. 9: 465-467.

Djajadiredja, R., T. H. Pandjaitan, A. Rukyani, D. Satyani and H. Supriyadi. 1983. Country Report. Indonesia, p. 19-30. *In* F. B. Davy and A. Chouinard (eds.) Fish quarantine and fish diseases in Southeast Asia. Report of workshop held in Jakarta, Indonesia, 7-10 December 1982. Intern. Developm. Res. Centre, Ottawa. Publ. No. IDRC-120e.

Dykova, I. and L. Lom. 1978. Histopathological changes in fish gills infected with myxosporidian parasites of genus *Henneguya*. J. Fish Biol. 12: 197-202.

Hoffman, G.L. 1963. Whirling disease in trouts and its prevention in hatcheries. U.S. Trout News 8:8-18.

Hoshina, T. 1952. Notes on some myxosporidian parasites on fish of Japan. J. Tokyo Univ. Fish. 39: 69-97.

McCraren, J.P., M.M. Landolt and G.L. Hoffman. 1975. Variation in response of channel catfish to *Henneguya* sp. infections (Protozoa: Myxosporidea). J. Wildl. Dis. 11:2-7.

Minchew, C.D. 1977. Five new species of *Henneguya* (Protozoa: Myxosporidia) from ictalurid fishes. J. Protozool. 24: 213-220.

Mitchel, C.W. 1977. Myxosporidia, p. 115-157. *In* J.P. Krier (ed.) Parasitic Protozoa. Vol. IV. Academic Press, New York.

Rukyani, A. 1978. Two species of Myxobolidae (Myxosporidia) from freshwater fishes in Java. Pew. Lemb. Penel. Perik. Dar. 1: 24-25.

Sachlan, M. 1952. Notes on parasites of freshwater fishes in Indonesia. Contr. Inland Fish. Res. Sta. Indonesia 2: 1-22.

Sanaullah, M. and A.T.A. Ahmed. 1980. Gill myxoboliasis of major carps in Bangladesh. J. Fish Dis. 3: 349-354.

Yasutake, W.T. and E.M. Wood. 1957. Some Myxosporidia found in Pacific northwest salmonids. J. Parasitol. 43: 633-642.