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Histopathological Changes in the Intestine of the Fish, *Synaptura orientalis* (Bl. & Sch.) Parasitised by an Acanthocephalid Worm, *Echinorhynchus veli* (George & Nadakal, 1978)

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Introduction

The acanthocephalans, as adults, occur as intestinal parasites of various vertebrates. They usually remain hooked up to the intestinal wall of their hosts and set up several tissue reactions and histopathological changes in the intestine. The parasitised animals may also suffer from nutrient depletion and intestinal occlusion depending upon the worm burden. Heavy mortality of animals infected by various species of acanthocephalans has also been reported (Webster, 1943; Clark *et al.* 1958; Takos and Thomas, 1958). A few acanthocephalan parasite host system has been studied with respect to intestinal histopathology; thus the fish, largemouth bass and *Neoechinorhynchus cylindratus* and *Leptorhynchoides thecatus* (Venard and Warfel, 1953); Starry flounder and *Echinorhynchus lageniformis* (Prakash and Adams, 1960); Salmonid fishes and *Acanthocephalus jacksoni* (Bullock, 1963); Catastomid fishes and *Pomphorhynchus bulbocolli* and *Octospinifer macilentis* (Chaicharn and Bullock, 1967); captive primates and *Prosthenorchis elegans* (Schmidt, 1972) and acanthocephalan infections in Rainbow trout (Abe, 1973). Divergent histopathological observations have been made by these workers in accordance with the nature and type of host-parasite system. The present report is concerned with the results of a study designed to throw light on the histopathological alterations made in the intestine of the fish, *Synaptura orientalis* parasitised by *Echinorhynchus veli.*

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Materials and Methods

Live specimens of brackish water fish, *S. orientalis* were collected from Veli Lake, Trivandrum. About 500 fish were sacrificed in an attempt to study the taxonomic, pigmentary and histopathological aspects of *E. veli.* The host intestine was dissected out and cut open longitudinally. Measurements of small intestine were taken and the number of worms recovered was noted. Body weight differences between the infected and uninfected fish were recorded. To establish the weight difference between infected and uninfected fish, the condition factor was calculated from a formula $\frac{w}{L^3} \times 10^3$. Observation was also made in regard to the specific localisation of worms within the gut. Intestinal contents other than worms were then washed in physiological saline and the tissues fixed *in situ* with Carnoy's fluid. The split intestine was cut into small bits with attached regions of the worm and the microtome sections of

6–8 μm thickness were taken. They were stained with Harris' haematoxylin-eosin following the routine procedure. Sections of uninfected fish intestine served as controls.

Observations

Infection rate of *S. orientalis* with *E. veli* was found to be heavy throughout the year. As many as 112 worms were recovered from a single host (Fig. 6). They were mostly found localised in a region 10 cm anterior to the cloaca and the occurrence of worms was remarkably low in duodenal and posterior part of the intestine. Gravid worms were found attached in the anterior and the immature worms in the posterior infection sites of the intestine.

The infection-free fish intestinal wall is 180–230 μm thick (Fig. 1) and the intestinal mucosa is not heavily thrown into folds. The globular proboscides and neck regions of worms were found embedded within the intestinal wall. The worms ruptured the villi, submucosa and penetrated deep into the muscularis (Fig. 2). This resulted circumscribed destruction of mucosa, submucosa and even muscular layers. The proliferation of fibroblasts and fibrous tissues of intestinal wall around the proboscis of worm gave rise to spongy granulomatous nodules (Fig. 5). The nodules pressed heavily on the muscular layers and the latter thus appeared thinned out in these regions. The nodule formation probably reflects only the more restricted reactions of the fish tissues to injury, compared to that seen in mammals and birds. In heavily infected host, large number of white spherical nodules were observed on the inner wall of the host intestine. In advanced stage of infection, the intestinal wall showed enlarged nodules measuring 480–500×670–700 μm (Fig. 3). Total destruction of the intestinal mucosa and submucosa was observed at the point of attachment. Besides these, hypertrophic and hyperplastic changes were also detected in the epithelial cells of mucosa along the infection site. Necrosis of cells surrounding the worm was slight. There were a few lymphocyte-like cells, macrophages and erythrocytes bordering the nodules. Epithelial cells, fibroblasts and cells resembling plasma cells of mammalian type were also seen in large numbers at the site of infection. The goblet cell number in the intestinal mucosa was found to be the same as in uninfected area.

It was found that the worms remained permanently in one place long enough to stimulate the formation of granulomatous nodules in the host intestine. In heavy infections, the worms produced multiple nodules (Fig. 4) and caused intestinal occlusion. The microscopical observations of lesions were revealed that there was no secondary bacterial infection and the mucous secretions remained normal in the infected area. A remarkable difference in body weight was noted between uninfected and infected fish host of 13–15×6–8 cm size. Average of 10 uninfected fish was 35.6 (21–48) while that of the infected fish was 20.7 (17–26).

Discussion

Parasitism by acanthocephalid worms is generally considered harmful to their hosts. They cause nutritional depletion, serious tissue damage, reduced absorptive efficiency of the intestine and even mortality of the host. The severity of the histopathological changes caused by these worms is generally correlated with depth of penetration to the host tissues and also with worm burden. In the present instance, *E. veli* penetrated deep into the intestinal wall causing destruction of tissues and producing spongy nodules, the size and number of which depend on the duration and degree of infection respectively. In strict pathological sense, these

nodules are mild parasitic granuloma which are quite unlike what is usually seen in mammalian tissues. The development of nodules in the intestinal tissues of *S. orientalis* resembles those reported in fishes such as whitesucker infected by *Pomphorhynchus bulbocolli* and *Octospinifer macilentis* (Chaicharn and Bullock, 1967); starry flounder infected by *E. lageniformis* (Prakash and Adams, 1960) and a bird infected by *Pomphorhynchus boschadis* (Pflugfelder, 1956). It is of considerable interest to note at this instance the experimental finding of Nadakal *et al.* (1973) that the cestode, *Raillietina echinobothrida* cause parasitic granuloma in the intestine of chickens. Schmidt (1972) has also described nodule formation, peritonitis, intestinal occlusion and secondary infection of lesions by bacteria in marmoset monkeys infected with *Prosthenorchis elegans.*

Neither deep penetration nor nodule formation has been observed in the gut of trout infected with *Acanthocephalus jacksoni* (Bullock, 1963); in brown trout by *E. truttae* (Marochino, 1926); in largemouth bass by *Neoechinorhynchus cylindratus* (Venard and Warfel, 1953) and in *Rachycenteron canadus* by *Serrasentis nadakali* (George and Nadakal, 1981) but cellular invasion at the infection sites was evident in all these instances. Formation of nodules and mobilization of cell types such as macrophages and lymphocyte-like cells in the lesions may be interpreted as a reflection of immunological responses of host fishes. Hyperplasia of epithelial cells along with the accumulation of fibroblasts around the site of infected area is probably a regenerative phenomenon associated with the injury of tissues. Another observation made in this study is that goblet cells in the vicinity of infection site did not differ in their number and activity from that of cells in the infection-free area of intestine.

Tissue damages were caused due to mechanical injury inflicted by worms by their armed conical proboscides. It could be intensified by secondary bacterial infections of the intestinal lesions. However, in *S. orientalis* there appears no indication of presence of bacteria associated with the lesions. A similar situation has also been reported for the starry flounder infected by *E. lageniformis* (Prakash and Adams, 1960).

Survey of large number of *S. orientalis* specimens revealed that the frequency and intensity of infection of this fish with *E. veli* are surprisingly high. As many as 112 worms were recovered from a single fish host. The presence of such a large number of worms in the intestine which is heavily nodulated internally, is likely to cause intestinal obstructions leading to fatal consequences. Significant weight loss of infected fish and the nature of histopathological changes in the intestine of parasitised fish indicate that *E. veli* is physiological hazard to the normal growth and development of fish, *S. orientalis.*

Summary

The acanthocephalid worms, *Echinorhynchus veli* by deep penetration into the intestinal wall of its host fish, *Synaptura orientalis* causes serious tissue damage including rupturing of the intestinal villi, submucosa and even muscularis. There was a total destruction of epithelial cells and submucosa at the point of worm attachment. Hyperplasia of the epithelial cells around the infected sites of intestine was also observed. Nodular formation by the proliferation of fibroblasts and fibrous tissues of intestinal wall around the proboscis of worm was of common occurrence. In heavy infections, the worms produced multiple nodules and intestinal occlusion. There was also accumulation of cell types such as macrophages, lymphocyte-like cells, erythrocytes and fibroblasts at the injured regions of the intestine.

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鉤頭虫 *Echinorhynchus veli* に寄生された魚 *Synaptura orientalis* の腸における組織病理学的変化

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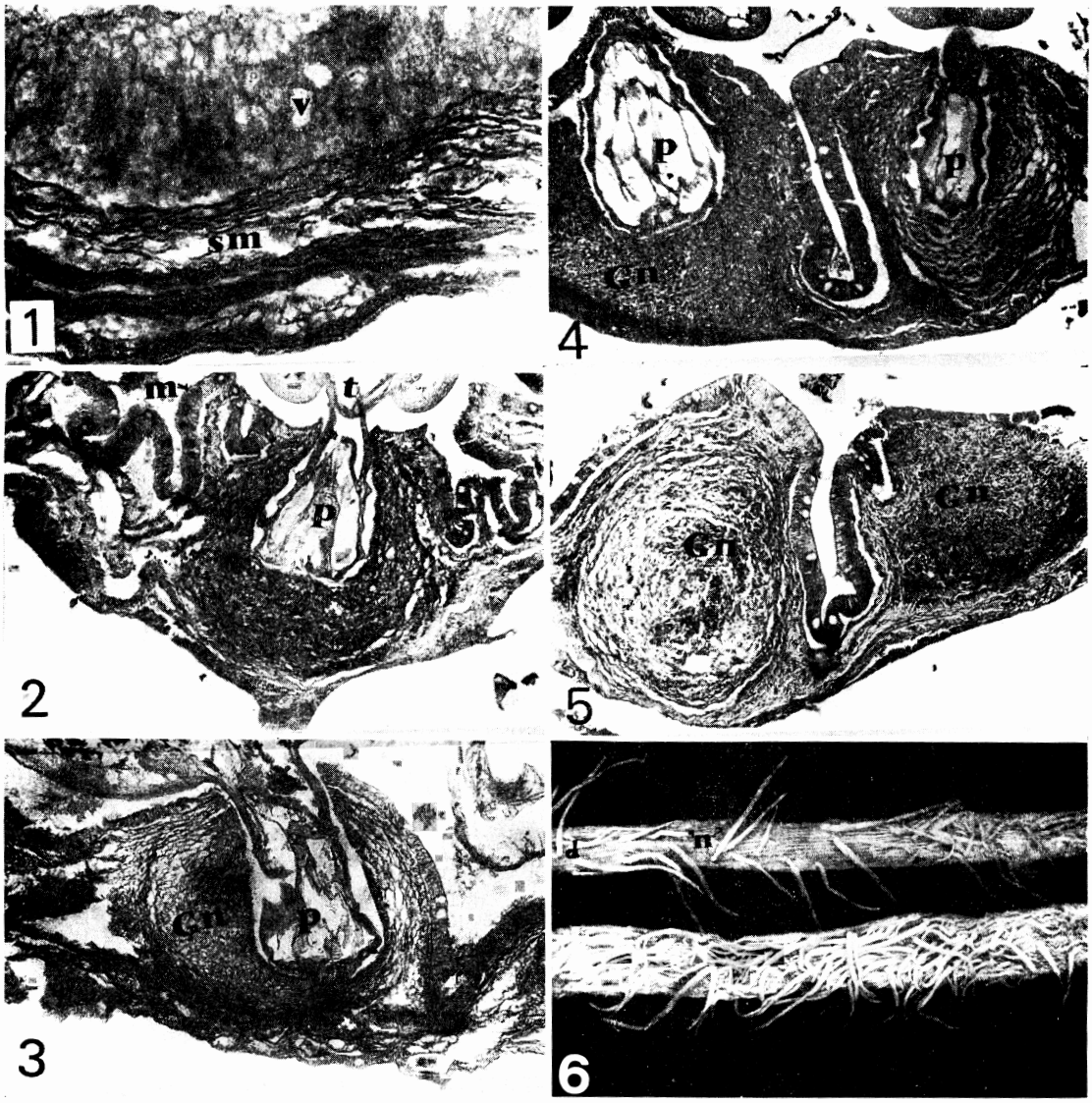
鉤頭虫 *Echinorhynchus veli* は，魚の *Synaptura orientalis* の腸壁深く穿入することによって，腸絨毛，粘膜下組織，それに筋肉さえも破壊する重症の組織障害をひきおこす．

虫の咬着点では，上皮系細胞や粘膜下組織の完全な破壊も認められた．腸管の感染部位周辺には上皮系細胞の増殖も観察された．虫の吻周囲の結合組織の集積による結節形成は普通にみられた．濃厚感染時には，虫は多数の結節を作り，腸管の閉塞をおこした．腸管傷害部には，また，マクロファージ，リンパ球様細胞，赤血球，線維芽細胞のような細胞の集積もあった．



Explanation of Figures

Fig. 1 Uninfected intestine of *S. orientalis* 400×.
Fig. 2 Infected area of intestine by *E. veli* 100×.
Fig. 3 An advanced stage of infection by *E. veli* 100×.
Fig. 4 Multiple nodulated area of infection 100×.
Fig. 5 Granuloma of the intestine of *S. orientalis* developed by the infection of *E. veli* 100×.
Fig. 6 *E. veli* in the small intestine of *S. orientalis* (Photo. by T. I. Jacob) 1.5×.