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Studies on Encapsulation of Immature Juvenile of the Acanthocephalid Worm, *Pallisentis nagpurensis* Bhalerao, 1931 in the Liver of Definitive Host, *Ophiocephalus striatus* (Bloch.)

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Introduction

Adult acanthocephalans inhabit the intestinal tract of vertebrates. The poikilothermic vertebrates also harbour these worms in their deeper tissues, serving as paratenic host. A significant aspect of acanthocephalan biology is that the immature juveniles of certain acanthocephalid worms can encapsulate in the mesenteries or liver of their final host. A number of instances of occurrence of encapsulated worms in the liver of final hosts has been reported. The worm encapsulation in the hepatic tissue of definitive host is known to cause serious histopathological changes and tissue inflammation. Information in this aspect of acanthocephalan biology is fragmentary. The present study is focused on the process of encapsulation of juvenile, *P. nagpurensis* and histopathological changes caused by them in the liver of the definitive host, *Ophiocephalus striatus*.

Materials and Methods

The gravid worms collected from the fish, *O. striatus* served as the source of viable eggs. The infection-free *Cyclops strennus* was kept in small bottles containing pond water and the embryonated eggs were introduced into them. The cyclops containing infective and uninfective juveniles were fed to the infection-free fish fry reared in small tanks. The worms were recovered from the infected fish host at regular intervals upto 50 days. The worms were recovered from the intestine as well as from the liver. The encapsulated worms intact with the liver and the intestinal tissues were fixed in Carnoy's fluid. Paraffin sections of 6–8 μm thickness were taken from the fixed tissues as usual and stained with Harris' haematoxylin-eosin. The uninfected liver tissues served as control. Encapsulated *P. nagpurensis* were also recovered from *Macropodus cupanus, Aplocheilus melastigma* (McClelland), *Barbus* sp., *Ophiocephalus gachua* (Ham. and Buch.), *Wallago attu* (Bloch.), *Heteropneustes fossilis* (Bloch.) and *Rana tigrina* (Daud.).

Results

P. nagpurensis attains its infectivity in *Cyclops strennus* 15–20 days post-infection and as many as 8 juveniles including both infective and uninfective juveniles were recovered from their haemocoel (Fig. 1). On ingestion, the former settled in the intestine

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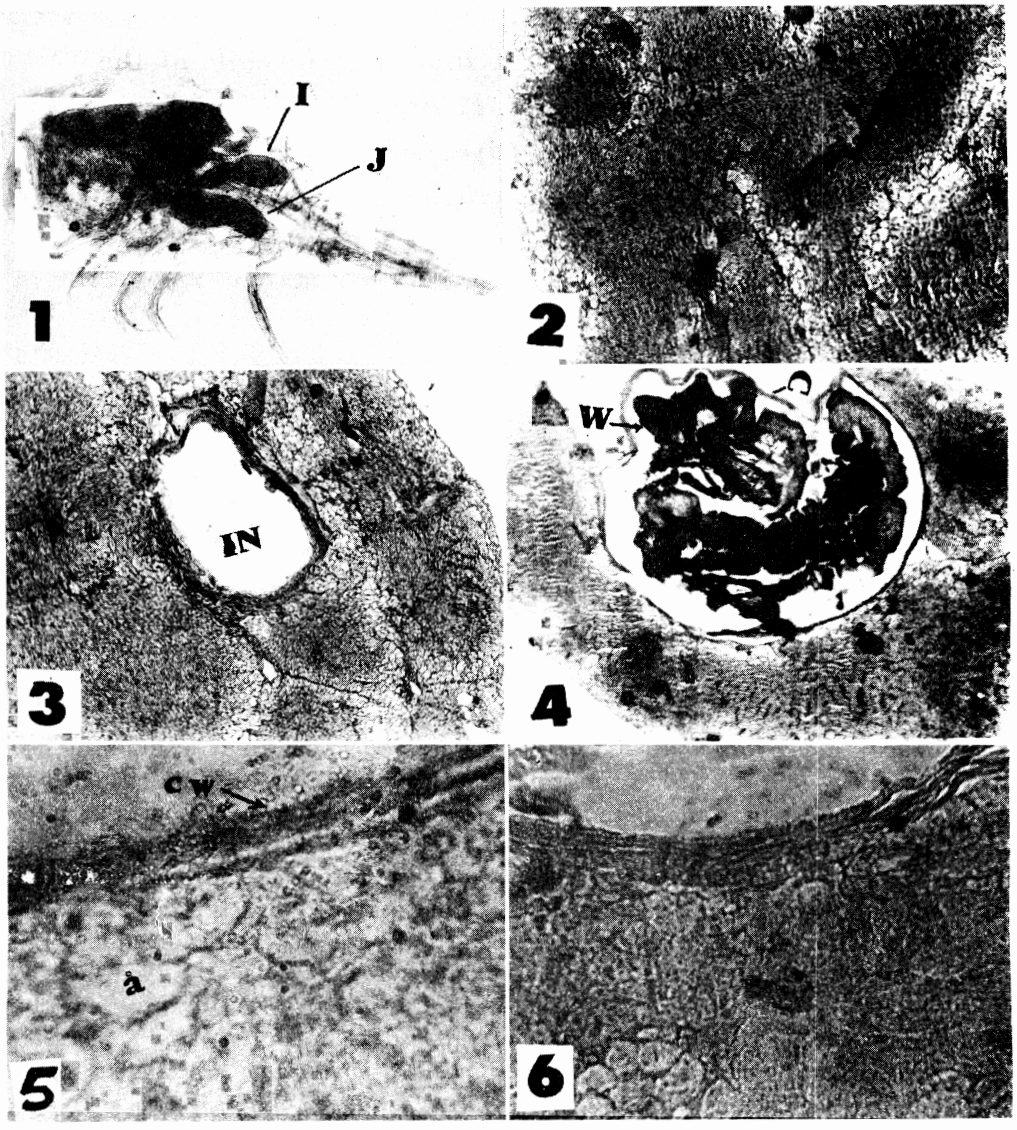

Explanation of Figures

Fig. 1 Infected cyclops by *P. nagpurensis* 100×.
Fig. 2 Uninfected liver of *O. striatus* 100×.
Fig. 3 Primary phase of encapsulation of *P. nagpurensis* 100×.
Fig. 4 Secondary stage of encapsulation of *P. nagpurensis* 70×.
Fig. 5 The liver tissues around the worm infection 1,000×.
Fig. 6 The liver tissues around the cyst wall 450×.

Abbreviations

a, area around the cyst; C, cyst; C. W. cyst wall; I, immature juvenile; IN, infected area; J, juvenile and W, worm.

and the latter emigrated to the liver (Fig. 2) of the final host. Within the final host, the immature juveniles penetrated through the intestinal wall and reaching the liver encapsulated therein. The larval emigration and encapsulation in the liver caused certain degree of tissue damage and inflammation. The former event initiates the process of capsule formation around the parasite. The permeability changes occur in the blood vessel surrounding the worm so that the plasma exudes and blood cells accumulated at the point of leakage. Blood circulation tended to slow down and the leucocytes migrated through the capillary walls and enveloped the worm. Later, host macrophages and other cells followed and reinforced the leucocytes. The parasite now becomes enclosed in a double walled, transparent and fluid filled capsule. The encapsulated worms were found in the outer part of the liver lobes. The tendency of immature juvenile for encapsulation in the definitive host is frequent in the young fish population.

The infective and uninfective juveniles measured 0.5–1×0.1–0.125 mm and 125–180 ×60–85 μm respectively. An encapsulated worm has attained the size of 3.1–4.1×0.3–0.31 mm. Four encapsulated worms were obtained from an infected fish. When released from the capsule, they showed all characteristics of mature worms. In the early stage of infection, the worms occupied usualy an area of 0.27–0.36×0.34–0.35 mm in the inner part of the liver (Fig. 3). On encapsulation, a worm seems to have destroyed an area of 0.57–0.67×0.4–0.51 mm of hepatic tissues (Fig. 4) and the encapsulated worm is exposed to the periphery of liver. The cells around the parasite showed vacuolar changes in the cytoplasm, focal degeneration and deterioration of cell structures. The intralobular blood vessel around the worm were engorged, probably as a result of dissolution of groups of liver cells around the worm and consequent release of pressure by the parasite.

Upon the ingestion of infected cyclops, the infective juveniles settled down in the intestine responding to certain intestinal stimuli. Soon the proboscides become everted and the worm grew rapidly and attained the characteristic pattern of hook arrangement. Sexual dimorphism became more marked.

Both immature and mature juveniles of *P. nagpurensis* encapsulated in the liver or mesenteris of transport hosts. The process of encapsulation in the transport host is similar to that in the definitive host. The encapsulated worms were found only in the mesenteries of *Wallago attu, Heteropneustes fossilis, O. gachua* and *R. tigrina.* However, they occur encapsulated in the liver of *M. cupanus, Aplocheilus melastigma* and *Barbus* sp. Encapsulated worms of *P. nagpurensis* were found under the process of degeneration in the mesenteries of *R. tigrina* and *O. gachua.*

Discussion

Encapsulation of immature juveniles of *P. nagpurensis* in the deeper tissues of hosts regardless of whether they are definitive or transport host, seems to be an inevitable adjunct to the life cycle of this worm. The juveniles before reaching infectivity can not survive in the intestinal environment and so they emigrate to the liver and become encapsulated. If they are ingested by a definitive host, they would resume their life cycle in the intestine. This would rendered easy by encapsulation of worms in young definitive host since the cannibalistic behaviour is frequent among the young *O. striatus* (George and Nadakal, 1973). Similar case of encapsulation of immature juvenile acanthocephalans has been reported in several definitive hosts (DeGiusti, 1939; Connell and Corner, 1957; Meads and Harvey, 1968 and Kanda, 1958).

The encapsulated worms remain viable

for a short period in the host and soon they undergo degeneration. Degenerate encapsulated worms have been reported from the mesenteries of the hosts (Ward, 1940; Connell and Corner, 1957 and Gupta, 1950). The process of encapsulation in the definitive hosts may be terminal for the parasites concerned unless they develop further to a state leading to normal infection and ingested by a cannibalistic definitive host (Kanda, 1958). This favours the re-establishment of worms in the intestine of final host. Such instances have been reported in *O. striatus* (George and Nadakal, 1973) and Weasels, *Mustela itatsi itatsi* (Kanda, 1958).

Adult acanthocephalans cause nutrient depletion, intestinal injury and mobilisation of cell types around the site of infection of host fishes (George and Nadakal, 1981). However, the encapsulated *P. nagpurensis* evoked to produce focal degeneration, deterioration of cell structures and the engorgement of intralobular blood vessels of liver.

The localisation of encapsulated worms towards the periphery of the liver lobes may be explained on the basis of worm's response to the host tissue reactions. It may also be interpreted that they simply occupy the physicochemically congenial site for encapsulation. The worms recovered from the liver showed marked size difference in comparison with the immature juveniles. This is an indication of growth during the sojourn of the worm in the liver.

The encapsulated worms remain bathed in a fluid similar in chemical composition to blood plasma and thus they find themselves in an extremely favourable milieu. Crompton stated (1970) that encapsulated worms inhabit an environment similar in some respects to the final host's small intestine: survival is possible because the parasites are adapted to obtain energy independently of oxygen. The double walled capsule was observed around the encapsulated *P. nagpurensis*. Similar instances were reported in the liver of fishes. *Lepomis* spp. (Bogitsh, 1961) and *Trichogaster chuna* (Hasan and Qasim, 1960).

Several transport hosts have been encountered in the life cycle of *P. nagpurensis* (George and Nadakal, 1973). The encapsulation of both immature and mature juveniles also occurs in several transport or abnormal hosts. The reason for emigration of infective juveniles from the intestine to the deeper tissues of transport hosts may be sought the incompatibilities of intestinal habitat for their growth and reproduction (Crompton, 1970). The encapsulation of *P. nagpurensis* in the deeper tissues of transport hosts also follow the same pattern of reaction as seen in the definitive host, *O. striatus*. The transport hosts may harbour encapsulated *P. nagpurensis* either in their liver or in the mesenteries. The exact stimulus for diverse habitat selection by the same parasite is not known. This may be due to histological differences existing in various hosts. The transport hosts are generally indispensable for the completion of acanthocephalan life cycle with which they are associated (Baer, 1961).

Summary

The juveniles of the acanthocephalan, *Pallisentis nagpurensis* attain infectivity in the haemocoel of *Cyclops strennus* 15–20 days post-infection. The infected *Cyclops strennus* harbours both infective and uninfective juveniles. On ingestion of infected cyclops by definitive host, the immature juveniles emigrate from the intestine to liver and become encapsulated therein. Encapsulated worms undergo developmental changes and cause histopathological changes in the hepatic tissues. The process of encapsulation and host tissue reactions were observed.

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鉤頭虫 *Pallisentis nagpurensis* Bhalerao, 1931 幼若虫の終宿主 *Ophiocephalus striatus* (Bloch.) の肝における被嚢に関する研究

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終宿主 *O. striatus* の腸管より採取した鉤頭虫 *P. nagpurensis* の虫卵と cyclops strennus を含む池水中で培養して cyclops に感染させ，感染 cyclops を *O. striatus* の幼魚に摂食せしめ，50日間にわたり発育を追跡した．cyclops 血体腔で充分な発育をとげた幼虫は腸管にとどまり成虫に発育するが，未成熟の幼虫は肝に移行し，被嚢する．

この被嚢は待期宿主中でもおこる．被嚢幼虫自体は成虫の形態をとるが，再度終宿主に摂食されない限り，変性し死滅する．幼虫の肝表面下侵入により，肝実質の破壊と虫体周辺に炎症細胞が集積し，虫体は二重の嚢壁につつまれ，透明な液性嚢胞を形成する．この被嚢形成は待機宿主では自然の経過である．