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MICROSPORIDIOSIS IN *PENAEUS MONODON* CULTURED IN THE BHERIES OF WEST BENGAL, INDIA

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Bhery fisheries play a significant role in the total shrimp production of West Bengal. Due to the traditional practices and utilization of untreated sewage water, the shrimps of the *bheries* are under the threat of parasitic attack. Microsporidians, a protozoan endoparasite, can cause shrimp mortalities and consequent decrease in total harvest in the systems like Bhery fishery. During this study, it was observed that the wild aquatic organisms are the main carriers of Microsporidiosis and the incidence level was 1.17%. The muscle fibers were degenerated due to Microsporidiosis and numerous small minute spores were diffusely distributed throughout the muscle.

INTRODUCTION

Microsporidiosis is otherwise known as 'Cotton shrimp disease' has generally been recognized by the progressive white opacity associated with the musculature. Microsporidians such as *Agmasoma* (=*Thelohania*), *Ameson* (=*Nosema*) and *Plistophora* (=*Pleistophora*) have been reported in shrimps/prawns of several genera notably *Penaeus, Pandalus* and *Crangon*. They cause diseases in epizootic proportions in feral crustaceans (Overstreet, 1973; Sindermann, 1990).

Bheries or wetland in West Bengal covering approximately 32,930 ha water spread area, is contributing a major portion of prawn/shrimp fishery (Pandit, 2000). The traditional practice in this system invites pathogen and a significant reduction in total production has been observed due to disease outbreak in recent years, mainly by white spot disease. Protozoan diseases also cause a significant health hazards in shrimp/prawn. The entry of wild organisms through incoming water into the system carries unwanted organisms (wild prawns, other crustaceans, small fishes, molluscs etc), which are considered as carriers of different pathogens. The present investigation was undertaken to determine the prevalence and pathology of microsporidians in *Penaeus monodon* in the *bheries* of West Bengal.

MATERIAL AND METHODS

A total of 769 live specimens of *Penaeus monodon* with carapace length ranging from 5 cm to 30 cm were randomly taken for pathological observations from the harvest of the *bheries* (8 sampling stations include 47 *bheries*) located in North and South 24 Parganas districts of West Bengal, India, during February-November, 2001. The gross and clinical signs and other abnormalities were observed carefully at site. Shrimps with opacity in the abdominal musculature were fixed in Davidson's fixative and processed as described by Bell and Lightner (1988) for histopathological observations.

RESULTS

The infected shrimps were weak, milky white and exhibited opaque abdominal musculature (Fig. 1). Shrimps weighing 5-10 g showed the clinical signs of cotton shrimp disease. Among the collected shrimps only nine had symptoms typical of Microsporidiosis. The incidence rate was 1.17% only. The histopathological sections of normal shrimps showed perfect myofibrilar arrangement compared to the affected shrimps (Fig. 2).

The squash preparations of muscle tissues from infected shrimps revealed degeneration of myofibrils and presence of numerous small spores. Histopathological examinations of the infected mussel tissues revealed that the microsporidians slowly degenerated the muscle fiber and replaced the muscle tissues with spores (Fig. 3). In the early phase of infection only small portions of the muscle fibers were infected; however, as the disease progressively spread over a greater area the muscle fibers were gradually transformed into necrotic structure. No clear pansporoblast membrane was seen and diffuse distribution of single spores in the muscle was observed. Heavy haemocytes infiltration in the gill and muscle of infected shrimps was seen (Figs. 4 & 5). Extensive accumulation of *Zoothamnium* sp., an ectocommensal peritrichous free living ciliate was noticed in the gill of microsporidian infected shrimps (Fig. 6).

Other organs affected include ovary, gut epithelial layer and hepatopancreas. Hepatopancreatic tubular degeneration was of common occurrence in the milky white shrimps (Fig. 7). Muscle tissues around intestine showed heavy accumulation of microsporidian spores (Fig. 8).

DISCUSSION

The number of spores per sporant could not be accurately determined, as it requires electron microscopic observations. However, the tissue infection characteristics are almost specific for each microsporidian species (Kelly, 1979). From the description of Anderson *et al.* (1989), the observation of diffuse distribution of many minute single spores in the muscle the microsporidian might be placed under genus *Ameson* (= *Nosema*).

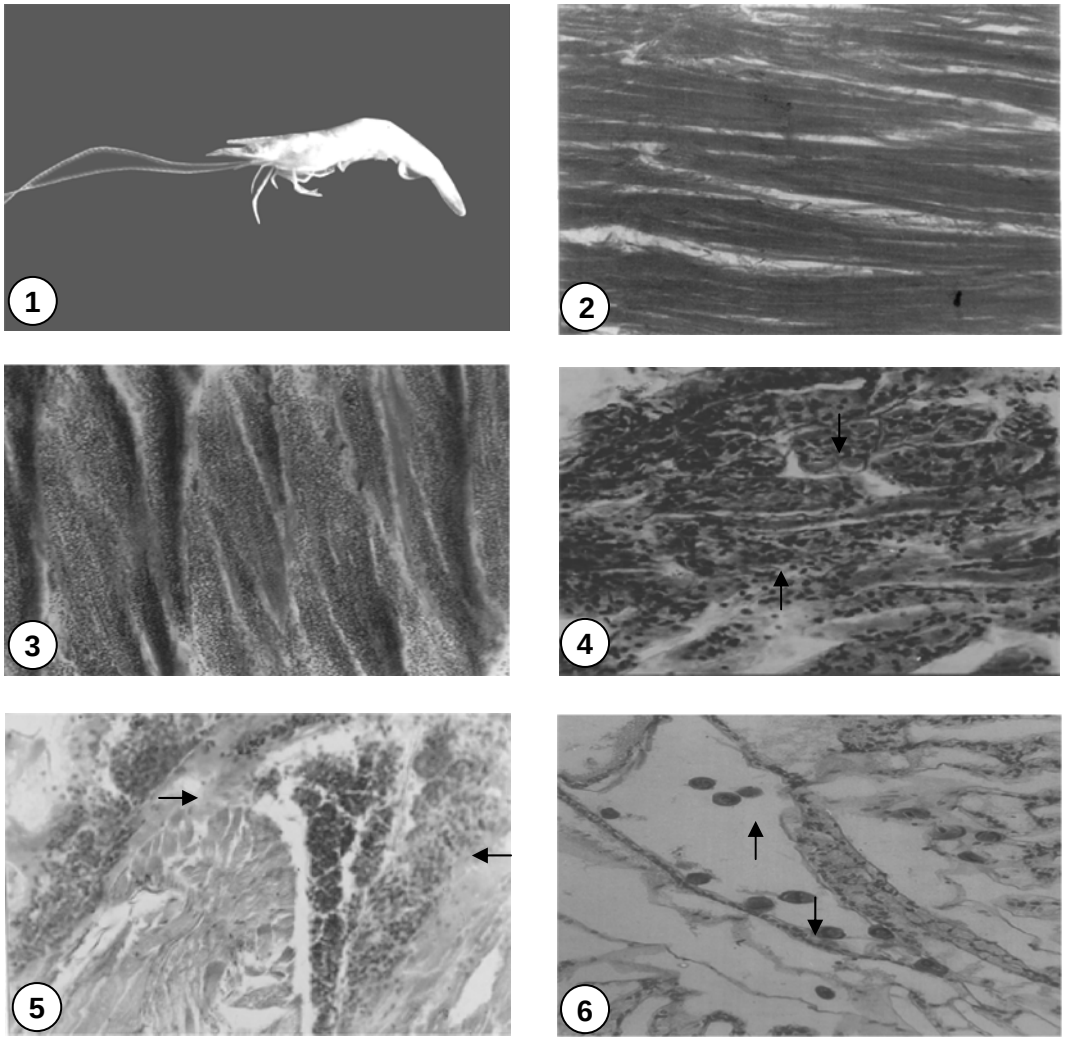

Fig. 1. Microsporidian infected shrimp with milky white appearance collected from the *Bheries* of West Bengal; Fig. 2. Longitudinal section of muscle of a normal shrimp showing perfect arrangements of muscle fibre (H & E X 200); Fig. 3. Histological section of the infected muscle showing diffuse distribution of microsporidian spores and subsequent muscle degeneration (H & E X 400); Fig. 4. Marked haemocytic response (Arrow) in the gill of microsporidian infected shrimp (H&E X 400); Fig. 5. Marked haemocytic response (Arrow) in the muscle of microsporidian infected shrimp (H&E X 400); Fig.6. Gill section of Microsporidian infected shrimp showing association of ectocommensal ciliate, Zoothamnium (Arrow) (H&E X 400).

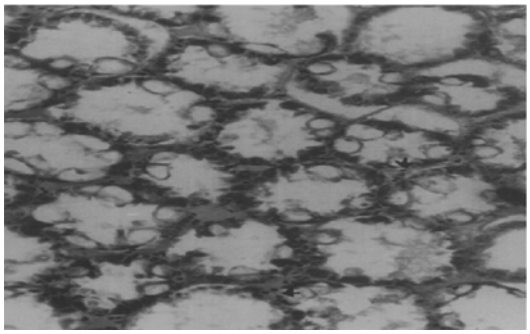

Fig. 7. Hepatopancreatic section of Microsporidian infected shrimp with tubular degeneration. Normal star shaped lumen is absent (H&E X 400)

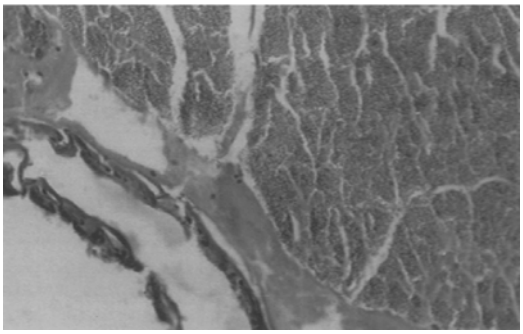

Fig. 8.Muscle tissue around midgut intestine showing degeneration by Microsporidian spores. Note, the degenerated midgut epithelial layer. (H & E X 200)

The muscle fiber degeneration observed in this study was similar to those described by Johnson (1990) and Ramasamy *et al.* (2000). The mechanism of muscle fiber degeneration and replacement by the Microsporidians yet remain unknown (Ramasamy *et al*., 2000). The heavy haemocytic response in the gill and muscle tissues might be a consequence of the first line of defense against the parasitic attack. The weight of the infected shrimp (5-10 g) indicated that the infection is common with advancing age, which agreed with the report of James (1986).

Although the Microsporidian infection rate in *bheries* was less and not so common, it should not be ignored as it has potential to cause mortality in shrimp. Since, most of the *bheries* receive sewage water without treatment, there is every chance of getting carriers such as weed fish, shrimp, molluscs and others into the system. According to Overstreet (1973) and Johnson (1990) wild shrimps are the potential carriers of Microsporidiosis.

The life cycle of microsporidians and it's mode of transmission are not clearly known till date (Limsuwan, 1993) and therefore, the nature and time of appearance of Microsporidiosis is unpredictable. General preventive measures of farm management are advised to prevent the disease.

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