PATHOLOGY OF DROPSY IN INDIAN MAJOR CARPS

The intensive aquaculture system has brought with it the spectre of profit-limiting diseases. Animals are reared in high density, predisposing them to diseases due to pathogenic organisms, often a direct consequence of stressors that are inherent in this type of farming. Previous workers have already reported the existence of common diseases like dropsy, haemorrhagic septicaemia, fin-rot, tail-rot and epizootic ulcerative syndrome in composite fish culture systems (Dey et al., 1982; Kumar et al., 1986; Karunasagar et al., 1989). Dropsy is an abnormal accumulation of fluid in the body of an animal which may either affect the whole body or be localised in some organs or tissues (Gopalakrishnan, 1961). After the first report of dropsy by Schaperclaus (1930), lots of investigations have been carried out in India (Gopalakrishnan, 1961; Kumar et al., 1986). Gopalakrishnan (1961) found only Aeromonas sp. as causal agent whereas Kumar et al. (1986) found mixed infection of Aeromonas hydrophila and Myxosporidian in dropsy of carps. Dropsy of carp (called carp rubella) is also considered due to viral etiology (Schaperclaus, 1965; Fijan et al., 1971). The various forms (latent, ascitic, subacute and chronic) complicate the diagnosis of disease (Gaines and Rogers, 1975).

During routine examination of diseased specimens received at the laboratory, twenty cases of dropsy (ascitic form) were registered during May to October, 1997. The samples received from the local area farmers had complain of sporadic mortality in the carp population in spite of regular feeding. The samples were examined for gross pathological lesions. Swabs were collected with aseptic measures from the abdominal fluid, kidney, liver and heart for bacterial isolation and identification using Shotts and Bullock’s procedure (1975). Squash preparations of gill and kidney were made for parasitological examinations. Subsequently, the tissues were preserved in 10% neutral buffered formalin and were processed for detail histopathological study. The pathological alterations observed have been dealt with here under.

In most of the cases, Aeromonas hydrophila were isolated from the materials collected. In few instances, the squash preparations revealed for the presence of myxosporidian cysts too.

On opening the specimens, the abdominal cavities were found to be filled with straw coloured, slightly gelatinous fluid. The liver was yellow and mottled; the heart was enlarged and the kidneys were flabby. The stomach was empty in most cases. Kumar et al. (1986) noticed superficial ulcerations, haemorrhages on body surfaces and collapsed posterior bladder along with other edematous changes in dropsy affected C. catla. Gopalakrishnan (1961) was reported both ascitic and ulcerative form of dropsy in carps.

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On microscopy, the heart revealed pericarditis, as well as myocarditis along with extensive mononuclear infiltrations, degenerative changes and loss of striations of cardiac muscle (Fig. 1). Kidney tissue showed presence of various stages of encapsulated myxosporidian cysts compressing haemopoietic tissues in few cases along with degeneration and necrosis of tubular epithelium (Fig. 2). Mild congestion of central vein and portal vessels and vasa vasorum of hepatic arteries, few mononuclear cell reaction around portal veins along with necrobiotic changes in the hepatocytes were noticed in the liver. There was dearrangement of hepatic cords, and individualization, swelling and vacuolization of hepatocytes (Fig. 3). Kumar et al. (1986) also found mixed infection of *Aeromonas hydrophila* and Myxosporidia in dropsy cases of Catla. However, they noticed excessive pigment accumulation in liver parenchyma, haemorrhages in kidney and damage to tubules and epithelial cell proliferations of posterior chamber of swim bladder. There was extensive damage to the gill lamella. The base of the gill filament in the present
cases were edematous with extensive plasma cell reactions (PAS-negative) in edematous space (Fig. 4). The typical plasma cell reaction may be suggestive of viral etiology.

Fig 3. Necrobiotic changes, swelling, vacuolization and individualization of hepatocytes, and derrangement of hepatic cords (H&E x 400).

Fig 4. Extensive damage to gill lamella and plasma cell reaction (−) in the edematous space at the base (H&E x 400).

However, the reactions seen in heart and liver along with isolation of *A. hydrophila* confirmed the involvement of bacteria. The presence of bacteria and parasitic cysts might be playing roles as secondary invaders. *A. hydrophila* in these cases might have stressed the fish to predispose them to be infected with Myxosporidia which is enzootic in this region (Mishra et al., 1982), as indicated from the major pathological alterations.

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