

A Histopathological Study on a Natural Case of Broken-Back Syndrome of the Channel Catfish

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Broken-back syndrome occurred among the channel catfish, *Ictalurus punctatus* at a low incidence in a commercial catfish pond in the United States in 1982. One of the fish suffering from broken-back syndrome was histopathologically studied. The fish showed kyphosis of the posterior abdominal vertebrae accompanied with hemorrhage. In histopathological examinations, the fractured vertebrae were found to be highly osteoporotic, degenerated and accompanied with proliferation of chondrocytes. Its periosteum was composed of thin and partly hyalinized collagen fibers, and proliferated chondrocytes, indicating impairment of collagen synthesis. The systemic connective tissues were consisted by defective collagen fibers. The pancreas, splenic pulps and hematopoietic tissue showed numerical atrophy. These histopathological signs resembled those of broken-back syndrome that was experimentally evoked with ascorbic acid-free diets in channel catfish.

Key words : Broken-back syndrome, channel catfish

Broken-back syndrome of channel catfish, *Ictalurus punctatus* (RAPINESQUE) was characterized by spinal deformity due to vertebral fractures. This disease is regarded as a nutritional disease associated with ascorbic acid (vitamin C) deficiency (LOVELL 1973, WILSON *et al.* 1973, MEYER 1975, LIM *et al.* 1978, LOVELL *et al.* 1978). MIYAZAKI *et al.* (1985) confirmed definitive histopathological signs of broken-back syndrome in the experimentally induced scorbutic channel catfish. In recent years when commercial diets for catfish were supplemented with sufficient levels of ascorbic acid, the incidence of this disease was decreased in the United States. But this disease still occurred sometimes among pond-cultured channel catfish. In 1982, this disease occurred among pond-cultured channel catfish in the state of Florida. In this study, histopathological signs of the

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fractured vertebrae and visceral organs were studied. Defective collagen synthesis indicating the primary signs of scorbutic condition was found in the crushed vertebrae and in the systemic connective tissues of this case.

Materials and Methods

One 2-year old channel catfish (body length, about 30cm) suffering from broken-back syndrome was caught in a commercial catfish pond when this disease occurred at a low incidence in the state of Florida in 1982. In this case, the condition of the diet, fish-population and pond environment were not investigated. The fractured vertebrae and visceral organs were fixed in 15% formalin solution and the vertebrae were decalcified using Prank-Ruichilo's method. These fixed tissues and organs were paraffin-thin sectioned and stained with Gill's hematoxylin and eosin (H-E), PAS reaction, Azan, toluidin blue (pH 2.5 and 7.0), alcian blue (pH 2.5) and Ziehl-Neelsen's method.

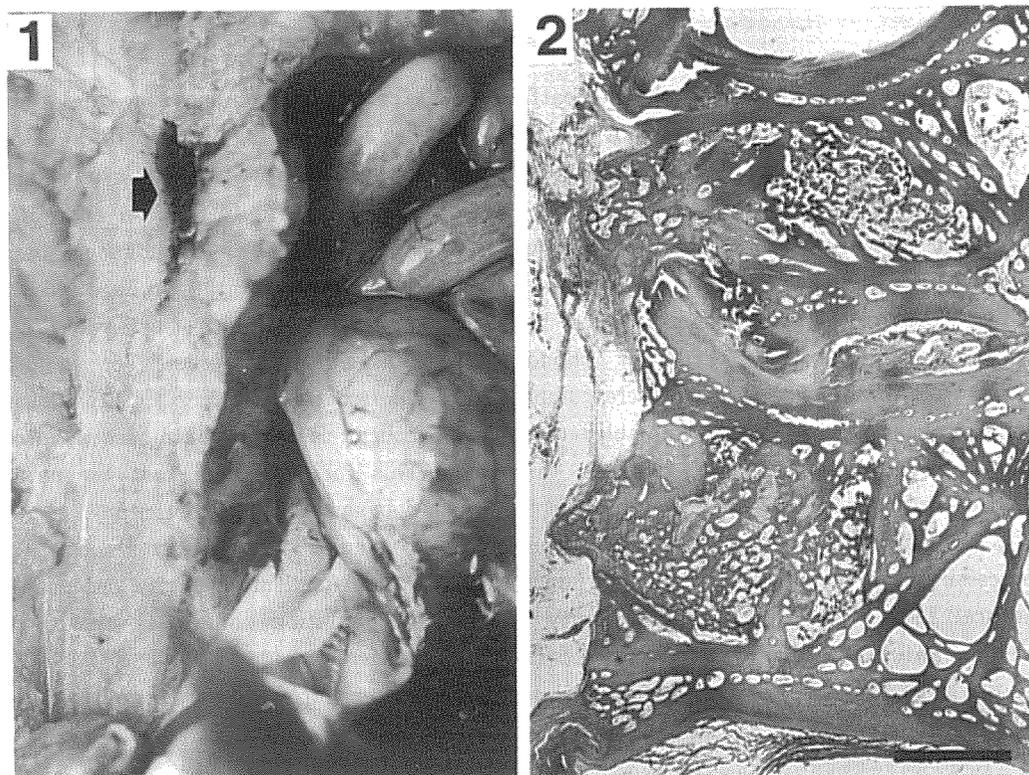
Results

External and dissected views

The diseased fish displayed kyphosis at the posterior area of the trunk. Dissection of the deformed area revealed old hemorrhage with dark brown color within the neural canal of the fractured abdominal vertebrae (**Fig. 1**). Visceral organs did not show any particular change.

Histopathological signs

In the kyphotic area, two abdominal vertebrae were fractured, followed by vertical dislocation between the adjoining vertebrae (**Fig. 2**). Those vertebrae had undergone extensive osteoporosis and degeneration of collagen bone matrix, involving overall centra, arches and spines (**Plate I - 1**). The degenerated bone matrix was highly hyalinized, obviously decreased in the glycosaminoglycan and partially replaced by chondrocytes. The interior of the osteoporotic vertebrae included thin collagen fibers, chondrocytes and dense masses composed of osteocytes and hyalinized matter. The periosteum of damaged vertebrae was deformed, and composed of thin or hyalinized collagen fibers and proliferated chondrocytes (**Plate I - 2**). The intervertebral discus was also deformed, hyalinized and showed proliferation of chondrocytes. Periosteum of the spines and arches were consisted by defective thin collagen fibers. Many macrophages infiltrated and phagocytized erythrocytes at the hemorrhagic areas around the fractured vertebrae and inside the neural canal. The spinal cord and cerebrospinal ganglia were degenerated at the area involved by the vertebral fracture. The lateral musculature was atrophic and slightly necrotic at areas near the fractured vertebrae. The myosepta were extensively composed of thin collagen fibers.



Explanation of Figures

- Fig. 1. A dissected view of channel catfish suffering from broken-back syndrome. Abdominal vertebrae showed kyphosis and hemorrhage (arrow).
- Fig. 2. A low power view of fractured vertebrae. Vertebrae had undergone extensive osteoporosis and hyalinization. H-E, X10, Scale bar : 1 mm.

The submucosa of the stomach was composed of fragmented thin collagen fibers and abnormally increased amorphous matter stained pale blue with Azan stain. The spleen showed sheathed arteries composed of the same amorphous matter as found in the stomach, a decrease of lymphoid elements in the pulps, thickened trabeculae and the capsule including fragmented thin collagen fibers (Plate I-3). The liver showed atrophic hepatic cells. The walls of intrahepatic blood vessels were composed of thin collagen fibers, including decreased numbers of pancreatic cells and increased numbers of ceroid-laden macrophages (Plate I-4). In the kidney, nephrons were nearly normal and the

hematopoietic tissue showed numerical atrophy. Chondrocytes of cartilaginous shafts of gill filaments were partially degenerated without deformity.

Discussion

Broken-back syndrome of channel catfish was experimentally induced by the feeding on ascorbic acid-deficient diets (LOVELL 1973, WILSON *et al.* 1973, MEYER 1975, LIM *et al.* 1978, LOVELL *et al.* 1978). The histopathological signs of broken-back syndrome of the scorbutic channel catfish were reported by MIYAZAKI *et al.* (1985). In those fish, fractured vertebrae showed one or two signs of osteoporosis, abnormally advanced ossification and dysplasia which were due to impairment of collagen synthesis. Especially the osteoporotic vertebrae had thin collagen fibers in their interior. Their periosteal and intervertebral discs were replaced by chondrocytes and chondroid cells. The systemic connective tissues of those fish were composed of impaired collagen fibers. Such histopathological signs very closely resembled those of a natural case of broken-back syndrome of channel catfish found in a pond in this study. This fact indicated that the natural case of broken-back syndrome might have been caused by ascorbic acid-deficiency.

In commercial diets for catfish, ascorbic acid is readily decreased in amount after processing and storage. So in catfish ponds, ascorbic acid-supplement partly depended on natural feeds. Therefore, a high density of catfish population sometimes evokes lack of the natural feeds, resulting in ascorbic acid-deficiency and broken-back syndrome (LOVELL *et al.* 1978). These facts indicate that pond-cultured catfish included factors which made susceptible to ascorbic acid-deficiency.

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Explanation of Plate I

- Fig. 1. A detail of fractured vertebrae. Highly osteoporotic vertebrae were crushed. H-E, X16, Scale bar : 500 μ .
- Fig. 2. A detail of fractured vertebra. Its interior included thin fibers. The periosteum was composed of thin fibers, hyalinized fibers (H) and proliferated chondrocytes (C). H-E, X16, Scale bar : 500 μ .
- Fig. 3. Spleen showed sheathed arteries (A) composed of amorphous matter, numerically atrophic pulp around the arteries and thickened trabeculae (T). Azan, X320, Scale bar : 50 μ .
- Fig. 4. Liver showed atrophic hepatocytes. The blood vessel (V) was composed of thin collagen fibers and included masses of ceroid-laden macrophages (M). X160, Scale bar : 100 μ .

