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A case study of gas bubble disease in Black Sea Bream (*Spondylosoma cantharus*)

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Gas bubble disease is a complex pathophysiological change common to a wide range of freshwater, migratory and marine fish species. The basic mechanism of pathogenesis is based upon the embolic effect of gas bubbles that "wander" through the vascular system and damage practically any tissue that absorbs them.

In most cases, the first changes develop in the epithelium of the gill, resulting in hyperplasia and epithelial desquamation, and necrosis in the final stage. Changes reversible at certain stages are triggers for hypoxic and anoxic conditions of the organism and, in most cases, the causa mortis.

Except for the easily visible gas bubbles in the dermis, features of the gas bubble disease are behavioural changes induced by the absorption and embolic trauma of gas bubbles in the brain tissue. Gross pathomorphological lesions are mainly restricted to lesions induced by the low feeding capability of affected fish.

This study deals with the causes and triggers of the gas bubble disease, its pathogenesis, and pathophysiological and gross pathomorphological changes in black sea bream (Spondylosoma cantharus) *caught in the wild and held in a concrete tank.*

Morbidity reached 50% during two months, and mortality reached 37.5%.

Key words: gas bubble disease, black sea bream (*Spondylosoma cantharus*), cultural conditions

INTRODUCTION

Gas bubble disease is a syndrome comprised of lesions in various tissues induced by an excess of dissolved gases in the water. The disease occurs in both vertebrate and invertebrate aquatic species. Usually the most problematic gases are oxygen and nitrogen, even though their solubility depends on different environmental factors - temperature and hydrostatic pressure. Once, it was presumed that the disease was primarily caused by supersaturation of nitrogen, but WEITHAMP & KATZ (1980) concluded that the main aetiology should be sought in total dissolved gas pressure. Nevertheless, it is not unusual that the gas bubble disease emerges in conditions of undersaturation of oxygen (SAEED & ALTHOBAITI, 1997).

The oversaturation of gases in water has multiple causes including elevated rates of photosynthetic activity (WEITHAMP & KATZ, 1980), air injection or entrapment of air in the water (dysfunctional pipe valves, pipe fittings, semi-submerged intakes; COLT, 1986), hydroelectric

stations (HARVEY & COOPER, 1962), and even helicopter transport of fry because of the rapid decompression resulting from the change in altitude (HAUCK, 1986).

Another problem in inland Dalmatia, where the soil is karstic, is ground water that passes through fissures in the soil, picking up excess nitrogen on the way, even though such water has many positive biochemical characteristics (such as its hardness, pH, mineral level, oxygen saturation).

Because of its multicausal aethiology, the range of hosts for the gas bubble disease is very wide, including mostly salmonids, such as the Atlantic croacker (CHAMBERLAIN *et al.*, 1980), chinook salmon (*Oncorhynchus tshawytscha*), steelhead trout (DAWLEY & EBEL, 1975), cutthroat trout (*Oncorhynchus clarki*), rainbow trout (*Oncorhynchus mykiss*; DOULOS & KINDSCHI, 1990), lake trout (*Salvelinus namycush*; HOFFERT *et al.*, 1971), eye (*Onchorhynchus mykiss*; FAIRBANKS *et al.*, 1969), and sockeye salmon (*Oncorhynchus nerka*; NEBEKER *et al.*, 1976). It also occurs in striped bass (*Morone saxatilis*; CORNACCHIA & COLT, 1984), channel catfish (*Ictalurus punctatus*; JONES & LEWIS, 1976), squawfish (MEEKIN & TURNER, 1974) and the mollusc, red abalone (*Haliotis rufescens*; ELSTON, 1983).

Morbidity and mortality depend on the degree of saturation, the total dissolved gas pressure, the duration of the exposure, zootechnical methods and fish specifics (such as age, sex, and species). Mortality is mainly attributed to lethal exposure to the disease or secondary induced bacterial opportunistic infection and stress (SPEARE, 1998).

MATERIAL AND METHODS

Eight black sea bream (*Spondylosoma cantharus*) were caught in the wild and held for two years in a cylindrical concrete tank of 10 m^3 with a constant water flow and a natural photoperiod. The fish were fed natural food.

A black sea bream showing marked signs of distress for a week died, was necropsied and gross pathomorphological changes were recorded. The diameters of bubble-like features on the fins and eyes were measured. In particular, the gills were observed. The extremities of the gills were cut, put into a saline solution, and examined for the presence of monogenetic trematodes or other parasites. After opening the abdominal cavity and gross examination, organs were prepared as wet mounts and examined for the presence of granular inflammations or myxosporidian parasites. Within two months, three more black sea bream showed the same signs and two died.

The morphometric parameters of the fish were recorded. Temperature and water flow were measured twice a day. Two water samples were collected in WINCKLER's bottles, a meter from the surface, from three sides of the pool, fixed with $MnCl_2$ and NaOH with KJ, and put into a cooler before Na-tiosulphate titration.

RESULTS

Three black sea bream died after showing the following behavioural changes: uncoordinated swimming, side swimming, loss of equilibrium and buoyancy, whirling with spasmodic convulsions, aimless swimming and lethargy with distressed opercular movements. Food consumption ceased. Morbidity was 50% and mortality reached 37.5%. The mean total weight of the fish was 384.9 g ± 32.1 g, and the mean total length was 27.6 cm ± 3.6. After parasitological examination, only one subject had five third-stage larvae of *Anisakis* sp. in the mesentery. The other fish were negative.

The water was 86% saturated with oxygen and had a mean temperature of 23.2°C ± 0.2.

External gross pathological changes

Disseminated, transparent gas bubbles (1-10 mm in diameter; Figs. 1,2) were clearly visible, under the epidermal surface of the dorsal, pectoral and anal fins. Also noticeable was the disruption of the epidermis of the dorsal and caudal fins, with cut edges (Fig. 3). No inflammatory

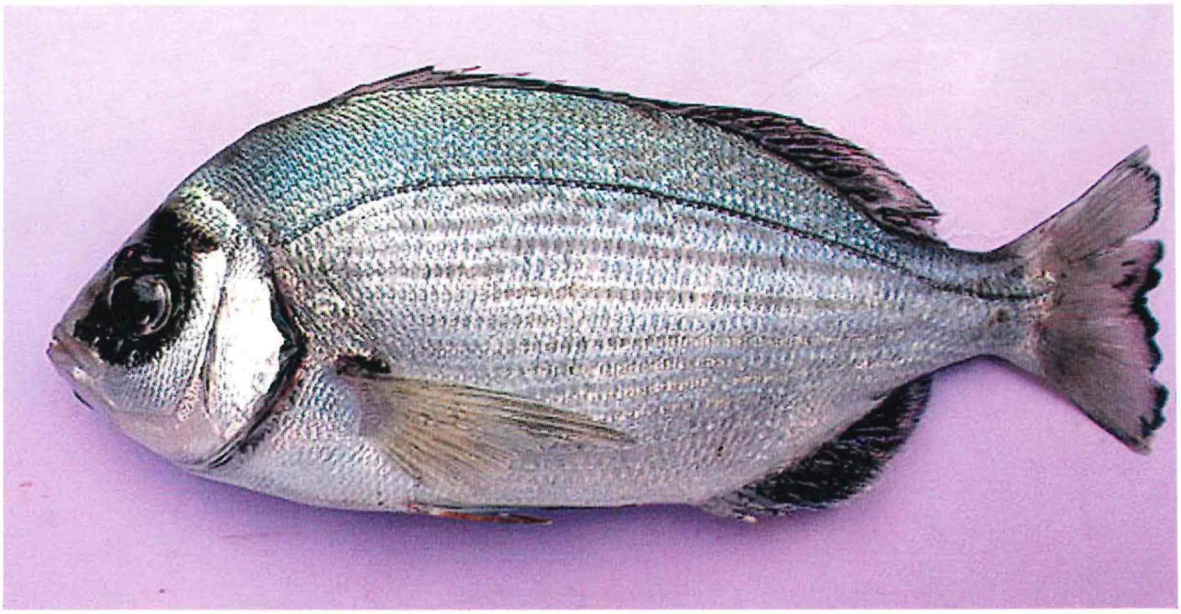

Fig. 1. Black sea bream (Spondylosoma cantharus) *showing unilateral exophthalmia without any other external signs*

Fig. 2. Gas bubbles disseminated in pectoral fin

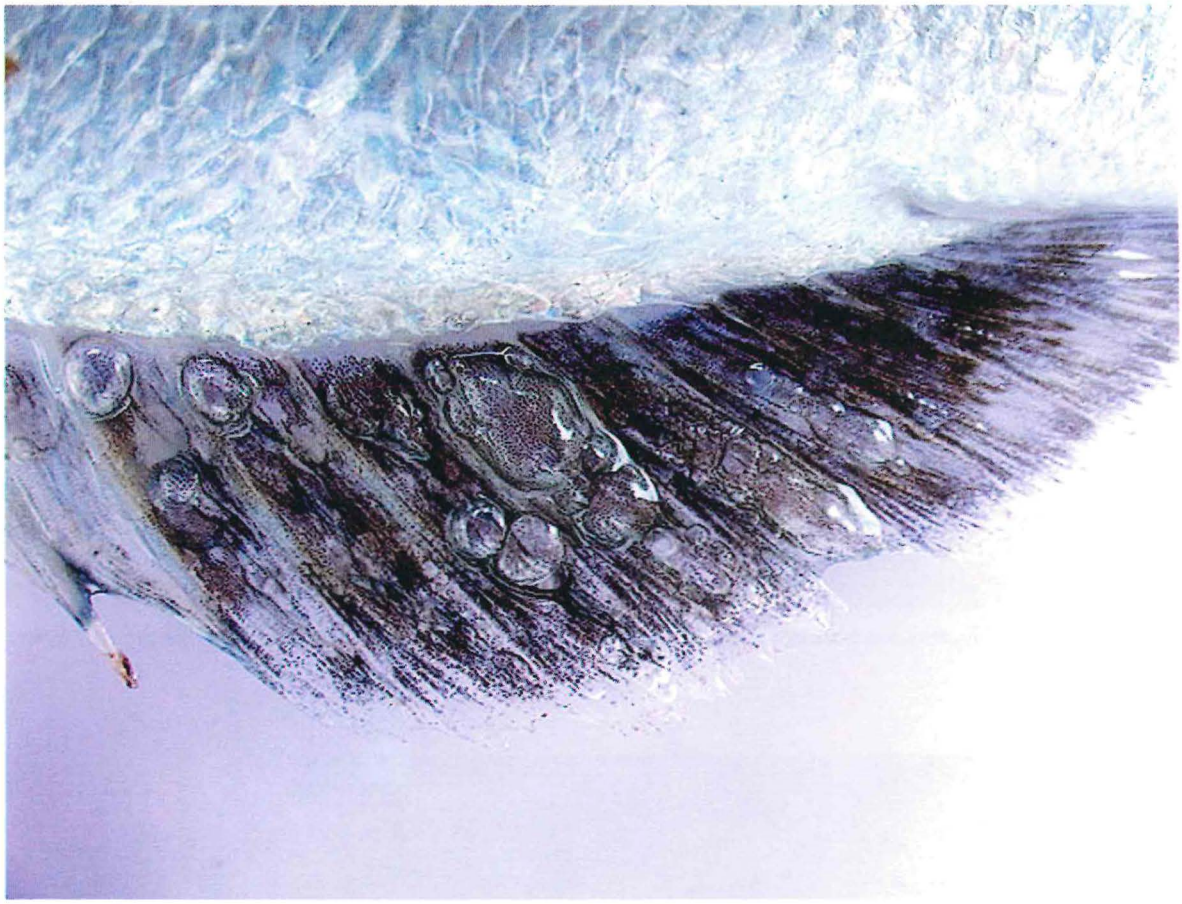

Fig. 3. Black sea bream (Spondylosoma cantharus) *showing unilateral exophtalmia and cut edges on the dorsal and caudal fins*

signs in the epidermis were noticed, except excessive production of mucus and loss of scales on the flanks.

The eye lesions showed marked exophthalmia with the equatorial anatomical axis of the eye expanded dramatically in the dorsal part (Fig. 4). Large gas bubbles developed under the cornea in the dorsal equatorial axis, 13.7 mm in diameter and motile. The orbit became elliptic and anterio-posteriorily convex. Synechia of the anterior layers of the eye with cornea developed. The cornea had a cloudy appearance with disseminated shallow erosions. The lens was cloudy with a cataract and the delamination process was in the initial phase. Periocular epidermis was clearly ramified with blood vessels.

Internal gross pathological changes

All gill arches were affected by early changes, visible in the ventral quarter of the arches (Fig. 5). Lamellae were yellowish, partially eroded, with excessive mucus production. The gills became less necrotic and more haemorrhagic moving dorsally. Under a light microscope, the wet mount of gills had stick-like, elongated gas bubbles through the afferent and efferent arterioles, and a marked thickening of the gill epithelium of the secondary lamellae.

The visceral organs showed only changes characteristic of emaciation (Fig. 6): decreased fat content in the mesentery, intestine filled with serous fluid, ascitic visceral cavity. The mesentery lacked any adipose aggregations, but had a small number of 1-2 mm (diameter) gas bubbles

Fig. 4. Exophthalmic eye with prominent dorsal distortion

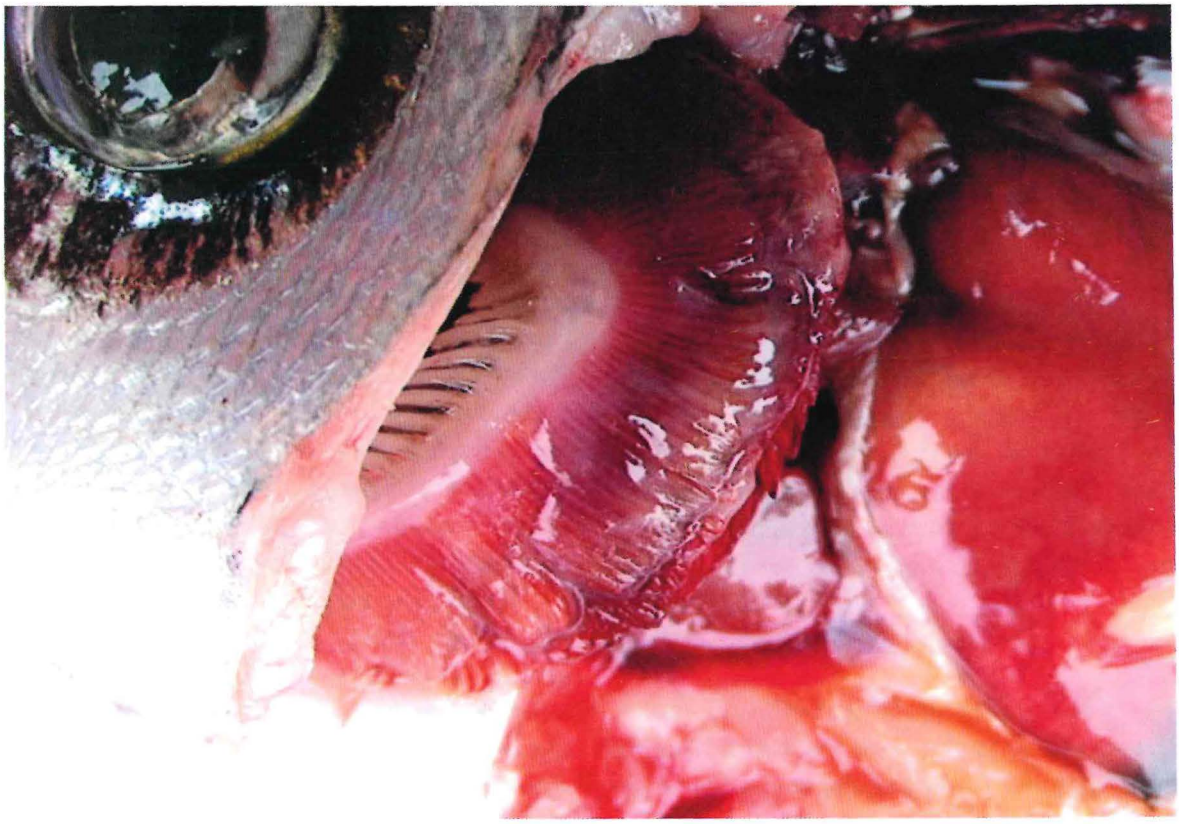

Fig. 5. Gill's ventral necrotic part and gill's haemorrhagic dorsal part affected by GBD

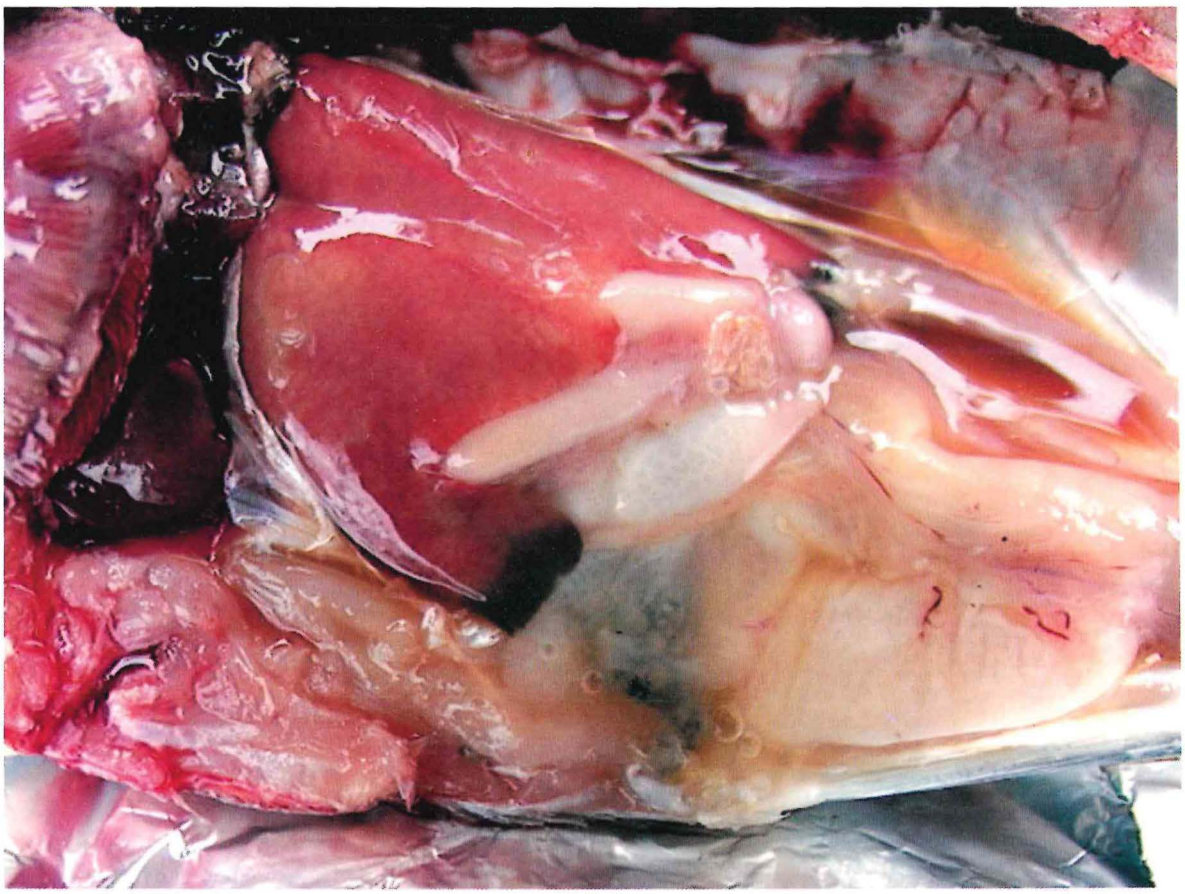

Fig. 6. Visceral organs and meteorism of intestine

in the anterior part. Sero-haemorrhagic liquid collected in the abdominal cavity. The intestinal mucosa were covered with yellowish content, probably desquamated epithelium, and the lumen was filled with a transparent serous fluid. Intestine distension and meteorism were noticed. The liver showed no change in size, shape or colour, except for *post-mortem* haemolysis in the ventro-caudal parts of the lobes. The kidney showed no change. No parasites were isolated from the diseased fish, except for five third-stage *Anisakis* sp. larvae.

DISCUSSION

Clinical signs of the gas bubble disease are related either to the acute pathophysiological event associated with vascular damage or to primary tissue destruction from "space-occupying lesions" (SPEARE, 1998). The first signs of gas bubble disease are evident after a chronic period of changed total dissolved gas pressure and mainly involve behavioural changes. As the fish remain mostly on the bottom of the tank, transparent bubbles on the fins and skin are not easily noticeable. After a period of 4-6 days, exophthalmia becomes evident and, without inspecting the whole fish, it can be mistaken for a mechanical ophthalmitis with secondary bacterial infection (KATAVIĆ *et al.*, 2000) or vitamin C deficit (MARŠIĆ-LUČIĆ *et al.*, 1992).

Morbidity and mortality from the gas bubble disease depend on the age and the fish species, in addition to the level of total gas hypersaturation. Two adult gilthead sea bream (*Sparus aurata*) and an adult pink dentex (*Dentex gibbosus*), held in the same tank with the black sea bream, showed no signs of gas

bubble disease, suggesting that species and age resistance attributed, perhaps, to oxygen needs and rate of absorption.

SAEED & ALTHOBAITI (1997) recorded 50% morbidity and 30% mortality in a subadult population of saltwater tilapia (*Oreochromis spilurus*), while in a second outbreak morbidity was 25% and mortality only 5%. In adult tilapia, both morbidity and mortality were lower (40% and 25%, respectively) while in three-month grouper (*Epinephelus fuscoguttatus*), mortality was 10%. In our study, morbidity and mortality were in accordance with the range observed by SAEED & ALTHOBAITI (1997), however, the disease was chronic for two months and no acute outbreaks occurred.

Presumably, the cause of the disease was the slightly-elevated, long-lasting and persistent total dissolved gas pressure that affected only the most sensitive fish species in the tank, and not the others. As was demonstrated in other studies (WILLIAMS *et al.*, 1995; SAEED & ALTHOBAITI, 1997), the oxygen saturation was within the optimal range.

All pathomorphological changes were induced by occlusions of arterioles in various tissues. Most of the prominent and life-threatening lesions were located in the gills, with almost one-third of total gill tissue necrotised due the embolic effect of gas bubbles in the vascular system of the gill. Some authors noticed heavy infection with monogenetic trematodes (SAEED & ALTHOBAITI, 1997) in the epithelium of gills that were already devastated by gas emboli, however, no such changes were recorded in our study, resulting in a much slower progression of the disease.

Hyperplasia and desquamation of the epithelium resulted in a lower respiration rate with initial bradhipnea, then tachipnea, leading to death by asphyxia. Gill lesions were the first changes induced by the high total dissolved gas pressure, evident from the divers stage of the pathologic process - from the early stage of haemorrhages to later stage of necrosis - that were lethal for the fish in our case. The process progressed in accordance with the rate of gill surface affected; the larger the affected surface, the more acute and faster was the mortality.

Ocular lesions alone are benign and in most cases repairable in subjects with good health. However, they eventually induce, as in our case of reduced feeding capability, anorexia and progressive weight loss. In conditions of reduced oxygen consumption, impaired health caused by emaciation, linked with a weakened immunological response, can easily lead to secondary parasitological or bacterial infection.

The internal pathomorphological picture reflects the poor food consumption and shows marked emaciation, a situation also observed by other authors (SPEARE, 1998). Microscopic lesions caused by gas emboli are noticeable only by means of histological examinations, but are reflected in gross lesions in the gills.

The chronic duration of the process was evident by the presence of bubbles in the fat tissue of the mesentery. Because poorly vasculated fat tissue has the slowest rate of uptake of gas from circulation, it can be used as an indicator of the longevity of the process; once absorbed, the gas stays in that tissue longer. According to STRAUSS (1979), highly perfuse brain tissue is a good example of a fast gas-absorbing and gas-clearing tissue. This is in accordance with the onset of the first behavioural changes, resulting from bubbles present in the brain and their damage to the tissue. This change is parallel with initial exophthalmia. When hypersaturation ceases, the brain is the first tissue to lose gas bubbles, so even harsh neurological symptoms can disappear without consequences.

Because of its more or less simple process and mechanism for inducing pathologies, the gas bubble disease is a zootechnological disease resolvable by prompt and careful technical correction of the problem (dysfunctional pipe valves, pipe fittings, semisubmerged intakes). The gas bubble disease indeed can be responsible for serious economic losses (low weights, mortalities) in aquaculture facilities and therefore must not be neglected or underestimated.

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Studija slučaja bolesti mjehurića plina (BMP) u kantara (*Spondylosoma cantharus*)

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SAŽETAK

Bolest mjehurića plina (BMP), kao kompleksna patofiziološka promjena, susreće se u širokom rasponu slatkovodnih, migratornih i morskih ribljih vrsta. Osnovni mehanizam patogeneze se osniva na emboličkom djelovanju "lutajućih" mjehurića plina kroz krvožilni sustav i njihovoj sposobnosti oštećenja praktički bilo kojeg tkiva koje ih apsorbira.

U većini slučajeva, međutim, prve promjene razvijaju se u škržnom epitelu, rezultirajući hiperplazijom i epitelnom deskvamacijom, te nekrozom kao krajnjim stadijem. Ove promjene, iako reverzibilne do stanovite razine, otponac su hipoksičnim i anoksičnim stanjima organizma i u većini slučajeva, jesu *causa mortis*.

Osim lako uočljivih mjehurića plina u dermisu, zanimljiva karakteristika BMP su promjene ponašanja, inducirane apsorpcijom i emboličkom traumom mjehurića plina u moždanom tkivu. Ostale patomorfološke lezije su većinom ograničene na lezije inducirane smanjenom sposobnošću hranjenja bolesne ribe.

Ova studija se bavi uzrocima i otponcima BMP, njenom patogenezom, patofiziologijom i patomorfološkim promjenama u kantara (*Spondylosoma cantharus*), ulovljenih u divljini i držanih u betonskom bazenu.

Morbiditet je dosegao 50 % tijekom dva mjeseca, s mortalitetom od 37,5 % zahvaćene ribe.

Ključne riječi: bolest mjehurića plina (BMP), kantar (*Spondylosoma cantharus*), uzgojni uvjeti