Ocular trematodiasis of gizzard shad

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Introduction

Gizzard shad (*Dorosoma cepedianum*) are often referred to as “trash fish” or buffer prey, and may be introduced into catfish ponds to reduce predation of the catfish by piscivorous birds [1]. Although these fish are not harvested for consumption, they potentially play an important role not only to protect the catfish from predation, but also as a sentinel for disease within the catfish pond. Being able to diagnose diseases early can help expedite treatment.

History and Presentation

Five freshly dead gizzard shad presented on April 27th, 2016 to the Aquatic Research and Diagnostic Laboratory in Stoneville, MS from a farm located in the Delta of Mississippi (Humphreys County). These shad were collected from a channel catfish pond. The farmer noticed that the shad started dying off, and his catfish had become inappetant. The farmer had also recently noticed an algal bloom in the pond. The farmer is concerned about the shad dying, and that something infectious may be affecting his catfish. There is a history of having atypical *Aeromonas hydrophila* in that pond before. He did not bring in any catfish for necropsy or water sample for water quality testing.

Diagnostic Approach/Considerations

Upon presentation, the Gizzard Shad were freshly dead and in good condition. They measured approximately 12cm in length. Two of the shad had areas of hemorrhage on the cornea of the eye, which was likely secondary to the ice they arrived on. White parasites were noted within the anterior and posterior chamber of each eye. No other abnormal gross findings were noted. A gill clip and skin scrape were performed and revealed *Trichodin*a. *Trichodina* is a protozoan ectoparasite that inhabits the gills and skin of fish. In small numbers, their presence means little.

If fish have a heavier infestation with these ectoparasites, it is usually indicative that the fish are in poor health due to disease, poor condition, stress or overstocking. [1,2].

Samples of the brain and posterior kidney were collected aseptically for culture and sensitivity. Samples were plated on blood agar and Mueller Hinton agar with neomycin. No significant colonies were obtained from brain culture, only contaminants which were apparent by the various size, morphology and color of the colonies. Culture of the kidney was more successful and a BBL (enteric/nonfermenter) was performed, identifying the agent as *Aeromonas hydrophila*. Sensitivity results reported that the *A. hydrophila* isolated from the kidney was most sensitive to Aquaflor.

The white parasites within the eyes were removed and examined macroscopically with a dissection microscope. They were grossly determined to be trematodes, and due to the morphology it was suspected that they were *A. ostrowskiae*. The trematodes were collected and preserved in ethyl alcohol. DNA isolation was then performed using the Puregene system. A PCR reaction was then performed using the PCoxF/PCoxR primers to amplify the *c* oxidase I (COI) gene. The amplification products were then purified using the QIAquickGel Extraction Kit, and the purified products were sent for sequencing to Eurofins. The sequences were then applied in SeqMan system to edit and align them. The DNA sequence was found to be consistent with *Austrodiplostomum ostrowskiae*.

After culturing *A. hydrophila* from the kidney, a water sample was obtained from the farmer and a water PCR was performed, confirming the presence of atypical *Aeromonas hydrophila*. Given the history that the farmer has had a problem with atypical *A. hydrophila* in the past, this was no surprise. These results must consider the findings on necropsy, as presence of the bacterium alone does not confirm an active infection. No signs of a bacterial septicemia

were noted on necropsy of the gizzard shad, and the farmer had not reported an increase in death rate of his channel catfish. Ideally a necropsy of catfish from the same pond would have been performed. A PCR for algal toxins or a bioassay of the pond water with sentinel fish may have be performed to investigate if algal toxins were contributing to the decreased appetite of his catfish.

Pathophysiology

Austrodiplostomum ostrowskiae is a digenean trematode that affects multiple species of fish, including catfish and gizzard shad [5]. Digenean trematodes that infect fish usually have an avian, mammalian or fish definitive host, an mollusk first intermediate host and sometimes a second fish intermediate host [1,2,4]. Often digenean trematodes affect the alimentary tract of fish, but can also affect ocular structures, the circulatory system and other organs [2]. Typically these trematodes do not cause a significant amount of morbidity and mortality [1,2].

In the definitive host, adults produce eggs which are passed in the feces into the environment. The definitive host for *A. ostrowskiea* is the double-crested cormorant (*Nannopterum auritus*) [5]. The eggs then hatch into miracidium which will infect the first intermediate host, a mollusk [1,2,4]. The first intermediate host of *A. ostrowskiae* is the snail *Biomphalaria havanensis* [5]. In the mollusk, the miracidium undergoes asexual reproduction to produce cercaria, which then leave the mollusk and infect the second intermediate host. This is accomplished by traversing the tissues of that host [1,2,4]. Often the migration of the cercaria through tissues is what causes the most pathology [2]. Once inside the intermediate host, the cercaria will undergo mature into metacercaria [1,2,4]. Metacercaria have all of the structures an adult digenean trematode has, but they lack a mature reproductive tract [2]. Although there have been reports of the metacercaria of *A. ostrowskiea* affecting more 20 species of fish, they have

not been morphologically confirmed to be *A ostrowskiae*, except in channel catfish and gizzard shad [5]. Once inside the second intermediate host, the metecercaria can exist for one or more seasons, depending on the species of digenean trematode [4]. The life cycle is then completed when the second intermediate host is consumed by the definitive host [1,2,4].

In the study by Rosser, which connected the definitive host, the mollusk intermediate host and the fish intermediate host in the lifecycle of *A. ostrowskiae*, they reported ocular changes secondary to presence of the trematodes within the eye. One such change included retinal degeneration. It was unclear if the degeneration was due to the fixative used or the trematode itself. More notably, they also noticed lenticular changes such as partial detachment of the capsule of the lens, cataracts of the lens and formation of Morgagnian globules within the lens [5]. The other common ocular digenean of catfish and gizzard shad (amongst other fish species) is *Diplostomum spathaceum*. The metacercaria of this fluke develop in the lens capsule causing significant ocular pathology, and has been studied in much more detail than *A. ostrowskiae*. Studies have been performed to look at the effect of *D. spathaceum* on production, and have found that feed efficiency, rate of growth and the ability to escape from predators may all be affected. It was also found that young and small fish affected with heavy loads of the cercaria of *D. spathaceum* acutely may cause significant morbidity and mortality due to the migration of the cercaria through tissues [1, 2,4,5].

There is one study from 1983 that found despite having blindness secondary to ocular trematodiasis, catfish were still able to grow at an acceptable rate, have acceptable food efficiency and survive. That same study also found that the ocular trematode did not reduce food quality for human consumption. The ability of catfish to thrive despite being infected is contributed to the sense organs catfish use to apprehend food. Catfish primarily use olfaction and

gustation to find food. Catfish have the majority of their taste buds in their mouth, and can also be found diffusely throughout their epidermis and in their barbels [1,5,6].

Another issue one might consider is the ability to have reproductive cyclicity if fish are blind secondary to ocular trematodiasis. Although there are no studies in gizzard shad that may address this issue, it is more important that we understand how it would affect the catfish. A study published in 1984 found that enucleation of the eye had no significant effect on cyclicity of female catfish. It can therefore be concluded that factors such as temperature (and thus time of year) and exposure to males plays a more significant role in the cyclicity of the catfish [1, 3].

Treatment and Management

There is no single treatment that is effective in controlling ocular and nonocular diplostomiasis (trematodiasis) in catfish. The lifecycle of diplosomids such as *A. ostrowskiae* and *D. spathaceums* are well propagated in the culture of catfish as all of the hosts are present in abundance. Catfish are cultured outdoors in ponds, leaving them exposed to piscivorous birds such as the double-crested cormorant. There are limited ways to manage the presence of piscivorous birds (such as the double-crested cormorant) but they may include extermination, introduction of a buffer prey such as gizzard shad to reduce predation on the catfish, or creating a physical barrier. Unfortunately, creating physical barriers between the fish and the birds requires maintenance and may not always economically fit. One should also note that complete physical separation is likely not attainable [1].

Another management step includes control of the other intermediate host, the snail. Unfortunately, the catfish ponds are an ideal environment for snails. Ways to manage the snail population include introduction of snail-eating fish and pond-side or direct pond treatment with copper sulfate or slaked lime (calcium hydroxide). These treatments are most effective if applied

overnight when snail activity is at its highest. Often multiple treatments are needed throughout a season. The snail population will never be completely exterminated, just kept at a more manageable level [1,2].

Case Outcome

A thorough case follow-up is not available. It is unlikely that the ocular trematodiasis was contributing to the decreased appetite of the catfish and death of the shad, and therefore treatment of the snails was not be recommended at that time until other possibilities were ruled out. Treatment of the *A. hydrophila* may be employed if the farmer noticed an increase in death rate in his pond, and the lesions were consistent with a bacterial septicemia and the organism was cultured from the catfish. Necropsy of the catfish, management practices and water quality should be investigated before prescribing treatment. A bioassay of the pond water to test for algal toxins could also be performed.

References

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