



Epizootic Ulcerative Syndrome (EUS): An Aquatic Ecosystem-Threatening Disease

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Abstract

The name “epizootic ulcerative syndrome” (EUS) was developed to characterize a severe cutaneous ulcerative epizootic disorder that affects a variety of wild and farmed freshwater and estuary finfish species. EUS-infected fish is characterized by the presence of single or multiple lesions with acute dermatitis, hyperemia, and edema leading to deep ulcers. It has been observed that EUS has an impact on more than 100 fish species. *Aphanomyces invadans*, a pathogenic fungus, has been associated with the transmission of EUS, however, the agent hasn't yet been demonstrated to enter and cause the disease by itself. Environmental variables such as heavy rainfall, bad water quality, and low pH have been linked to the development of EUS. Ash, turmeric, neem branches (*Azadirachta indica*), dried banana leaves, CIFAX, and other therapies have been tried with varying degrees of success for preventative treatments of the EUS-infected fish.

Keywords: *Aphanomyces invadans*, Aquatic Animal Health Management, CIFAX, Epizootic Ulcerative Syndrome (EUS)

Introduction

Epizootic ulcerative syndrome (EUS) is the most destructive disease amongst wild and cultured freshwater and estuarine finfish. In 1971, the disease was initially identified in Japan's farmed freshwater ayu (*Plecoglossus altivelis*). The disease spread through several Asian countries. Till now EUS has been recorded from 26 countries on 4 continents. The spread of the disease in countries of the Asia-Pacific region and Africa has led to dangerous consequences for the fish resources and livelihood of marginal fish farmers. In May 1988, India had the first outbreak of Epizootic ulcerative syndrome in the Asia Pacific region and since that time, different states in India have seen the disease gradually spread among freshwater and brackish water fishes. Hardly any fish disease in India has been as virulent and dangerous as the recent outbreak of Epizootic Ulcerative Syndrome. The EUS has affected natural fish populations of open water resources by transcending the confines of culture ponds. The alarming rate of infections with repulsive lesions and the trail of destruction left behind by the epizootic have already robbed thousands of fish farmers and riparian fisherfolk

of their daily bread in the different affected states of India and have become a matter of grave concern for the fishery scientists, administrators, and fishermen folk.

Due to the noticeable clinical symptoms in the affected fish, the disease was previously named differently in different geographic areas; for example, mycotic granulomatosis (MG) in Japan, red spot disease (RSD) in Australia, and ulcerative mycosis (UM) in the USA. In Indonesia, the same disease was called infectious dropsy or hemorrhagic septicemia, whereas in Malaysia the necrotic ulceration was called 'Webak Kudes. It is now known to all that the disease is the same as EUS. EUS-infected fish is characterized by the presence of single or multiple lesions of varying shapes with acute dermatitis, hyperemia, and edema leading to the development of typically large shallow or deep ulcers with a hemorrhagic necrotic base anywhere in the body with muscular inflammation and multiple granulomas. EUS is one of the OIE-listed fish diseases. The disease has been found to infect more than 100 species of fish and cause huge economic losses. Fish with the EUS infection harbored several diseases, including viruses.

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The Global Spread of EUS

Following the Mycotic Granulomatosis outbreak in Japan's farmed freshwater ayu (*P. altivelis*) in 1971, and the Red Spot Disease outbreak in estuarine fish, grey mullet (*Mugil cephalus*) in eastern Australia in 1972, there has been evidence of the slow westward spread of a disease across Asia characterized by cutaneous ulceration and causing significant mortalities in several freshwaters and estuarine fish species. In 1979-80, peninsular Malaysia experienced the first verified report of the symptoms of severe ulcerative disease. In the 1970s, EUS gradually spread across southeast Asian nations to South Asia via Singapore in 1977, Thailand in 1981, Myanmar, the Lao PDR, and Cambodia in 1984, the Philippines in 1985, Sri Lanka in 1987, Bangladesh in 1988, Nepal, Bhutan, and north India in 1989, Indonesia in 1990, and peninsular India in 1991. Afterward, it expanded to Pakistan in 1996, the USA in 1997, and southern African nations Botswana, Namibia, and Zambia in 2007. A total of 24 nations across four continents - North America, southern Africa, Asia, and Australia - have now recorded cases of EUS, which can infect finfish in the wild as well as aquaculture systems.

Spread of EUS in India

The EUS disease first occurred in northeast regions of India such as Tripura, Assam, Meghalaya, and West Bengal in May 1988 and subsequently, it spread to other states of India as Bihar, Orissa, Uttar Pradesh, Madhya Pradesh, Maharashtra, Andhra Pradesh, Tamil Nadu, Kerala, Karnataka, Haryana, and Rajasthan.

Fish Species Affected Globally

It has been observed that EUS has an impact on more than 100 fish species. Nearly all freshwater and brackish water fish species are susceptible to infection, even though certain species such as striped mullet (*M. cephalus*), *Channa* spp. (Snakeheads), Indian major carp (*Catla catla*, *Labeo rohita*, and *Cirrhinus mrigala*) and some other significant food species are particularly susceptible. While some species are reported to be resistant to EUS such as common carp (*Cyprinus carpio*), Nile tilapia (*Oreochromis niloticus*), milkfish (*Chanos chanos*), catfish (*Parasilurus asotus*), loach (*Misgurnus anguillicaudatus*), Japanese eel (*Anguilla japonica*) and European eel (*Anguilla anguilla*). Previous investigations have revealed that the majority of the species impacted by natural outbreaks are either bottom dwellers or have air-breathing organs.

Fish Species Affected by EUS in India

Thirty freshwater and brackish water fish species have been identified as being affected by EUS in India, of which four species are exotic and the rest native. The range of incidence of the disease was recorded from the different species of infected fish and different types of water bodies. It reveals that some genera of fish, such as *Channa*, *Puntius*, *Mastocembelus*, *Mystus*, *Glossogobius*, *Anabus*, *Clarias*, and *Heteropeneustes* are highly susceptible to EUS.

Etiology

It has been suggested that the prevalence of EUS-like ulcerative disease outbreaks is related to a varied array of infections and causes. Various types of viral agents have

been isolated from EUS-infected fishes across different countries. Rhabdoviruses and birnaviruses are the two viral subgroups with the greatest degree of isolation (John and George, 2012).

Various bacterial species have also been isolated from EUS-affected fish. Necrotic ulcers have been related to bacteria including *Aeromonas*, *Vibrio*, *Pseudomonas*, and *Micrococcus*, which are generally opportunistic pathogens and are assumed to be the secondary invaders killing severely ulcerated fish. *Aphanomyces*, *Saprolegnia* and *Achlya* are only a few of the fungi, metazoans, and protozoans in addition to viruses and bacteria that have regularly been identified from fish infected with the EUS. *Aphanomyces invadans*, a fungus-like organism, are regularly linked to the disease according to several types of research. Fish with EUS infection have consistently been linked to bacterial infections. Environmental variables such as heavy rainfall, bad water quality, and low pH have been linked to the development of EUS. Mulletts in estuaries developed EUS as a result of low salinity and unexpected rains.

Behavioral and Clinical Symptoms of EUS

The symptoms of EUS are noticeably distinct from those of the other low-level ulcerative disorders previously observed in fish. Fish with EUS have identical behavior in both open water and enclosed water. Fish float on the water's surface and become lethargic, often sticking their heads out. Initial symptoms of the disease include hemorrhagic red lesions. Sometimes, like in *Wallago attu*, it may appear as raised blisters. These red lesions eventually deepen, spread, and take on the appearance of ulcers. As the ulcer progresses, scales frequently fall off, penetrate muscular layers, and become deeply hemorrhagic. In severe cases, the entire caudal peduncle is lost, and the skull is obliterated, exposing the brain where the lesions are in the head area. Only fish that can survive in unfavorable environmental conditions, such as *Channa* sp., exhibit the disease's typical acute phases (Das and Das, 1993).

Process of the Disease Development

Due to the aseptate hypha's pattern of asexual spore morphogenesis, *Aphanomyces invadans* is classified as an *Aphanomyces* within the saprolegniaceans even though it lacks the typical sexual reproductive components. The principal and secondary zoospore forms of this oomycete are typical. Primary zoospores develop inside a zoosporangium and are released either by the mouth of a terminal sporangium or through evacuation tubes. The primary spores immediately take into the second form, which is subspherical, laterally biflagellate, and free-swimming. These mobile zoospores are crucial in the progression of the disease. When the motile spore adheres to the fish's skin, it will germinate under favorable conditions, and its hyphae will enter the fish's skin, muscles, and internal organs. When a suitable host is not available or when secondary zoospores are exposed to poor environmental circumstances, they can encyst in the aquatic environment while they wait for favorable conditions for the reactivation of the spores (Kamilya and Baruah, 2014).

Low pH, intraspecies aggressiveness, or fish infection are some of the predisposing variables that may lead to skin injury. These elements collectively are thought to function as entry points for infection. Low water temperatures brought on by rain or the winter season, changes in various water quality indicators, exposure to toxins, and infection with pathogens like ectoparasites, bacteria, or viruses are other associated variables that might affect the disease's development. It has also been observed that *A. invadans* can spread horizontally from one fish to another through the water supply without causing any prior damage.

Diagnostic Methods

Clinical symptoms, gross pathology, the presence of mycotic granulomas in the histological section, and the isolation of the oomycete from internal tissues are used to make the diagnosis of EUS. More precise diagnoses are being made using molecular methods.

Clinical Signs, Gross Pathology and Histopathology

Fish with EUS typically exhibit loss of appetite, sluggishness, and flopping around erratically. The infected fish typically exhibits minute red spots the size of pinheads on various body areas, which develop into small dermal ulcers during the middle stage. The appearance of large hemorrhagic and necrotic open ulcers on the body's surface characterizes advanced-stage lesions, which are also defined by their existence. According to histopathology, the oomycete's wide, aseptate hyphae are present in the tissues of fish that have been exposed to EUS. The appearance of epithelioid granulomas in many organs, such as muscle, the liver, the kidney, etc., is the most conspicuous diagnostic characteristic of EUS.

Molecular Diagnosis

Western blot and electrophoretic analysis, pyrolysis mass spectrometry, and monoclonal antibody-based detection are other techniques for identification. However, the ability to identify the oomycete accurately is constrained by traditional and modern approaches. For the early identification of *Aphanomyces invadans* from infected fish, various molecular methods have recently been developed. Fluorescent in situ hybridization tests (FISH) and species-specific PCR utilizing primers that target the pathogen's internal transcribed spacer (ITS) region or neighboring DNA regions are now being explored. These tests are used to screen infected fish populations and quickly detect and identify *A. invadans*.

Control and Prevention

Several strategies have been demonstrated in small confined water bodies and fish ponds to lower the threat of EUS outbreaks or reduce mortality. It is impossible to regulate EUS in natural waterways. Liming the water, improving the water's quality, and removing the diseased fish have

occasionally proved successful in lowering mortality in outbreaks that occur in tiny, confined water bodies. Fishermen must watch wild fish throughout the dry and cold seasons. Farmers should stop exchanging water if there are fish with the EUS in the wild. This little step can slow down or stop the spread of the EUS. The disease problem can also be reduced by preventing any potential vectors, such as birds or terrestrial animals, contaminated fishing gear, and nets, from entering fish ponds. The Ash, turmeric, neem seeds or branches (*Azadirachta indica*), dried banana leaves, CIFAX (an Indian proprietary product), and other therapies have been tried with varying degrees of success for preventative and treatments of the EUS-infected fish. There is currently no vaccination available to protect against *A. invadans*. However, after being treated with various antigenic preparations from *A. invadans*, snakehead (*Channa striatus*) and rainbow trout (*Oncorhynchus mykiss*) exhibited antibody responses, showing the participation of humoral immune responses against the oomycete. Recent research showed that three antigenic preparations-fungal extracts, fungal extracts combined with Freund's incomplete adjuvant, and extracellular products-could be candidates for a vaccine against Catla, *C. catla*.

Conclusion

EUS is one of the most virulent and deadliest diseases among the broad spectrum of fish species. The illness's epizootic origin and wide range of vulnerable hosts; however, give it the ability to spread to unaffected regions. Considering its global nature, it was listed by OIE in 2013. The disease has been found to infect more than 100 species of fish and cause huge economic losses around the world. This article describes the gradual spread of EUS in India and the world along with its etiology, disease development, diagnosis, control, and prevention measures.

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