# Postgraduate Fish Disease Course

# Code (185)

***By Dr. Mortada M. A. Hussein***

# Bacterial septicemic syndrome

Recently, many attempts advocated the expansion in fish farming to optimize the real need for animal protein. Concurrently, and as a result of fish farming and its intensification, many fish diseases started to appear and spread particularly among cultured fishes and even among wild fishes due to importing of some exotic fish species either for farming or ornamental. Certainly, an increasing range of Gram-negative and Gram-positive bacteria has become associated with diseases outbreaks among not only freshwater but marine water fishes also.

Bacterial septicemic syndrome (BSS), from clinical point of view, is a generic term used to designate similar, but different, diseases in which many members of either Gram-negative or Gram-positive are incriminated.

In contrast, fishes with septicemic syndrome exhibit very similar symptoms and clinical signs regardless of the etiological agents. The main pathogenic species responsible for this syndrome are some members of aeromonads, vibrios, yersenia, edwardsiella as well as Streptococci. Outbreaks with such pathogens have had increasing prominence as agents of fish disease with particularly sever losses in captive and wild different fish species at different seasons as well as different localities. Infections associated with these micro-organisms are characterized by anorexia, septicemia, ascitis, exophthalmia, hemorrhages, skin ulceration together with high morbidities and mortalities.

*By Dr. Mortada M. A. Hussein*

# Motile Aeromonas septicemia (MAS)

**Bacterial Hemorrhagic Septicemia, Red Pest, Carp Dropsy, Red fin Disease, Red-Leg Disease of frog or MAS**

**Definition**

"Acute, subacute, and chronic bacterial disease of freshwater fishes, reptile (turtles), and amphibians characterized by septicemia, high mortalities (young fishes), exophthalmia, ascitis, and skin ulceration".

**Etiology**

***Aeromonas hydrophilia*** (***A.punctata***), Gram negative short rods, aerobic and facultative anaerobic microorganism, motile by polar (axial) flagellum, non-spore forming, non acid fast, non capsulated.

The organism grows well on ordinary medium, Nutrient agar (NA), typto-soy agar (TSA), MacConkey agar and Brain heart agar (BHA) at 25-28 ºC giving white to pinkish round small colonies. On Rimler-Shotts medium (selective medium for aero monads & psuedomonads) it grows as small yellowish round colonies at young stage (18-24 h) this color changed to greenish color in old culture. The color change of the old colonies is mainly due to attacking the maltose with production of acid that react with the bromothymole blue and give the greenish coloration leading to misdiagnosis with Pseudomonas flurences. The organism is resistant to the vibriostatic agent 0/129 and cannot grow on media containing 6.5%NaCl.

The organism produces haemolysine, haemagglutinine protease, enterotoxins, cytotoxin, as well as dermo-necrotic toxins.

**Susceptibility**

All freshwater fishes are susceptible to MAS, snakes, frogs, turtles, and amphibians are also susceptible to the disease.
The microorganism reported as the cause of swimming pool abscesses, septicemia, and peritonitis in human being.

**Predisposing Causes (stressors)**

- Overcrowding.
- Low dissolved oxygen.
- Presence of large amount of organic matter.

- Nutritional deficiencies.
- Injuries of the skin or gill either by trauma or ectoparasites.
- Temperature variation between seasons (eg. summer & spring).
- Rough handling especially during transportation.

**Mode of infection**

Mainly through ingestion and injuries of skin and /or gills. Shaded microorganisms from infected aquatic animals, infected dead carcasses as well as polluted water with the microorganism act as the source of infection.

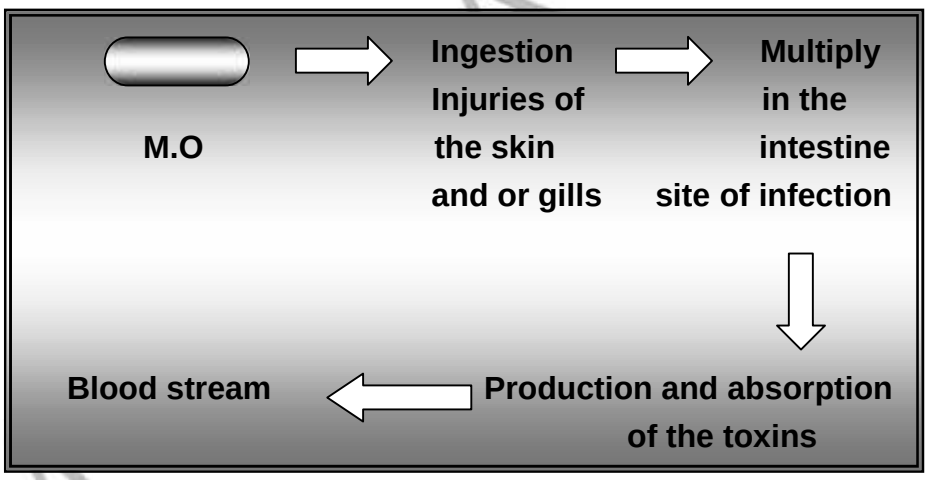
**transmission**

The transmission or the spread of the disease is usually horizontal (from infected material to the fish and /or aquatic animals.

**Source of infection**

Shaded microorganisms from infected fishes and /or other infected aquatic animals, polluted eggs in hatcheries and the water itself contains the organism as a commansales.

**Pathogenesis**




**Disease signs**

**Acute:**

- Onset of rapid fatal septicemia associated with mortality rate of 90-100%. The dying fish are easily to be handled and crowed at the water surface.

**Subacute:**

- Erythema at the base of fins, mouth, groves under the lower jaw and around the anal opening.
- Accumulation of clear yellowish to sanguineous fluid in different parts of the body cavities (e.g. abdominal cavity, scale pouch, under the skin).
- Exophthalmia as a result of accumulation of the body fluid in the eye socket.
- Ascitis sometimes associated with anal prolapse.

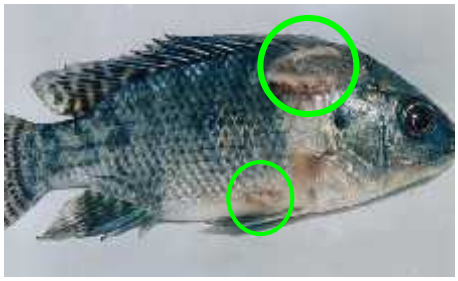
Common carp, *cyprinus carpio,* shows typical external signs of MAS.

Nile tilapia, *Oreochromis niloticus,* affected with MAS, you can see haemorrhages of the dorsal fin and body surface.

**Chronic or ulcerative:**

- This form characterized by ulcer formation especially at heavy emasculated parts of the body (e.g. caudal peduncle, nuchal region).
- The ulcer is usually hemorrhagic and circular or elliptical in shape.

Nile tilapia, *Oreochromis niloticus,* affected with MAS, haemorrhages of body surface and protrusion of the anal opening.



Nile tilapia, *Oreochromis niloticus,* affected with MAS. Note the ulcers on the nuchal region and abdomen.

**Postmortem Findings**

- Ascetic fluid together with peritoneal and visceral adhesion.
- Splenomagally and enteric hyperanemia.
- Kidney and liver become friable and soft.

**Microscopic Pathology**

- Subepidermal hemorrhages and edema together with coaggulative necrosis of the epidermal cells.
- Swollen acidophilic skeletal muscle fibers and surrounded by erythrocytes and small lymphocytes.
- Coagulative necrosis of hepatocytes and renal tubules.

**Diagnosis**

I. Case history revealed that:

- Loss of appetite or may the fishes refuse food.
- Sluggish swimming and the fish seek the sides of the holding facilities.
- Presence of mortalities.

II. The disease signs (as mentioned above).

III. P. M. findings.

IV. Laboratory diagnosis:

- Samples: kidney, liver, spleen, blood, muscles especially the deep layer of the ulcer.
- Squash smear from the organs stained with Gram.
- Isolation and identification:
  Aeromonads could be isolated on NA, BHA, TSA, and MacConkey agar at 22~36 ºC from kidney of the fish with hemorrhagic septicemia.
  Identification through using biochemical tests, API kits, gel-diffusion test, FAT, ELISA, and PCR (polymerase chain reaction).
- Histopathological findings (as mentioned above).

**Therapy & Control**

**Chemotherapy**

- In early stages of infection antiseptic bathes are recommended.
- Oxytetracycline 55mg/Kg fish in the food for 10 days.
- Sulfamerazine 264mg/Kg fish in the food for 3 days followed by 154mg/Kg fish for additional 11 days.
- Sulfaguanidine + sulamerazine combination (1 : 2) 130mg/kg fish for 3days followed by 90mg/Kg fish for 11days
- Oxolinic acid 10~30mg/Kg fish for 10 day.

**Control**

Good hygiene and removal of all stressors is the proper way for disease control this can be achieved through:

- Avoid overcrowding.
- Proper disposal of dead and dying fishes either by burning or burying.
- Control of aquatic animals such as reptiles and amphibians.
- Proper disposal of infected fish if in small number.
- Proper drainage, drying, and disinfectant of the pond (quick lime 4 tone/acre.
- Vaccination.

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# Furunculosis

## Ulcer Disease, *Aeromonas salmonicida* infection

**Definition**

" Peracute, acute, subacute, chronic and latent bacterial disease of fresh, brackish, and marine water fishes, primarily affect salmonids, characterized by formation of furuncules (boil-like lesions) in various tissue of the body, and the later stage, ulcerative lesions on infected fish".

**Etiology**

***Aeromonas salmonicida***, Gram-negative (bipolar) short rods to oval (1X1.7~2 µm) psychrophilic (typical and atypical).

The atypical strains of *A. salmonicida* based on the host from which they were isolated and the associated pathology.

The microorganism, aerobic but has the capability to grow as facultative anaerobic microorganism, non-spore forming, non-acid fast, non capsulated. It is **NON-MOTILE**.

The microorganism is cytochrome oxidase positive, O/F positive, catalase positive.

The organism grows well on ordinary medium, Nutrient agar (NA), typto-soy agar (TSA), and Brain heart agar (BHA) at 18-22 ºC (with range from 6~34.5 ºC) with pH range of 6.4~8. On culture media virulent colonies are small (pin-headed) and friable.

Chromogenic strains of *A. salmoncida* produce brown to brown-red water-soluble pigment. Addition of blood serum to the isolating media enhances the pigment production.

The microorganism also shows resistance to the vibriostatic agent 0/129.

**Biochemical pathogenicity**

The organism produces proteases, endotoxins, cytotoxin, and leucocytolytic responses (early infection).

The secreted proteoases mainly prefer gelatin and collagen as

the main substrate and therefore, cause liquifactive necrosis to the host.

The endotoxins that librated from the microorganism repress the haemopiosis in kidneys and spleen leading to repression of the defense mechanism.

The rapid utilization of glucose by the microorganism cause hypoglycemic shock to the host.

**Susceptibility**

Salmonid species (brown trout and brook trout) are the most susceptible fish, however, other non-salmonid freshwater (e.g. carp, goldfish, Japanese eel) marine water (e. g. flounder, black rockfish, turbot) fish species were found to be susceptible to the disease.

It is believed that, the level of susceptibility to infection by *A. salmonicida* among most susceptible fish species is variable even within the same fish species. Furthermore, both early stages and old-age fishes are more susceptible.

**Predisposing Causes (stressors)**

- Overcrowding.
- Low dissolved oxygen.
- Presence of large amount of organic matter.
- Nutritional deficiencies.
- Injuries of the skin or gill either by trauma or ectoparasites.
- Temperature variation between seasons (eg. summer & spring).
- Rough handling especially during transportation.

**Mode of infection**

Mainly through ingestion.

Shaded microorganisms from infected aquatic animals, infected dead carcasses, the material released from ruptured furuncules as well as the polluted water with the microorganism act as the source of infection.

The transmission or the spread of the disease is usually horizontal (from infected material to the fish and /or aquatic animals.

**Transmission**

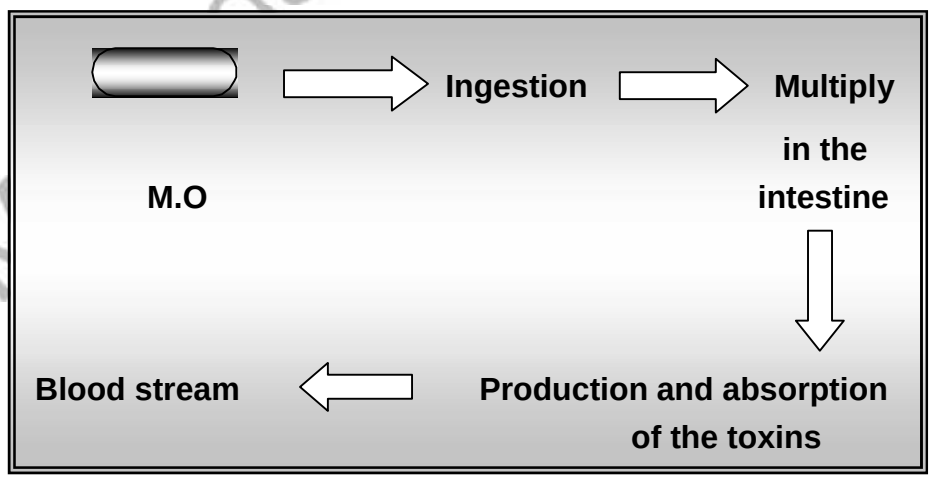
**Through eggs from infected fish (False Vertical transmission)**

As the micro-organism can be found within the ovaries of the infected fish, it could be passed at the time of spawning to become a source of infection to other eggs and other fish. The bacterium apparently on the surface of the egg and easily destroyed by external disinfectant.

**Source of infection**

- Shaded microorganisms from infected fishes in latent or chronic form, infected dead carcasses, the material released from ruptured furuncules as well as the polluted water with the microorganism act as the source of infection.
- Amphibians, reptiles, and other vertebrates that harbor the microorganis within their bodies.
- Migratory susceptible fish.

**Pathogenesis**




**Disease signs**

**Per acute:**

- This form is only restricted to young fish.
- Onset of rapid fatal septicemia associated with mortality rate of 90-100% with little exophthalmia.

**Acute:**

- This form usually affecting growing fish.
- Hemorrhagic septicemia including bloody anal vents.
- Hemorrhagic batches along dorsal and sides of the body surface.
- Rise of furuncules over the body surface (dermal layer).

**Subacute & chronic:**

- This form usually common in old fishes.
- Darkening of the skin.
- Erythema at the base of fins, mouth, groves under the lower jaw and around the anal opening.
- Expression sanguineous fluid from nars and anal opening.
- Slight exophthalmia.
- Development of furuncles over various parts of the body that undergo ruptured oozing sanguineous fluid leaving raw surface (ulcer).

**Postmortem Findings**

- Ascetic fluid together with peritoneal and visceral adhesion.
- Hemorrhages of the visceral organs with local necrotic foci.
- Kidney and liver become friable and soft.

**Microscopic Pathology**

- Subepidermal hemorrhages and edema together with coaggulative necrosis of the epidermal cells.
- Swollen acidophilic skeletal muscle fibers and surrounded by erythrocytes and lymphocytic infiltrations.
- Liquifactive necrosis of hepatocytes and renal tubules.

**Diagnosis**

I. Case history revealed that:

- Lethargic swimming and surfacing of the fish.
- The fish seek the sides of the holding facilities and refuse to feed.
- Presence of mortalities.

II. The disease signs (as mentioned above).

III. P. M. findings.

IV. Laboratory diagnosis:

- Samples: kidney, liver, spleen, blood, muscles especially the deep layer of the ulcer.
- Squash smear from the organs stained with Gram.
- Isolation and identification:

  *A. salmonicida* could be isolated on NA, BHA, TSA, and MacConkey agar at 20~22º C from kidney of the fish with hemorrhagic septicemia.

  Identification through using the presumptive tests includes four important parameters:

  Morphplpgy, Gram stain, motility and pigment production.

  Pigment production test:

  **One colony + one drop freshly prepared 0.1% paraphenyelendiamine-HCl ⟶ purple-brown color within 30 seconds**

  Biochemical tests, slide agglutination test, gel-diffusion test, FAT, ELISA, and PCR (polymerase chain reaction).
- Histopathological findings (as mentioned above).

**Therapy & Control**

**Chemotherapy**

- Oxytetracycline 55mg/Kg fish in the food for 10 days (if combined with sulfa-drugs, use the half of the recommended dose).
- Sulfamerazine 264mg/Kg fish in the food for 3 days followed by 154mg/Kg fish for additional 11 days.

- Sulfaguanidine + sulamerazine combination (1:2) 130mg/kg fish for 3days followed by 90mg/Kg fish for 11days
- Furazolidone at 35mg/Kg fish in the food for 20 days.
- Oxolinic acid 10~30mg/Kg fish for 10 day.

**Control**

Good hygiene and removal of all stressors is the proper way for disease control this can be achieved through:

- Test and slaughter, quarantine and restriction of the movement during outbreaks.
- Disinfections of eggs and hatcheries facilities with suitable disinfectant.
- Avoid overcrowding.
- Proper disposal of dead and dying fishes either by burning or burying.
- Control of aquatic animals such as reptiles and amphibians.
- Proper disposal of infected fish if in small number.
- Vaccination.

*By Dr. Mortada MA Hussein*

# VIBRIOSIS OF FISH

**Red pest of eel, Red sore disease, Pike pest, Red boil, Salt water furunculosis**

**Definition**

"Acute, sub-acute, and chronic bacterial disease of marine, brackish and freshwater fishes characterized by septicemia, erythema and hemorrhages on the skin with high morbidities as well as high mortalities".

**Etiology**

***Vibrio* (*Listonella*) *anguillarum***, and ***Vibrio ordalii*** are the most ***Vibrio*** species incriminated in fish vibriosis. They are, Gram negative straight or curved rods, motile by polar (axial) flagellum, non-spore forming, non acid fast, non capsulated and doesn't produce pigment. The organisms facultative anaerobic ferment glucose with production of acid only.

There are 3 serotypes, which are responsible for the disease outbreaks.

**Susceptibility**

About 48 fish species are susceptible to be infected with ***V. anguillarum*** or ***V. ordalii*** most of them are marine and estuarine ones (salmonids, sole, cod, mullet, herring, flounder and turbot). Also freshwater fishes (eels, tilapia,carp,trout, barbes) are susceptible to vibriosis.

**Predisposing causes (stressors)**

- Overcrowding.
- Low dissolved oxygen.
- Presence of large amount of organic matter.
- Nutritional deficiencies.
- Injuries of the skin or gill either by trauma or ectoparasites.
- Temperature variation between seasons (eg. summer &

spring).

- Rough handling especially during transportation.

**Mode of infection**

Mainly through ingestion and injuries of skin and/or gills.
Feeding on infected fish offal's.

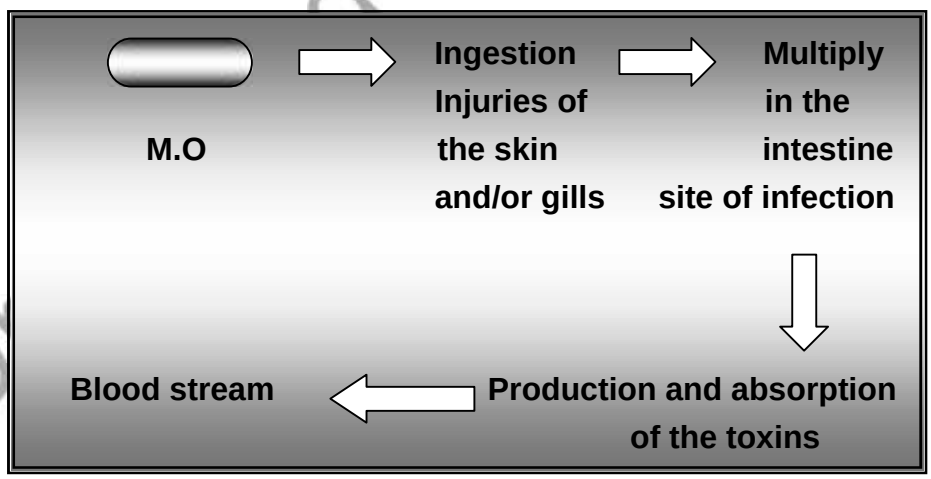
**transmission**

The transmission or the spread of the disease is usually horizontal (from infected material to the fish).

**Source of infection**

- Shaded microorganisms from infected aquatic animals, infected dead carcasses as well as polluted water with the microorganism act as the source of infection.
- Carriers specially herring fish.
- Polluted eggs in hatcheries and the water itself contain the organism as a commansales consider as a source of infection.

**Pathogenesis**




**Disease signs**

**Peracute or Acute:**

- Onset of rapid fatal septicemia associated with mortality rate of 90-100%. The dying fish are easily to be handled and crowed at the water surface.

**Subacute:**

- Erythema at the base of fins, mouth, groves under the lower jaw and around the anal opening.
- Boil like lesion under the skin and in the muscle, which may breakout to the exterior leaving opened sores that oozed blood-tinged exudates.

Atlantic salmon, *Salmo salar,* with vibriosis. Erythema around anus.

Goldfish, *Carassius auratus,* with vibriosis. Boil like lesion under the skin.

Rainbow trout, *Oncorhynchus mykiss,* with vibriosis. Opened hemorrhagic sore on the peduncle region.

Photo courtesy TFH Publications

**Chronic or ulcerative:**

- Deep glaucomatous muscle lesion on various parts of the body.
- Eye lesions including corneal edema, exophthalmia and ulceration.

**Postmortem (P.M.) Findings**

- Cut surface of the muscle revile presence of petechial hemorrhages and large boil-like lesions.
- Petechial hemorrhages in the peritoneum and visceral organs.
- Splenomagally and enteric hyperanemia.
- Kidney and liver become friable and soft.

**Microscopic Pathology**

- Congestion, dilated blood vessels and edema together with hyper-anemia of the hypodermis as well as epidermis.

- Zenker's necrosis in the center of the dermis and hypodermal layers.
- Focal coagulative and lquifactive necrosis of the liver, kidney's cells.
- Deposition of hemosidrine in the melano-macrophage centers of the spleen and hematopiotic tissues of the kidneys.

**Diagnosis**

I. Case history revealed that:

- Cessation of feeding or the fish refuse the food.
- Sluggish swimming and the fish swimming just below the water surface.
- Presence of mortalities.

II. The disease signs

III. The P. M. findings.

IV. Laboratory diagnosis:

- Samples: kidney, liver, spleen, blood, muscles especially the deep layer of the muscles.
- Squash smear from the organs stained with Gram.
- Isolation and identification:

  Vibrios could be isolated and grow well on ordinary medium supplemented with 1.5-3.5% NaCl, Nutrient agar (NA), typto-soy agar (TSA), MacConkey agar and Brain heart agar (BHA) at 18-20 ºC giving white to pinkish round small colonies.

  On Rimler-Shotts (R-S) medium it can grow only as small white round colonies if the vibriostatic agent (0/129 and/or novobiocin) isn't included.

  Identification through using biochemical tests, API kits, gel-diffusion test, FAT, ELISA, and PCR (polymerase chain reaction).

- Histopathological findings (as mentioned above).

**Therapy & Control**

**Chemotherapy**

- In early stages of infection antiseptic bathes are recommended.
- Oxytetracycline 55mg/Kg fish in the food for 10 days.
- Sulfamerazine 264mg/Kg fish in the food for 3 days followed by 154mg/Kg fish for additional 11 days.
- Sulfaguanidine + sulamerazine combination (1 : 2) 130mg/kg fish for 3days followed by 90mg/Kg fish for 11days
- Nitrofurzone 56mg/kg fish for 10 days.
- Oxolinic acid 10~30mg/Kg fish for 10 day.

**Control**

Good hygiene and removal of all stressors is the proper way for disease control this can be achieved through:

- Avoid overcrowding.
- Proper disposal of dead and dying fishes either by burning or burying.
- Control of aquatic animals such as reptiles and amphibians.
- Destruction of the carriers and disinfectant of the eggs.
- Proper disposal of infected fish if in small number.
- Proper drainage, drying, and disinfectant of the pond (quick lime 4 tone/acre.
- Vaccination using oral bacterine, hyperosmotic infiltration poly-vaccine.

*By Dr. Mortada MA Hussein*

# Pseudomonosis

## Psuedomonas spetecemia

**Definition**

"Acute, sub-acute, and chronic bacterial disease affect mainly cultured and aquarium freshwater, brackish and marine fishes, characterized by generalized septicemia accompanied with skin ulceration, high morbidities as well as high mortalities".

**Etiology**

***Pseudomonas fluorences***, is the most specie of this genus incriminated in fish pseudomonosis, however, there usually is a different species or strains of pseudomonades associated with each epizootic. Other pseudomonades have been isolated from fish septicemia, but most resemble *Pseudomonas fluorences.*

It is Gram-negative, rod-shape, oxidase positive, motile by one polar (axial) flagellum or three polar flagella in some strains, non-spore forming, non-acid fast, non-capsulated and produce diffusible pigment (dirty green) that fluoresce under UV light. The organisms attack glucose oxidatively with production of acid only and never fermentively {OF (+/-)}. The optimum growth temperature 20-25° C.

The organism produces hemolysins, dermolysins and proteolytic enzymes.

**Susceptibility**

All fish species are susceptible to be infected with *P. fluorences* particularly cultured and aquarium ones.

Pseudomonosis can occur as single-fish cases or as epizootics. Epizootics occur when all fish in the population become more or less susceptible to infection at the same time, such as the epizootics resulting from malnutrition.

*Pseudomonas* species consider as saprophytes opportunistic microorganisms, so outbreaks of pseudomonosis usually occurs secondary to chemical, physical and environmental stressors.

**Predisposing causes (stressors)**

- Overcrowding.
- Low dissolved oxygen.
- Presence of large amount of organic matter.
- Nutritional deficiencies.
- Injuries of the skin or gill either by trauma or ectoparasites.
- Temperature variation between seasons (eg. summer & spring).
- Rough handling especially during transportation.

Usually pseudomonosis is one time thing, if care is taken to keep fishes healthy under normal conditions.

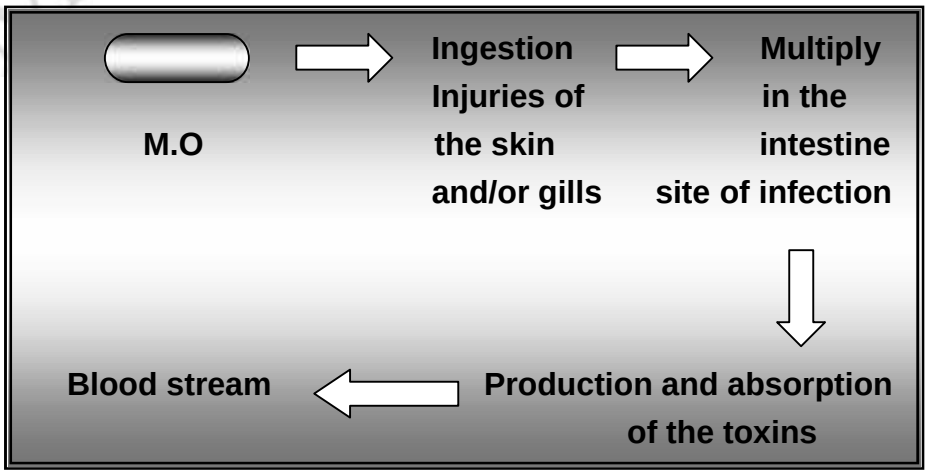
**Mode of infection**

Mainly through ingestion and injuries of skin and/or gills.

**Source of infection**

- Shaded microorganisms from infected fishes, infected dead carcasses as well as polluted water with the microorganism act as the source of infection.

**Pathogenesis**




As other septicemic disease with characterizations of:

- Erythema at the base of fins, mouth, groves under the lower

**Disease signs**

jaw and around the anal opening.

- Exophthalmia, edema and skin ulceration.

**Postmortem (P.M.) Findings**

- Generalization of petechial hemorrhages in the peritoneum and visceral organs.
- Intestine hemorrhagic and contain bloody fluids.
- Muscles may be more or less hemorrhagic according to the severity of the epizootic.

**Microscopic Pathology**

- Congestion, dilated blood vessels and edema together with hyper-anemia of the hypodermis as well as epidermis.
- Zenker's necrosis in the center of the dermis and hypodermal layers.
- Focal coagulative and lquifactive necrosis of the liver, kidney's cells.
- Deposition of hemosidrine in the melano-macrophage centers of the spleen and hematopiotic tissues of the kidneys.

**Diagnosis**

I. Case history revealed that:

- Cessation of feeding or the fish refuse the food.
- Sluggish swimming and the fish swimming just below the water surface.
- Presence of mortalities.

II. The disease signs

III. The P. M. findings.

IV. Laboratory diagnosis:

- Samples: kidney, liver, spleen, blood, muscles especially the deep layer of the muscles.
- Squash smear from the organs stained with Gram.
- Isolation and identification:

Pseudomonads could be isolated and grow well on ordinary medium, Nutrient agar (NA), typto-soy agar (TSA), MacConkey agar and Brain heart agar (BHA) or basal medium

supplemented with 5% hourse, bovine or sheep blood at 25-30º C giving yellowish-green round small colonies.

On Rimler-Shotts (R-S) medium it can grow as small yellowish-green colonies even the vibriostatic agent (0/129 and/or novobiocin) is included.

Identification through using biochemical tests, the oxidation-fermentation test (OF) thus becomes a prime method for separation of pseudomonads from aeromonads and vibrios.

API kits, gel-diffusion test, FAT, ELISA, and PCR (polymerase chain reaction).

- Histopathological findings (as mentioned above).

**Chemotherapy**

*Quite often there is no drug of choice for treatment of the disease because of the difference in species or strain of pseudomonads, and each may be controlled by a different therapeutics.*

**Therapy & Control**

- Oxytetracycline 55mg/Kg fish in the food for 10 days.
- Sulfamerazine 264mg/Kg fish in the food for 3 days followed by 154mg/Kg fish for additional 11 days.
- Sulfaguanidine + sulamerazine combination (1:2) 130mg/kg fish for 3days followed by 90mg/Kg fish for 11days
- Nitrofurzone 56mg/kg fish for 10 days.
- Oxolinic acid 10~30mg/Kg fish for 10 day.

**Control**

Good hygiene and removal of all stressors is the proper way for disease control this can be achieved through:

- Avoid overcrowding.
- Proper disposal of dead and dying fishes either by burning or burying.
- Control of aquatic animals such as reptiles and amphibians.
- Destruction of the carriers and disinfectant of the eggs.

- Proper disposal of infected fish if in small number.
- Proper drainage, drying, and disinfectant of the pond (quick lime 4 tone/acre.

*By Dr. Mortada MA Hussein*

# Edwarsiellosis

## Emphysematous Putrefactive Disease, *Edwardsiella* Septicemia

**Definition**

"Septicemic disease, which is temperature (seasonal) related especially at the mid-summer when the temperature exceed or above 30° C affecting freshwater, brackish water and marine water fishes, characterized by septicemia, slowly developed mortalities and skin ulceration".

**Etiology**

***Edwardsiella tarda***, Gram negative short rods, aerobic and facultative anaerobic microorganism, motile by peritrichous flagella, non-spore forming, non-acid fast, non-capsulated, cytochrome oxidase negative, produces hydrogen sulfide and indole.

The organism attacks glucose oxidatively and fermentatively with production of acid and gas, while attacks mannitol with production of acid only and doesn't attack other carbohydrates.

The organism produces extracellular enzymatic and/or toxic products as haemolysine, enterotoxins, as well as dermo-necrotic toxins. Also a slimy layer that acts as a production layer against the host defense mechanism surrounds the organism.

The organism grows well on ordinary medium, Nutrient agar (NA), typto-soy agar (TSA), MacConkey agar and Brain heart agar (BHA) at 30-35º C.

On Rimler-Shotts medium (selective medium for aero monads &psuedomonads) it grows as large deep greenish round colonies with black center.

**Susceptibility**

Freshwater fishes are the most susceptible ones, catfish, tilapia, Japanese eel, flounder, mullets, turtles, and amphibians are also susceptible to the disease.

***Edwardsiella tarda*** is reported as an important zoonotic

organism as it causes commonly gastroenteritis.

**Predisposing Causes (stressors)**

- Increase of temperature above 30 º C.
- Overcrowding.
- Low dissolved oxygen.
- Presence of large amount of organic matter.
- Nutritional deficiencies.
- Injuries of the skin or gill either by trauma or ectoparasites.
- Presence of carries and/or infected aquatic animals.
- Rough handling especially during transportation.

**Mode of infection**

Not defined but mainly through ingestion and injuries of skin and /or gills.

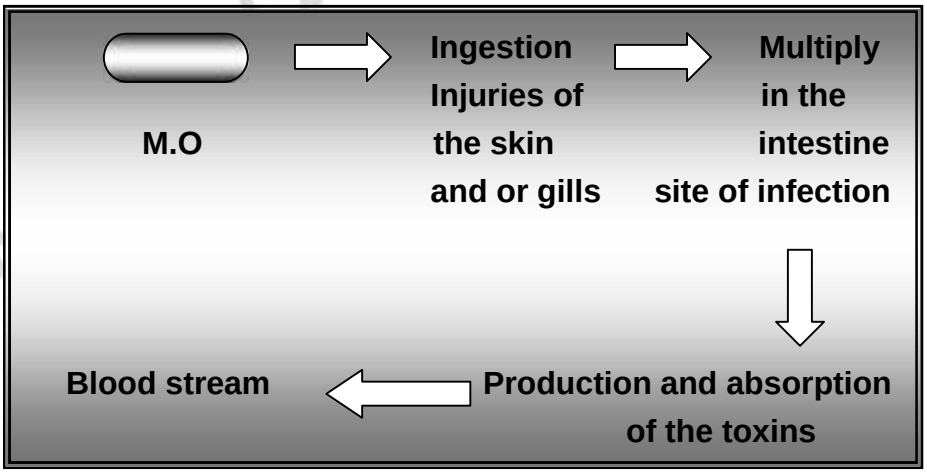
**transmission**

The transmission or the spread of the disease is usually horizontal (from infected material to the fish and/or aquatic animals.

**Source of infection**

- The organism is a normal inhabitant of the intestine of snakes that may shad their faces in the water leading to contamination of the water.
- Shaded microorganisms from infected fishes and/or other infected aquatic animals.

**Pathogenesis**




***The clinical signs of edwardsiellosis were considerably variable from fish species to another***

**In catfish:**

**Disease signs**

- Mainly the lesions are initially seen as subcutaneous abscesses or as reddened foci on the sides of the flank and

posterior regions, which undergo ruptured oozing singuinous fluid of offensive foul-smelling.

- Although the affected fish loses the control of its posterior part, it may continue to eat even if severely affected.

**In tilapia:**

- Skin depigmentation, swollen abdomen and corneal opacity.
- Hemorrhagic inflammation of the vent was also recorded.
- Internally, ascites together with presence of white small nodules scattered on the visceral organs and intestine.

Channel catfish, *Ictalurus puctatus,* with shows external abscess on the head

Nile tilapia, *Oreochromis niloticus,* affected with edwardsiellosis. Note the loss of pigmentation of the skin.

**In eels:**

- Abscesses that may opened and ulcerate through the body surface.
- Internally, abscessiation of the visceral organs with distribution of necrotic foci.

**Postmortem Findings**

- Ascetic fluid together with peritoneal and visceral adhesion.
- Kidney and liver become friable and soft.
- Presence of white small nodules scattered on the visceral organs and intestine.

**Microscopic Pathology**

- Subepidermal hemorrhages and edema together with coaggulative necrosis of the epidermal cells.
- Swollen acidophilic skeletal muscle fibers and surrounded by erythrocytes and small lymphocytes.

- Coagulative necrosis of hepatocytes and renal tubules.

**Diagnosis**

I. Case history revealed that:

- Loss of appetite or may the fishes refuse food.
- Sluggish swimming and the fish seek the sides of the holding facilities.
- Presence of mortalities.

II. The disease signs (as mentioned above).

III. P. M. findings.

IV. Laboratory diagnosis:

- Samples: kidney, liver, spleen, blood, muscles especially the deep layer of the ulcer.
- Squash smear from the organs stained with Gram.
- Isolation and identification:
  ***Edwardsiella tarda*** could be isolated on NA, BHA, TSA, and MacConkey agar at 22~36 ºC from kidney of the fish with hemorrhagic septicemia.
  Identification through using biochemical tests, API kits, gel-diffusion test, FAT, ELISA, and PCR (polymerase chain reaction).
- Histopathological findings (as mentioned above).

**Therapy & Control**

**Chemotherapy**

- Addition of vitamin C as a food supplements at a rate of 150mg/Kg fish in the food.
- Oxytetracycline 55mg/Kg fish in the food for 10 days.
- Sulfamerazine 264mg/Kg fish in the food for 3 days followed by 154mg/Kg fish for additional 11 days.
- Sulfaguanidine + sulamerazine combination (1:2) 130mg/kg fish for 3days followed by 90mg/Kg fish for 11days
- Oxolinic acid 10~30mg/Kg fish for 10 day.

**Control**

Good hygiene and removal of all stressors is the proper way for disease control this can be achieved through:

- Elimination and control of snakes is a principle for control of the disease occurrence.
- Avoid overcrowding.
- Proper disposal of dead and dying fishes either by burning or

burying.

- Control of aquatic animals such as reptiles and amphibians.
- Proper disposal of infected fish if in small number.
- Proper drainage, drying, and disinfectant of the pond (quick lime 4 tone/acre.
- Vaccination, formalized whole cultured or living attenuated one.

## Enteric Septicemia of Catfish

### ESC, *Edwardsiella ictaluri* infection

**Definition**

"Acute and chronic septicemic disease mostly affecting catfish (mainly channel catfish), especially at the mid-summer when the temperature around 24~28° C characterized by septicemia, spiral swimming, ascites, skin hemorrhages and ulceration and hole formation on the skull".

**Etiology**

***Edwardsiella ictaluri***, Gram negative short rods, aerobic and facultative anaerobic microorganism, motile by peritrichous flagella, non-spore forming, non-acid fast, non-capsulated, cytochrome oxidase negative, doesn't produce hydrogen sulfide.

**Susceptibility**

Enteric septicaemia of catfish is a highly infectious bacterial disease of catfish, especially ictalurids.

Black bullhead (Ictalurus melas)
Blue catfish (Ictalurus furcatus)
Brown bullhead (Ictalurus nebulosus)
Channel catfish (Ictalurus punctatus)
Glass knife fish (Eigenmannia virescens)
Rosy barb (Puntius conchonus)
sind danio (Danio devario)
sutchi catfish (Pangasius hypophthalmus)
Walking catfish (Clarias batrachus)
White catfish (Ictalurus catus)
Yellow bullhead (Ictalurus natalis)
zebra fish danio (Brachydanio rerio)
chinook salmon (Oncorhynchus tshawytscha)
Rainbow trout (Oncorhynchus mykiss)
Tilapia spp.

**Disease signs**

**Acute form:**

- Infected fish occasionally hang head up in the water and exhibit spiral swimming usually followed by death.
- Abdominal distention, exophthalmia, or pale gills.
- Petechial hemorrhages on the dorsum, flanks, jaws and operculum.

**Chronic form:**

- Formation of hole in the head, which undergo ulcerate.
- Nervous manifestations characteristics to meningitis.

**Postmortem Findings**

- Body cavity cotains clear or bloody fluid.
- Hamorrhages and necrosis of the liver.
- Hypertrophy of the spleen and kidenys.
- Petechial hemorrhages in the muscles.

**Microscopic Pathology**

- Enteritis, hepatitis, myositis, and interstitial nephritis begin as acute lesions and developed into chronic foci.
- Inflammation of the olfactory nerve and eventually reach the olfactory lobe of the brain.
- Meningoencephalitis r with aggregation of macrophages in the lesion that often harboring the invading bacteria.

**Diagnosis**

I. *Case history* revealed that:

- Loss of appetite or may the fishes refuse food.
- Sluggish swimming and the fish seek the sides of the holding facilities.
- Presence of mortalities.

II. *The disease signs* (as mentioned above).

III. P. M. findings.

IV. Laboratory diagnosis:

- Samples: kidney, liver, spleen, blood, muscles especially the deep layer of the ulcer.
- Squash smear from the organs stained with Gram.
- Isolation and identification:
  ***Edwardsiella ictaluri*** could be isolated on NA, BHA, TSA, and MacConkey agar at 22~28 ºC from kidney of the fish with hemorrhagic septicemia.
  Identification through using biochemical tests, API kits, gel-diffusion test, FAT, ELISA, and PCR (polymerase chain reaction).
- Histopathological findings (as mentioned above).

**Therapy & Control**

**Chemotherapy**

*The treatment should be start as fast as possible before the fish refuse to food*

- Oxytetracycline 55mg/Kg fish in the food for 10 days.
- Sulfamerazine 264mg/Kg fish in the food for 3 days followed by 154mg/Kg fish for additional 11 days.
- Sulfaguanidine + sulamerazine combination (1:2) 130mg/kg fish for 3days followed by 90mg/Kg fish for 11days
- Oxolinic acid 10~30mg/Kg fish for 10 day.

**Control**

Good hygiene and removal of all stressors is the proper way for disease control this can be achieved through:

- Elimination and control of snakes is a principle for control of the disease occurrence.
- Avoid overcrowding.
- Proper disposal of dead and dying fishes either by burning or burying.
- Control of aquatic animals such as reptiles and amphibians.
- Proper disposal of infected fish if in small number.
- Proper drainage, drying, and disinfectant of the pond (quick lime 4 tone/acre.
- Vaccination, formalized whole cultured or living attenuated one.

*By Dr. Mortada MA Hussein*

# Yersiniosis

**Enteric Red Mouth Disease (ERM), Red throat Disease, Pink Mouth Disease**

**Definition**

"Acute, subacute, and chronic bacterial disease of **Salmonids**, in particularly, cultured ones characterized by septicemia, high mortalities (young fishes), exophthalmia, skin and mouth erythema, and hemorrhages within the visceral organs".

**Etiology**

***Yersinia ruckeri***, one member of the family entrobacteriacae, Gram negative rods, cytochrome oxidase negative, motile by peritrichous flagella, non-spore forming, non acid fast, non capsulated, doesn't produce pigment. It doesn't survive well outside its host.

The organism grows well on ordinary medium, Nutrient agar (NA), Triple sugar iron agar (TSI), MacConkey agar and Brain heart agar (BHA) at 18-22 ºC giving white translucent round small colonies.
On selective medium (contains tween 80, sucrose and bromothymol blue) it grows as small greenish round colonies with a surrounding area of hydrolysis.
Attack glucose and mannitol oxdatively and fermentatively with production of acid only and doesn't other carbohydrates.

**Susceptibility**

Almost all salmonids are the susceptible fish species with a pronounced clinical signs.
The organism could be isolated asymptomatically from other fish species such as Carp, goldfish, eels, sea bass, and turbot.

**Predisposing Causes (stressors)**

- Overcrowding.
- Low dissolved oxygen.
- Presence of large amount of organic matter.
- Nutritional deficiencies.
- Injuries of the skin or gill either by trauma or ectoparasites.
- Temperature variation between seasons (eg. summer & spring).
- Rough handling especially during transportation.

**Mode of infection**

Mainly through ingestion and injuries of skin and /or gills. Shaded microorganisms from infected aquatic animals, infected dead carcasses as well as polluted water with the microorganism act as the source of infection.

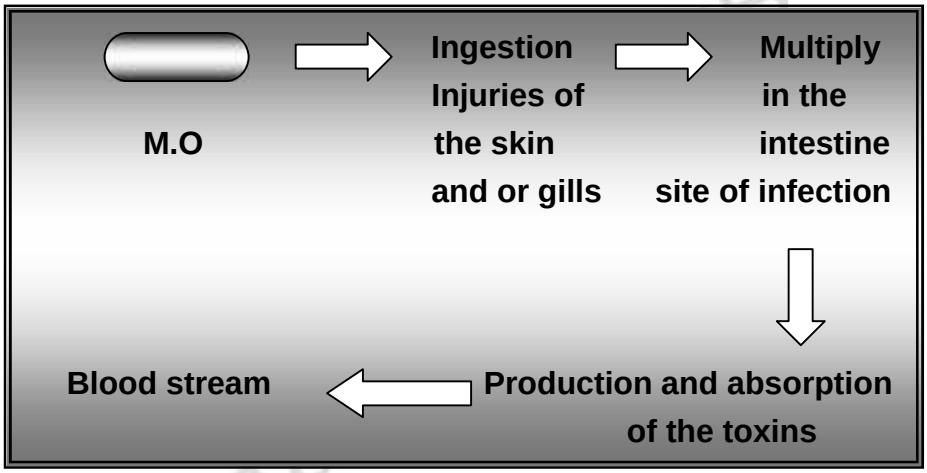
**transmission**

The transmission or the spread of the disease is usually horizontal (from infected material to the fish and /or aquatic animals.

**Source of infection**

- Shaded microorganisms from infected fishes and/or other infected aquatic animals such as crayfish.
- Carriers.

**Pathogenesis**




**Disease signs**

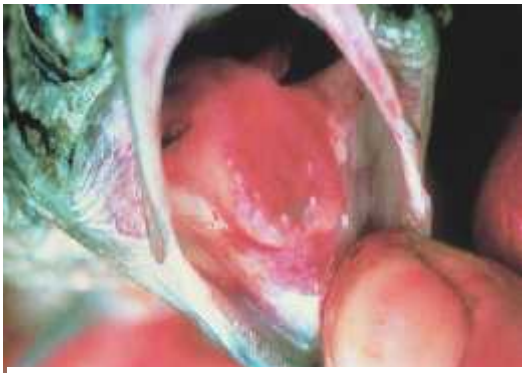
***Fish with yersiniosis may show one or more of the signs below, but disease may still be present in the absence of any signs.***

**Acute:**

- Onset of rapid fatal septicemia associated with low mortality rate that expand gradually to reach up to 75%. The dying fish are easily to be handled and crowed at the water surface.

**Subacute:**

- Reddening of corners of mouth, gums, palate and tongue (hence the name of the disease)
- Exophthalmia that may be associated with hemorrhages and "blood spot" on the cornea
- Ascitis, skin rashes sometimes associated with dark pigmentation.



Rainbow trout, *Onchorynchus mykiss,* shows typical signs ERD. Note the redden mouth and tongue.

Rainbow trout, *Onchorynchus mykiss,* shows typical signs ERD., Note hemorrhages on the body surface and eye.

**Chronic or ulcerative:**

- Darkening of the skin associated with unilateral or bilateral blindness.
- Fish become lethargic and float aimlessly.
- There may or may not erythema at the base of fins, mouth, or operculum.

Rainbow trout, *Onchorynchus mykiss,* shows typical signs ERD. Note hemorrhages at the base of the lower jaw.

**Postmortem Findings**

- Hemorrhages in the fat, gonads, peritoneum and swim bladder.
- Ascetic fluid together with peritoneal and visceral adhesion.
- Splenomagally and enteric hyperanemia.
- Swollen Kidney and liver become dark in color.

Rainbow trout, *Onchorynchus mykiss,* shows typical signs ERD. Note hemorrhades and visceral adhesion

**Microscopic Pathology**

- Deep hemorrhages, edma and hyperemia of the liver, kidney, and spleen tissues.
- Necrosis of the intestinal lining epithelia and may be sloughed.
- Colonization of the causative bacterium within the visceral organs.

***Most of these pathological alterations are non specific for yersiniosis and could be represented within other septicemic diseases.***

**Diagnosis**

I. Case history revealed that:

- Loss of appetite few mortalities increase day by day.
- Sluggish swimming and fish swim aimlessly.
- Some fish showed some of the clinical signs

II. The disease signs (as mentioned above).

III. P. M. findings.

IV. Laboratory diagnosis:

- Samples: kidney is the best organ for isolation of ***Y. ruckeri.***
- Squash smear from the organs and the lower intestine stained with Gram.
- Isolation and identification:
  ***Y. ruckeri*** could be isolated on NA, BHA, TSA, and MacConkey agar at 18~22 ºC from kidney of the fish with hemorrhagic septicemia.
  Identification through using biochemical tests, API kits, gel-diffusion test, FAT, ELISA, and PCR (polymerase chain reaction).
- Histopathological findings (as mentioned above).

**Therapy & Control**

**Chemotherapy**

- In early stages of infection antiseptic bathes are recommended.
- Oxytetracycline 55mg/Kg fish in the food for 10 days.
- Sulfamerazine 264mg/Kg fish in the food for 3 days followed by 154mg/Kg fish for additional 11 days.
- Sulfaguanidine + sulamerazine combination (1 : 2) 130mg/kg fish for 3days followed by 90mg/Kg fish for 11days
- Oxolinic acid 10~30mg/Kg fish for 10 day.

***Morbidity may reach 100%, however, mortalities depends upon the age of the affected fish as well as culture management and may not exceed than 50%.***

**Control**

Good hygiene and removal of all stressors is the proper way for disease control this can be achieved through:

- Prophylactic doses of bactericidal agents are recommended.
- Avoid overcrowding.
- Proper disposal of dead and dying fishes either by burning or

burying.

- Control of aquatic animals such as reptiles and amphibians.
- Hygienic destruction of the carriers.
- Proper disposal of infected fish if in small number.
- Proper drainage, drying, and disinfectant of the pond (quick lime 4 tone/acre.
- Vaccination.

# Streptococcosis

**Definition**

"Sub-acute, and chronic bacterial disease of marine, brackish and freshwater fishes characterized by depression or excitability, anorexia, unilateral or bilaterial exophthalmia with or without eye hemorrhages, erratic swimming and whirling, "C-shaped" posturing, septicemia, and death ".

**Etiology**

Three different genera of gram-positive cocci including Streptococci, Lactococci, and Vagococci are the most gram-positive cocci species incriminated in fish streptococcosis. ***Streptococcus iniae*** and ***Lactococcus garvieae*** are the principal pathogens of Streptococcosis although several other species and genera of pathogens associated with the disease have been identified causing similar disease symptoms.

They are one of the major problems of warm water aquaculture, but has very few limitations in regard to geographic boundaries or host ranges.

**Susceptibility**

Infections are reported in 22 fish species, both cultured and wild. The affected species reported include rainbow trout (*Oncorhynchus mykiss*), tilapia *(Oreochromis* spp.*),* yellowtail *(Seriola quinqueradiata)*, amberjack *(Seriola dumerili),* European seabass (*Dicentrarchus labrax*), European seabream (*Sparus aurata*), red drum (*Sciaenops ocellatus*), bastard halibut (*Paralichthys olivaceus),* eels *(Anguilla anguilla)* and Asian seabass (*Lates calcarifer*).

**Predisposing causes (stressors)**

- Overcrowding.
- Low dissolved oxygen.
- Presence of large amount of organic matter.

- Nutritional deficiencies.
- Injuries of the skin or gill either by trauma or ectoparasites.
- Temperature variation between seasons (eg. summer & spring).
- Rough handling especially during transportation.

**Mode of infection**

Mainly through ingestion of contaminated food (e.g. fish offal's) and injuries of skin and/or gills.

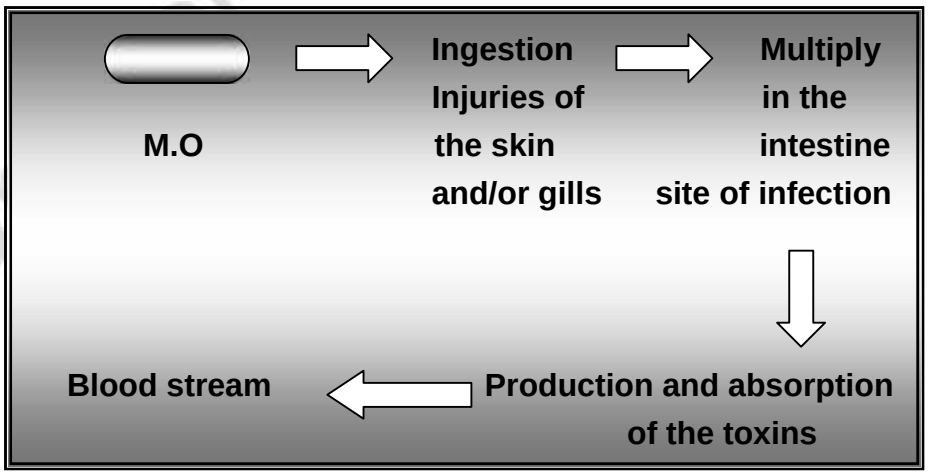
**transmission**

The transmission or the spread of the disease is usually horizontal (from infected material to the fish).

**Source of infection**

- Shaded microorganisms from infected aquatic animals, infected dead carcasses as well as polluted water with the microorganism act as the source of infection.
- Carriers and latent infected fishes.
- Polluted water with animals byproducts (e.g. integrated fish-agriculture) consider as a source of infection.

**Pathogenesis**

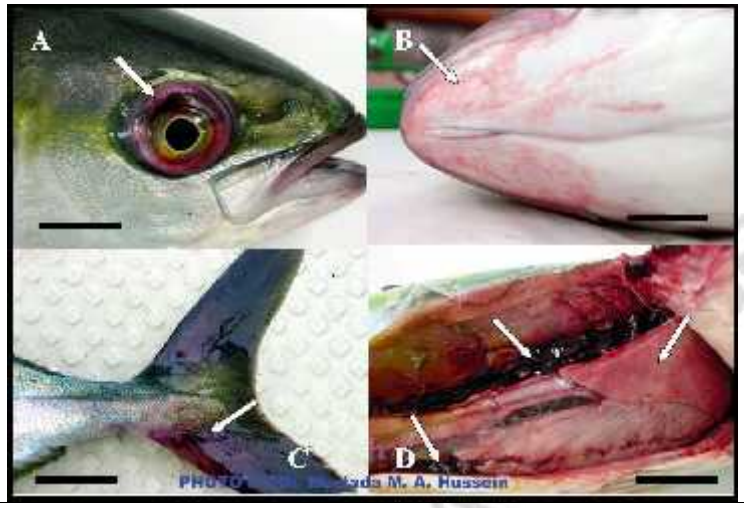



**Disease signs**

- Abnormal swimming behaviors including erratic swimming, swimming and whirling on the water surface, and "C-shaped body" curvature while on water surface.
- Ocular abnormalities include peri-orbital and intra-ocular

hemorrhages, opacity, purulence and exophthalmia.

- Hemorrhages were scattered on the body surface, particularly, at the base of lower jaw, base of the dorsal fin, and massively at the peduncle region and tail fin.

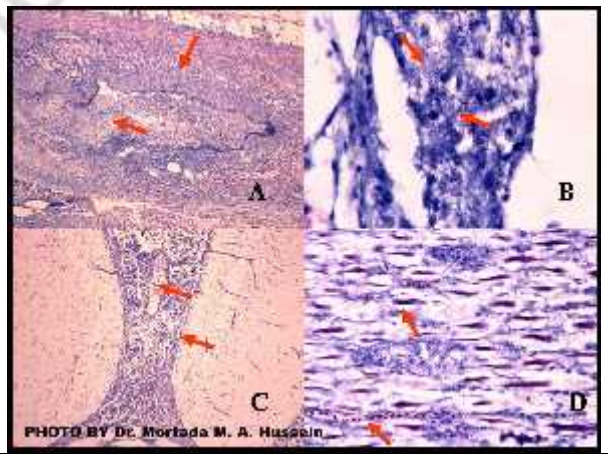



Yellowtail fish with streptococcosis, *(A) Exophthalmia together with peri-orbital and intra-ocular hemorrhages. (B) Hemorrhages at the base of the lower jaw. (C) Massive hemorrhages at the peduncle region and tail fin. (D) Paleness of the liver and slight enlarged spleen and kidney (arrows). Scale bar = 5 cm.*

**Postmortem (P.M.) Findings**

- Internally, there were no clear clinical change except paleness of the liver and slight enlarged spleen and kidney.
- Kidney and liver may become friable and soft.

**Microscopic Pathology**




*Histopathological alterations associated with streptococcosis infection in naturally infected yellowtail and amberjack. (A) Focal necrosis in periorpetal tissue together with inflammatory cell infiltration, hemorrhages and fibroplasias (H&E, x40). (B) Sever edema, infiltration of lymphocytes and macrophages in the periorbital tissue together with massive invasion of cocci bacterial cells (Giemsa, x100). (C) Brain showed meningitis, dilatation of blood vessels and massive cellular infiltration (H&E, x40). (D) Optic nerve showed edema, fibroplastic degeneration together with dilated blood vessels and erythrocytes infiltration (Giemsa, x100). Arrows.*

**Diagnosis**

I. Case history:

- Cessation of feeding or the fish refuse the food.
- Abnormal swimming behaviors and the fish swimming just below the water surface.
- Presence of mortalities and moribund fishes with characteristic lesions of streptococcosis .

II. The disease signs

III. The P. M.

IV. Laboratory diagnosis:

- Samples: kidney, liver, spleen, blood, muscles especially the deep layer of the muscles.
- Squash smear from the organs stained with Gram.
- Blood smear stained with Giemsa.
- Isolation and identification:

  Streptococci could be isolated and grow well on Todd Hewitt medium supplemented with 3-5% blood, typto-soy agar (TSA) with sodium azide, Brain heart agar (BHA) at 20-25º C giving white round small colonies.

  Identification through using biochemical tests, API kits, gel-diffusion test, FAT, ELISA, and PCR (polymerase chain reaction).

- Histopathological findings.

**Chemotherapy**

**Therapy & Control**

As with many gram-positive infections, treatment is problematic. Success has been achieved using a number of different antibacterial compounds; however, problems with antibiotic resistance haven't been encountered.

- In early stages of infection antiseptic bathes are recommended.

- Oxytetracycline 55mg/Kg fish in the food for 10 days.
- Sulfamerazine 264mg/Kg fish in the food for 3 days followed by 154mg/Kg fish for additional 11 days.
- Sulfaguanidine + sulamerazine combination (1 : 2) 130mg/kg fish for 3days followed by 90mg/Kg fish for 11days
- Nitrofurzone 56mg/kg fish for 10 days.
- Oxolinic acid 10~30mg/Kg fish for 10 day.

**Control**

Good hygiene and removal of all stressors is the proper way for disease control this can be achieved through:

- Avoid overcrowding.
- Proper disposal of dead and dying fishes either by burning or burying.
- Control of aquatic animals such as reptiles and amphibians.
- Destruction of the carriers and disinfectant of the eggs.
- Proper disposal of infected fish if in small number.
- Proper drainage, drying, and disinfectant of the pond (quick lime 4 tone/acre.
- Vaccination using oral bacterine, hyperosmotic infiltration poly-vaccine and DNA vaccines.