DISEASES CAUSED BY CYTOPHAGACEAE BATERIA

Family Cytophagaceae or Myxobacteria species are free living in the soil and water streams (fresh & marine) as saprophytes but under certain conditions they become opportunistic pathogens of fish and some amphibians causing high economic losses particularly in aquaculture field. They are Gram negative filamentous long-rod bacteria displaying motility by gliding. Almost all species could produce pigment (yellow, orange & red).

COLUMNARIS Disease

Mouth Fungus or Cotton wall Disease

Definition

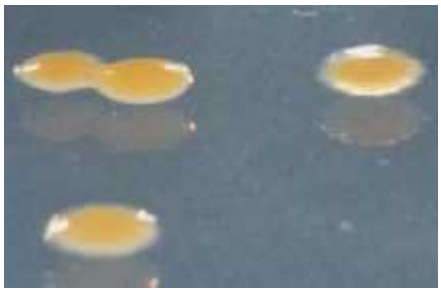
"***Sub acute to chronic bacterial disease of freshwater, brackish and marine fishes as well as aquarium ones characterized by formation of tiny wads of cotton spots on the body surface specially mouth, gills, head and fins*** ".

Etiology

Flavobater columnar (Flexibater columnaris)

Gram negative straight long rods, motile by gliding, non-spore forming, non acid fast, non capsulated and produce pigment, doesn't attack carbohydrates. The organism is strictly aerobic. It grows on Ordal's or Cytophaga agar media giving rhizoid pale yellow pigmented colonies. On sheep blood medium it grows as small smooth colonies surrounded with β-hemolytic zone.

Flavobater (Flexibater) maritimus: Cause salt water columnaris disease. It is similar to *F. columnaris* but is has an obligatory requirement for sea water.



From biochemical pathogenicity point of view the organism produces proteolytic enzymes.

Susceptibility

All fish species are susceptible to Columnaris disease particularly young and cultured ones. Freshwater fishes (e. g. Salmonids, eels, tilapia, carp, mullet, barbs). Marine fish such as sea bream and flounder.

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.
- **The disease is temperature-related most epizootics develop at 25° C.**
- Rough handling especially during transportation.

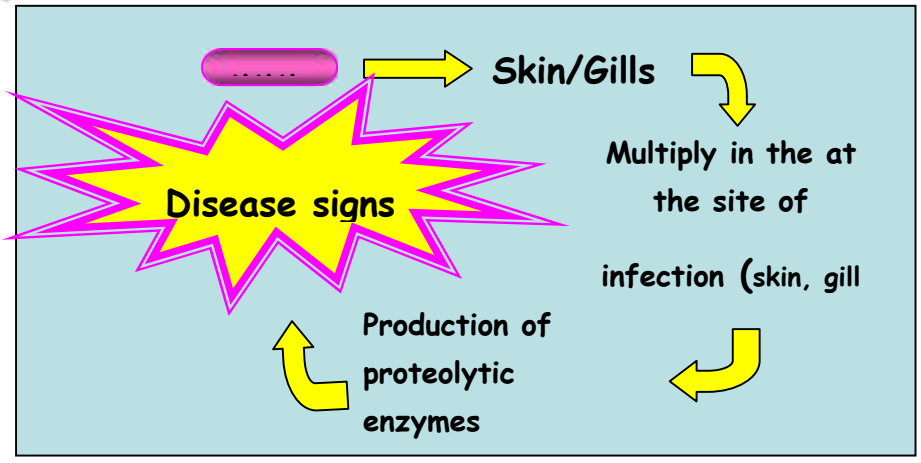
Mode of infection

Mainly through injuries of skin and/or gills.

Transmission & 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.

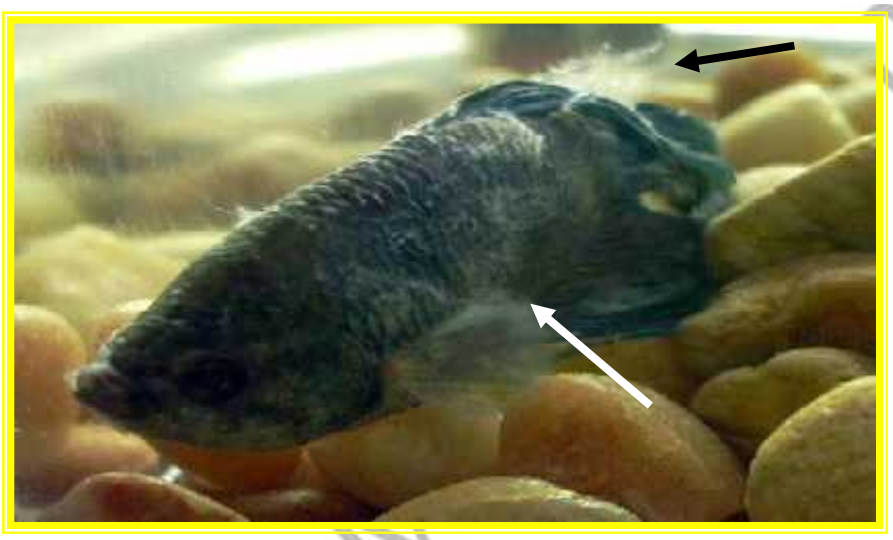
Pathogenesis



The severity of the infection depends upon the severity of the fish injuries and virulence of the bacterial strain as well.

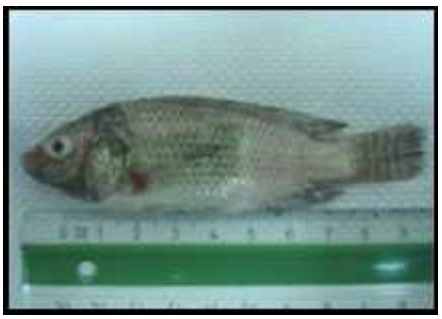
Disease signs

- Thickening of the mucous as various spots on the head, fins, mouth, operculum and around injured parts.
- Tuffs or flaks of epithelia and bacteria appear loosely attached to the affected parts as tiny wads of cotton.

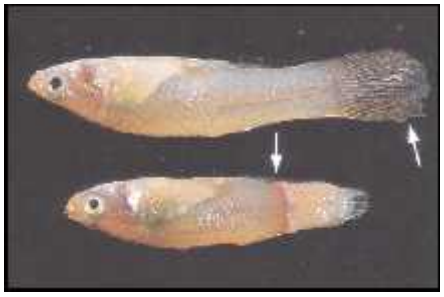


- Affected gills lamellae become clumped and eroded, which under go sloughed as actual loss of gill tissue.

- Grayish white discoloration on the outer margin of the fins that gradually spread and the body surface appears to be covered with mucus.



- Sever cases exists necrosis of the skin in the back region with appearance of small flaks of tissue dangling in the water.



- ***Scale-less fish:*** The lesion usually appears as small circular with grayish blue center and red margin between dorsal fin and caudal fin giving the syndrome namely "***Saddle back Disease***"
- ***In salt water columnaris:*** Slight blistering of the skin surface, which under go destructed leaving row hemorrhagic dermal surface.

N. B: ***Bactriaemia usually associated with excessive necrosis of the skin and gills.***

Mortalities are usually due to:

- **Osmoregulatory failure (Skin Lesion).**
- **Asphyxia and loss of excretory function (Gill Lesion).**
- **Bacterial toxins (Bacteriaemia).**
- **Toxic katabolic substances by the host body resulted under effect of photolytic enzymes of *F. columnaris***

Gross Pathology

- Clumped and eroded gills, which under go sloughed with different degrees of actual loss of gill tissue.
- Erosions of the fins, mouth and skin with different degrees according to the severity of the infection.
- Usually no internal lesion.

Microscopic Pathology

- ***SKIN*****,** Epidermal necrosis, peripheral hyperemia and hemorrhages.

- ***GILLS*****,** hyperplasic proliferation of gill epithelium with dilatation of blood vessels. Fusion of the secondary gill lamellae together with different degrees of gill necrosis.

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 dangling a cotton wall materials from their body surface and/or mouth opening.
- Presence of mortalities.

II. The disease signs

III. The P. M. findings.

IV. Laboratory diagnosis:

- ***Wet mount preparation:*** from Skin and gill scraping, and pieces of damaged fins directly examined under microscope. Presence of **hay sticks** bacterial colonization is definitive.
- Smear stained with Gram.
- Isolation and identification:

  Ordal's or Cytophaga agar media and Sheep blood agar at 25-28º C for 24-36 hours giving rhizoid pale yellow pigmented colonies. On sheep blood medium it grows as small smooth colonies surrounded with β-hemolytic zone.
- **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.
- Cupper sulphate 40-500 mg/L for 1 min.

- Potassium permanganate 2mg/L indefinite.
- Hydrogen peroxide 20ml of 3%/L for 10-15 min.
- Furance (nitrofuran) 1.5mg/L for 1hour for 3 successive days.
- 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.

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.

Peduncle Disease

Cold water Disease, Fin Rot

Definition ***"Chronic bacterial disease affects mainly cool and cold water fishes, characterized by destruction of the caudal fin as well as muscular layer of the caudal peduncle ".***

Flexibater psychrophilus:

Etiology

Gram negative straight long rods, motile by gliding, non-spore forming, non acid fast, non capsulated and produce pigment. It grows on Ordal's or Cytophaga agar media giving yellow pigmented colonies. Generally, grow well at 4-24° C (cold water), however, some strains are reported to grow at 20-32° C (warm water).

From biochemical pathogenicity point of view the organism produces proteolytic enzymes.

Susceptibility

All cold and cool water fish species are susceptible particularly young and cultured ones. (e. g. Salmonids, Ayo, carp).

Aquarium fishes are susceptible to warm water strains.

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.
- **Low temperature.**
- Rough handling especially during transportation.

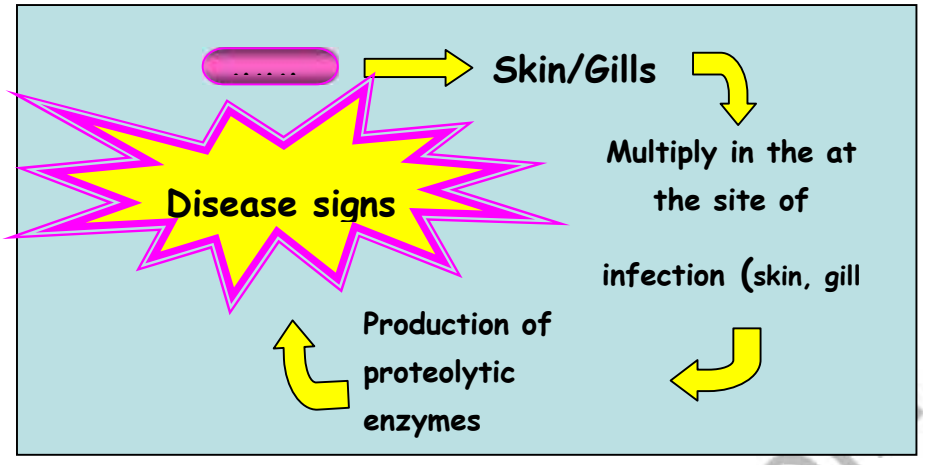
Mode of infection

Mainly through injuries of skin and/or gills.

Transmission & 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.

Pathogenesis



The severity of the infection depends upon the severity of the fish injuries and virulence of the bacterial strain as well.

Disease signs

- White to gray line start along the margin of the caudal fin and gradually extend towards the base of the fin, caudal peduncle and so on……
- Destruction of the caudal fin, which usually extend to include muscles of the caudal peduncle.

- In sever cases exposure of the vertebral column may occur.

Diagnosis

- I. Case history

II. The disease signs

III. The P. M. findings.

IV. Laboratory diagnosis:

Wet mount preparation: from Skin and gill scraping, and pieces of damaged fins directly examined under microscope.

Presence of **hay sticks** bacterial colonization is definitive.

- Smear stained with Gram.
- Isolation and identification:

  Ordal's or Cytophaga agar media and Sheep blood agar at 10-15 ºC for 24-36 hours giving rhizoid pale yellow pigmented colonies (orange to red color also recorded).
- **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.
- Cupper sulphate 40-500 mg/L for 1 min.
- Potassium permanganate 2mg/L indefinite.
- Hydrogen peroxide 20ml of 3%/L for 10-15 min.
- Furance (nitrofuran) 1.5mg/L for 1hour for 3 successive days.
- 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.

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**.**

Bacterial Gill Disease

BGD, Environmental Gill Disease

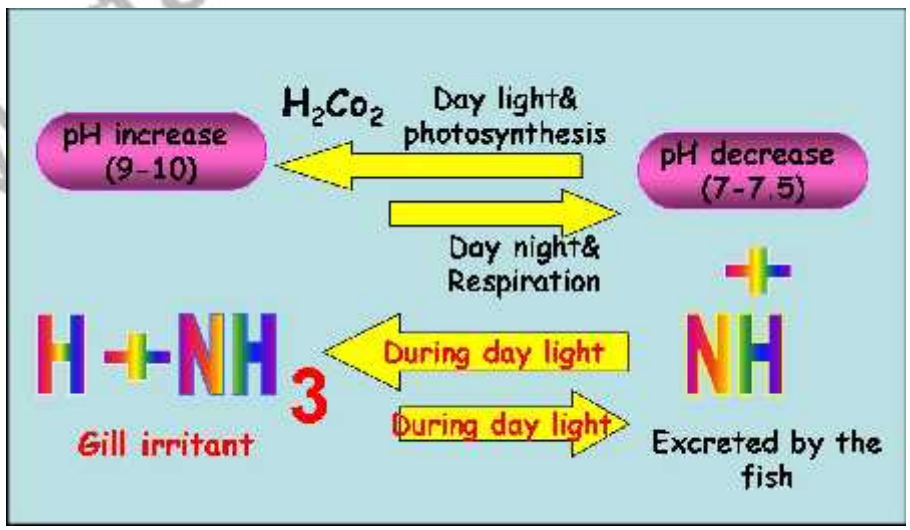
Definition

"Chronic to acute disease affects all fishes, characterized by different degree gill fusions accompanied with different degree of respiratory distress together with high morbidity and 25%-mortality".

Etiology & Pathogenesis

Flexibacteria, and other opportunistic pathogens such as Aeromonads, Pseudomonades, Flavobacteria …..etc, invade the gill epithelium resulting in epizooticof BGD.

Often a single bacterial specie or strain is involved during any epizootic.



Any shift of the pH (due to presence of aquatic plants) will shift ammoni-ammoium ions leading to gill irritation.

Susceptibility All fish species are susceptible particularly young and cultured

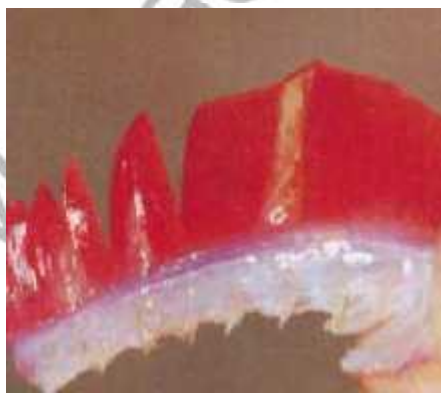
ones.

Predisposing causes (stressors)

- Overcrowding.
- Low dissolved oxygen.
- **Presence of large amount of organic matter.**
- **Nutritional deficiency specially** Pantothenic acid**.**
- Injuries of the skin or gill either by trauma or ectoparasites.
- **High temperature.**
- Rough handling especially during transportation.

Disease signs

- Fish stop feeding suddenly, swim sluggish, surfacing, accumulate near the water inlet.
- ***Increase mucous secretion by the gill.***
- In advanced cases, the gill lamellae clubbed, swollen and fused.
- Mortalities percentage is depends upon the severity of gill affections and/or super-invasion with opportunistic pathogen but never exceed 30%.



Diagnosis

- I. Case history

II. The disease signs

III. The P. M. findings.

IV. Laboratory diagnosis:

Wet mount preparation: from Skin and gill scraping, and pieces of damaged fins directly examined under microscope.

- Smear stained with Gram.
- Isolation and identification:

  Ordal's or Cytophaga agar media, R-S media, BHI.

  at 20-25 ºC for 24-36 hours.

- **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

- Increase water flow is indicative and may relieve the signs.
- If a pathogen is isolated antiseptic bathes are recommended.
- Cupper sulphate 40-500 mg/L for 1 min.
- Potassium permanganate 2mg/L indefinite.
- Hydrogen peroxide 20ml of 3%/L for 10-15 min.
- Furance (nitrofuran) 1.5mg/L for 1hour for 3 successive days.
- 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.

Control

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

- Avoid overcrowding.
- Supplementation with a good balanced ration.
- Avoid accumulation of high organic matter especially at hot

season.

- Control the rates of fertilization especially during summer season.
- Proper disposal of dead and dying fishes either by burning or burying.
- Partial change of the water during the summer season together with periodical examination of the phytoplankton concentrations using transparency disk.
- Proper disposal of infected fish if in small number.
- Proper drainage, drying, and disinfectant of the pond (quick lime 4 tone/acre.

Mycobacteriosis in Fish

Mycobacteriosis in fish is a disease caused by certain bacterial species within the genus *Mycobacterium*. There are many different species of *Mycobacterium* that cause diseases in animals. Some of these cause important diseases in humans. However, those species that cause Mycobacteriosis in fish are referred to as non-tuberculous mycobacteria and do not cause major disease in normal, healthy people.

Non-tuberculous mycobacteria are relatively common environmental bacteria. This means that they can live in the aquatic environment and do not require the presence of fish or fish tissue to survive. They have been isolated from drinking water supplies, swimming pools, coastal waters, and aquaculture facilities.

Mycobacteriosis is a disease of concern in fish for several reasons. First, it causes a chronic wasting condition with continuous low, to moderate, level mortality within the affected population, and can result in significant financial loss to fish culturists. Secondly, the disease is considered non-treatable once established in the fish host. Finally, *Mycobacterium*-infected fish do have the potential to cause disease in humans.

The genus *Mycobacterium* contains many species of bacteria that cause disease in mammals, birds and reptiles, as well as fish. Three species believed to account for most incidences of mycobacterial disease in fish are *M. marinum*, *M. fortuitum*, and *M. chelonae*. Of these, *M. marinum* has been reported in fish from both freshwater and marine tropical waters, and *M. fortuitum* in fish from tropical or temperate waters. *M. chelonae* has been associated with disease in hatchery-reared Pacific salmon.

Definition

"***Chronic progressive disease of freshwater, brackish and marine fishes as well as aquarium ones characterized by emaciation, unilateral or bilateral exophthalmia together with stunting of growth rate***".

Etiology

<u>Mycobacterium marinum (in marine fishes) & Mycobacterium fortuitum and M. chelonae</u> (in fresh and brackish water)

Gram positive straight long rods, non motile, non-spore forming, acid fast, non capsulated and doesn't produce pigment unless grow on special medium.

The organism is strictly aerobic. It grows on only a few media Dorset's medium, glycerol agar, Lowenstein's medium, Loeffer's medium and CGY medium (casein glucose yeast extract). Grow well at 25° C and the growth start to appear at least after 5 days (may take a few weeks).

Susceptibility

All fish species are susceptible and involved in the maintenance and transmission of the disease.

Freshwater fishes (e. g. Salmonids, eels, tilapia, carp,barbs). Brakish water such as mullet and rook fish. Marine fish such as sea bream, yellowtail, amberjack and flounder.

Mycobacteria cause disease in fish were reported as an important zoonotic organism as it cause a skin infection in people that is referred to as:

<u>"fish handler's disease" or "fish tank granuloma."</u>

Predisposing Causes

- Increase of temperature above 30 º C.
- Overcrowding.
- Low dissolved oxygen.
- Presence of large amount of organic matter.

(stressors)

- 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.

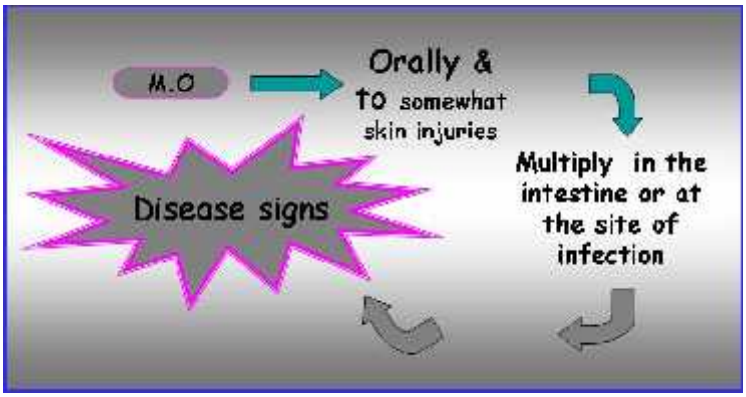
Transmission

Epidemiology, which is the study of the prevalence and spread of the disease in a population, is poorly understood for mycobacteriosis in fish. The most common method of transmission is assumed to be ingestion of infected material. This can occur if fish are fed fish products that have not been thoroughly cooked, or if an infected fish dies and is consumed by other fish in the population. Fish may also contract the infection through open wounds in their skin if the number of bacteria in the environment is high enough or if the fish has a poorly operating immune system. It is suspected that vertical transmission (transmission from parent to offspring) may occur through egg or sperm products. If this is the case, maintenance of mycobacteria-infected broodstock would be totally unacceptable for fish culturists.

Source of infection

- Shaded microorganisms from infected fishes and/or other infected aquatic animals.

Pathogenesis



Disease signs

- **Fish become listless and lethargic.**
- **Suspend their head down and maintain in this position**

for long time.

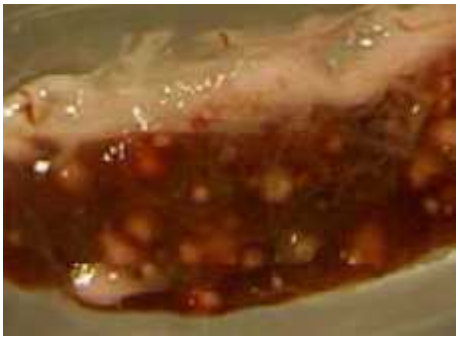
- **Emaciation and inflammation of the skin.**

- **Unilateral or bilateral exophthalmia “pop-eye”**

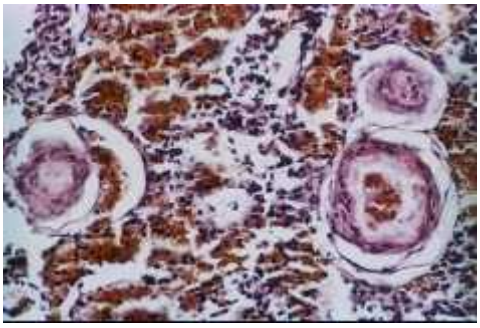
- **Open lesions, or ulceration**

Postmortem Findings

- **Internally, there were gray-white granulomas (nodules) develop in the liver, kidney, spleen, heart, and muscles.**
- **Enlarged spleen and kidney.**



Microscopic Pathology



- **Sever edema, infiltration of lymphocytes and**

macrophages in the periorbital tissue together with necrosis of the center of the lesion.

- **There may be caseation in the center surrounded with inflammatory cells and fibrous Tissue.**

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.

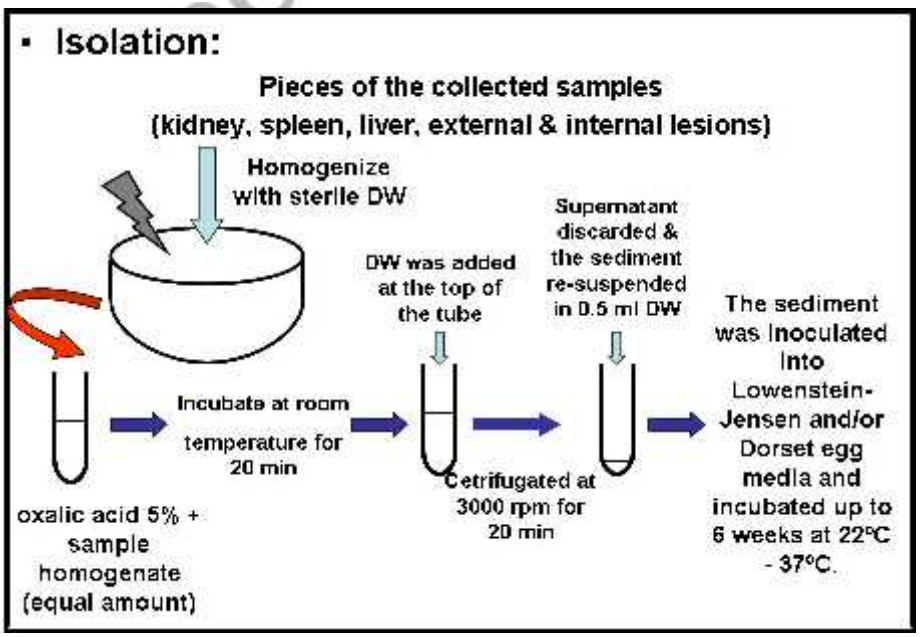
II. The *disease signs* (as mentioned above).

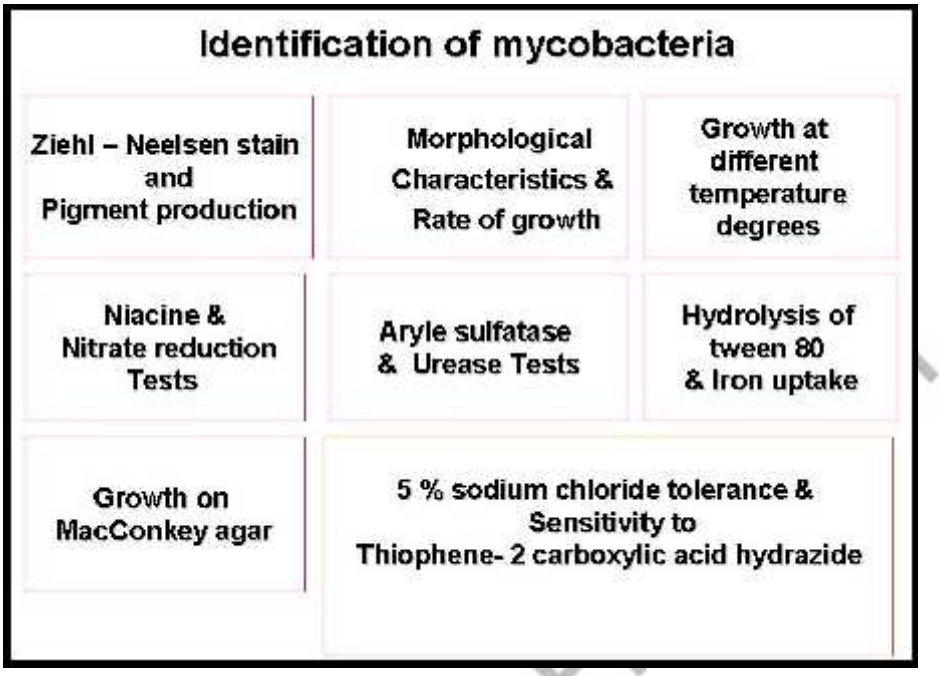
III. P. M. findings.

IV. *Laboratory diagnosis*:

- *Samples*: kidney, liver, spleen, , muscles especially the lesions (nodules)
- Squash smear from the organs stained with **Zeihl – Neelsen stain**
- *Histopathological findings* (as mentioned above).

- Isolation and identification:





Therapy & Control

Mycobacterial infections of fish should be considered non-treatable. Therefore, it is best to destroy infected stocks and thoroughly disinfect facilities before restocking.

Following depopulation, the entire system, especially the filter bed, must be thoroughly disinfected with a mycobactericidal product.

Chlorine at the rate of 10,000 ppm has been reported necessary to kill mycobacteria. Mycobacteria are sensitive to 60-85% alcohol.

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, formalized whole cultured or DNA vaccine.