II-Diseases caused by flagellates:-

II- 1-Diseases caused by ectoparasitic flagellates:-

II-1-1- Ichthyobodoosis (Costiosis):

-This disease is caused by excessive parasitism with one or more species of Ichthyobodo (Costia).

-It is characterized by high mortalities especially in young fish with respiratory distress and skin slime.

II-1-1- A-Etiology:

-There are 2 species of Ichthyobodo, *I. necatrix* (*Costia necatrix*) which is common allover the world and *I. pyriformis* which presents only in North America.

-*I. necatrix* is larger than *I. pyriformis*.

-They are facultative parasites, parasitize gills, fins& skin of fresh water fish, but they are primarily gill parasites.

-The free swimming stage (trophozoite) is ovoid to spherical in shape.

-It has 1 pair of posteriorly directed axostyles and 1 pair of freely moving flagella.

-One of the two flagella is longer than the other causing characteristic flickering movement.

-They have also, a small contractile vacuole and a rounded vesicular nucleus.

-The parasitic stage is oval in shape and resemble the free living stage in its structure and attach to host by its pointed anterior end after loosing its flagella.

II-1-1- B-Epizootiology:

-*Icthyobodo necatrix* is worldwide in distribution while *I. pyriformis* occurs only in North America.

-They parasitize freshwater fish.

-Cultured & aquarium fish are more susceptible.

-Younger fish are more susceptible than older ones.

-Malnourished and/or young fish are the most susceptible individuals.

-Overcrowdness, malnutrition, water pollution and higher temperature predispose fish to disease outbreak i.e. disease spread in spring and summer seasons.

-Disease affects any fish species because the causative agent is not host specific.

-The parasite has a free living stage and a parasitic stage.

-Parasitic stage is usually attached to tips of secondary gill lamellae and dorsal fins.

-Adverse conditions cause the organism to encyst either on the fish or freely in the water.

-Both parasitic and free swimming stages multiply by longitudinal binary fission.

-After conditions improvement, trophozoites derived from cysts should find host within short time or they will die.

-The optimum temperature for disease outbreak is 10-25ºC, the parasite encysts at 8ºC and dies above 30ºC.

II-1-1- C- Pathogenesis:

As in diseases caused by ectocommensal ciliates

II-1-1- D-Diagnosis:

II-1-1- D-1- Presumptive diagnosis:

II-1-1- D-1-a. Case history:

Season, stress exposure and/or affection of young fish more than adults.

II-1-1- D-1-b-Clinical signs:
-Anorexia, lethargy & skin darkening.
-Affected fish remain apart from others.
-Excessive mucous on gills and body surface (skin slime) is detected.
-Skin clouds up in some areas then skin comes off leaving bloody areas.
- Respiratory distress and flashing are also seen.

II-1-1- D-2- Definitive diagnosis:
-As previously described in ectocommensal ciliates.
-Costia species have approximate size of erythrocyte and they characterized by flickering movement in mucous & gill preparations.
-Interpretation of the microscopical examination as in ectocommensal ciliates.

II-1-1- E&F- Prevention & control:
As previously mentioned in ectocommensal ciliates.

II- 2-Diseases caused by opportunistic endoparasitic flagellates:

II- 2-1-Hexamitosis & Spironucleosis:
-They are chronic diseases caused by excessive numbers of endoparasitic flagellated protozoa of the hexamitids (diplomonads) in the alimentary tract of fishes or by systemic hexamitids (diplomonads) infection.
-Hexamitosis & spironucleosis affect wild, cultured and aquarium freshwater, brackish and marine fishes.
-Diseases cause considerable losses only when the host is adversely affected by stress factors.

II- 2-1-A-Etiology:
-These diseases caused by endoparasitic flagellates belonging to Hexamita & Spironucleus genera which are collectively called hexamitids (diplomonads).
-Hexamitids trophozoites are oval or pyriform in shape, tapering gradually toward the posterior end.
-These organisms are bilaterally symmetrical.
-They have 3 pairs of locomotory flagella extend anteriorly and a pair of posterior flagella i.e. recurrent one passes posteriorly through the body and emerges as a free flagellum.
-The anterior flagella arise from the blepharoplasts at the anterior end of axostyles, while the recurrent one arises from the axostyles at the posterior end of the parasite body.
-There is a pair of oval nuclei in Hexamita species or " S " shaped ones in Spironucleus species at the anterior end of the body.
- Each nucleus is surrounded by a delicate membrane.
-The hexamitids cysts are oval or spherical in shape and each containing a transparent membrane, four nuclei, axostyles and blepharoplasts.

II- 2-1-B- Epizootiology:
-Hexamitosis & spironucleosis are worldwide in distribution.
-They affect freshwater, brackish &marine fishes.
-Cultured fish are more susceptible than wild.
-Young fish in a "state of poor growth" are highly susceptible.
-They affect many hosts other than fish e.g. amphibians, reptiles &birds.
-There is somewhat a host specificity where those species which affect cyprinids or cichlids, do not infect salmonids.

-Salmon & trout culturists consider hexamitosis & spironucleosis are from important diseases that affecting those fish.
-Hexamitids are normal inhabitant of the alimentary canal of fish.
-The flagellated form (trophozoites) swims freely in the upper intestine & pyloric region and less commonly in the gall bladder & rectum.
- In healthy fish, only few number of the hexamita organisms invade the epithelial cells of the intestine or ceca to reproduce.

-The flagellated form reproduces by longitudinal binary fission, while the intracellular parasite (a quadrinucleate encysted stage) reproduces by schizogony (multiple fission to produce large number of progeny).
-When fish become unhealthy as a result of exposure to adverse factors e.g. change of diet, change of water temperature, overcrowding, low DO, considerable size variation between fish, malnutrition (especially vitamins), improper handling, concurrent nematode or acanthocephelans, septicemic infections infestation and long transportation, rapid reproduction of the flagellated form begin and increase in the number of organisms & invading of epithelial cells of the intestine & ceca occur.

-The generation time for the flagellated form is about 24 h & for the schizogony is 24-48 h.
-The life cycle is direct.
-Hexamitosis & spironucleosis are transmitted through oral route by ingestion of the food contaminated with cysts. Also, rectal infection is possible.
-In addition, hexamitosis and spironucleosis can be transmitted through encysted organisms in or on the egg i.e. it is egg born disease.
-Cysts are quite resistant and can survive outside of the host for several weeks.
-Food contaminated with the flagellated forms can not transmit the diseases because they are destroyed by the extremely high acidity of the stomach of most fish (pH 1-2.5).
- Amphibians, birds and reptiles may be sources of infection as well as diseased fish, contaminated water, food and fish eggs.

II- 2-1-C- Pathogenesis:

-Hexamitids feed normally on intestinal bacteria and when parasites numbers become high, they interfere with host nutrition by competing with fish for essential nutrients and/or cause structural & functional damage of the intestinal epithelium.
-The trophozoites become in an intimate association with the intestinal epithelium causing obstruction between the lumen and the microvilli so, inhibit absorption of the digested food.
-Also, they share cytoplasm with host cell i.e. feed directly on the cytoplasm of host cell.
-They can escape from the host's immune response by having an outside surface that mimics the glycocalyx covering of the microvilli.

-In systemic infections, trophozoites invade the intestinal epithelium and dissemination of the infection to the blood & internal organs occur.
-Necrotic changes which accompany septicemia or nematodes and acanthocephalans infestations help the access of the hexamitids to the blood and other internal organs.
-The affected internal organs are liver, kidney, spleen, heart, eyes, brain, gall bladder, musculature, abdominal cavity & mesentery.

II- 2-1-D Diagnosis:
II- 2-1-D-1- Presumptive diagnosis:
II- 2-1-D-1-a- Case history:

Stress exposure and poor growth.

II- 2-1-D-1-b-Clinical signs:

-In intestinal form:-

- lethargy
- Anorexia
- The diseased fish seek the sides or corners of its holding facility with inability to maintain a place in the flowing water, so they are swept downstream toward the lower screen area of the tank i.e. drainage opening or water outlet.
- Dark coloration.
- Emaciation (fish head appear relatively larger than the body which become extremely slender indicating poor growth).
- Pale stringy feces & red vent are also seen.
- Mortalities may be moderate to severe.

-In systemic form:-

- Weakness
- Poor growth
- Hole like lesions on the body especially head which produce yellow cheesy strings of mucous (hole in the head disease).
- Bilaterally ulcers arise from large erosions in the cranial cartilage.
- Abdominal distension and exophthalmia are seen.

II- 2-1-D-1-c- Post-mortem lesions:

-In intestinal form:-

- The gills are light in color with general anemia & pale liver.
- The gut is usually free of food or feces, but filled with yellow & watery or jelly like content with excess mucous.

-In systemic form:-

- Whitish nodules in the mottled liver.
- petechial hemorrhages in the enlarged kidney and spleen.
- Brown abscesses in the caudal musculature.
- Serosanguinus ascetic fluids are also seen.

II- 2-1-D-2- Definitive diagnosis:

Samples for hexamitids diagnosis should be collected from moribund fish.

II- 2-1-D-2-a-Microscopical examination of wet mount preparations from intestinal smears (at various sites along the intestinal tract), blood, gall bladder and internal organs imprints:

- By light microscope, the flagella can not be seen and the live organisms appear like rapidly moving lentil- shaped objects.

- The severity of infection could be determined through the count of hexamitids organisms by low-power /microscopic field as follows:

No organisms seen in the internal organs imprints, indicate no systemic infection

No organisms seen in several microscopic fields, indicate no intestinal infection

1-5 organisms / microscopic field indicate slight infection.
5-15 " / " " " moderate "
15-30 " / " " " marked "
Over 30 " / " " " severe "

II- 2-1-D-2-b-Histopathological examination:-

- Intestinal & cecal tissues can be examined histologically for detection of the intracellular encysted stages and the hexamitids trophozoites in the intestinal lumen & less frequently in the mucosal epithelium or in the internal organs & surface lesions.

II- 2-1-E- Prevention:

- keeping fish under adequate environmental conditions.
- Proper nutrition.
- Quarantine of newly purchased fish.
- Keeping wild fish, frogs, reptiles &birds away from cultured fish
-Presence of different sized fish in the same pond should be avoided.
- Drainage, drying and disinfection before fish stocking.

II- 2-1-F- Control:

-In case of slight or moderate hexamitids infections, no therapy is used, but control must be done by good management and nutrition.
- In case of marked or extremely severe hexamitids infections, do the following:
- Hygienic disposal of dead & infected fish.
-Control of the amphibians, reptiles and birds are essential.
- water change and increase water flow.
-Two types of therapeutic compounds used to control marked or extremely severe cases of hexamitosis & spironucleosis.

- Purgatives such as Magnesium sulphate (Epsom salt) can remove the flagellated and encysted parasites from the gut by mechanical means i.e. causes peristalsis & mechanically removing the organism through fecal discharge.
- Other drugs can kill the organisms in situ as Arsenical compounds (carbarsone) and Mercurial compounds (calomel). These drugs should not be given for food fish due to accumulation of the residues in fish tissues.

-The diet drug mixture is usually fed for 3 days, then fish fed a non-medicated diet for other 3 days & sample of treated fish should be examined.
- More three days of drug diet therapy are indicated if the infection has not been controlled.
-The reason for this regime of therapy is to give a time for all stages of the organism undergoing schizogony to be completed & flagellated stages released into the lumen of the intestine or ceca.
-Medicated ration should be used as soon as possible because fish in late stage of the diseases are off food.
-
-Drainage, drying and disinfection before restocking.

III- Diseases caused by obligatory endoparasitic Apicomplexans:

-They are characterized by production of simple resistant spores and/or oocysts.

-Apicomplexans are protozoa having a special cell organelle called the apical complex.

-This organelle facilitate the invasion of host cell and seen only by electron microscope.

-They have no cilia or flagella except occasionally on male gamete.

-Fish apicomplexans are endoparasites, infect intestinal & cecal epithelial cells i.e. intestinal parasite and internal organs e.g. spleen, liver, kidney, gonads and swim bladder i.e. non intestinal parasite.

-The intestinal and non intestinal species cause piscine coccidiosis.

III-1-Piscine coccidiosis:

-It is fish disease caused by members of genus Eimeria.

-Infection with low number of species of the genus Eimeria has harmless effect, but disease condition is associated with excessive number of them.

-It is characterized by poor growth, emaciation and mortalities in badly affected cases.

III-1-A- Etiology:

-Species of intracellular apicomplexan belonging to the genus Eimeria.

-They parasitize the intestinal & cecal epithelium mainly and few species infect liver, kidney, gonads, spleen, swim bladder and other organs.

-The only life stage which present outside the host is the oocyst or sporocyst (which derived from the oocyst).

-Each oocyst of fish coccidian contain 4 sporocysts and each sporocyst contain 2 sporozoites.

-The oocyst has a sensitive thin cuticular double wall where there is no need for thick wall because there is no desiccation in aquatic environment.

-The sporocysts are round, elliptical or oval in shape and its wall is thin but more resistant than that of the oocyst.

-The sporozoites are banana, sausage or comma-shaped and lie in head to tail presentation in sporocysts.

III-1-B- Epizootiology:

-Fish coccidia is worldwide in distribution.

-It affects freshwater, brackish & marine fishes either cultured or wild.

-It has less host specificity than mammalian coccidian i.e. fish coccidia infect closely related host species of the same genus.

-Fish coccidia have direct life cycle i.e. need one host.

-The infective stages of coccidiosis is the sporozoite in the sporocyst or oocyst.

-Sporocysts are released from the oocysts either inside the host or soon after excretion with faeces in case of intestinal species and after fish death in case of non intestinal species.

-The infection may be direct through ingestion of the infective stage or indirect through ingestion of paratenic host (reservoirs in which sporozoites do not undergo development) as tubifex or crustaceans.

- The invasion of the host cells by sporozoites occur, then merozoites are liberated and reproduce sexually and asexually and finally large numbers of oocysts are formed.

The developmental stages of the fish coccidia is divided into merogony, gamogony & sporogony.

- Oocysts are passed from host with faeces in intestinal species. Sporulation may take place within or outside the fish. In non intestinal species, sporulation occur within the host and sporulated oocysts liberated after host death.

-Most fish coccidian species produce oocysts continuously throughout the year but there are some species produce oocysts in spring only.
-The oocysts and sporocysts are resistant and can remain viable for long periods until swallowed by new host.
-The sources of infection are diseased fish, carriers, contaminated food and water.

III-1-C- Pathogenesis:

-The damage to fish tissues depends upon the intensity of coccidian infection.
-In intestinal species, the infection cause partial or complete destruction of the intestinal mucosa & submucosa where mature oocysts & free sporocysts were passed out in faeces with necrotic tissue i.e. host cell reminant.
-The infection was aggravated by inflammation "chronic enteritis" which characterized by numerous yellow bodies and inflammatory & necrotic cells in the lamina propria of the gut.
-Yellow body consists of 2-3 oocysts with necrotic host cell, mucoid substances, ferrous ions, cytoplasm of the host cell and lipofuscin which derived from degenerating cell membrane.
-The oocysts are rejected & get into the intestinal lumen as a result of secondary damage of epithelial layer.
-The unrejected oocyst are pushed in the subepithelial layer &became surrounded by connective tissue capsule & gradually died
-In non intestinal species, the fish damage depends upon the intensity of coccidian infection as well as the involved organ.
-Death may occur in case of heavy infection of vital organ.

III-1- D- Diagnosis:

III-1-D-1- Presumptive diagnosis:

III-1-D-1-a- Case history:

Poor growth and emaciation in spite of feeding well-balanced diet.

III-1-D-1-b- Clinical signs:

-Anorexia
-lethargy
-The affected fish group in certain area of the pond, float in the water with their head downward.
-Difficult swimming indicates swim bladder infection.
-Emaciation
-Poor growth.
-Long mucous sheath containing masses of oocysts protrude from the vent.

III-1-D-1-C-Post-mortem lesions:

-White blisters in size of lentil found on the intestinal wall or spleen or liver or kidney.
-Swollen intestine with reddened intestinal wall lined with thick viscous mucous (it is derived from plasm of the injured cells than from secreting activity).

-Faeces is covered with thick, sometimes reddish mucus layer.
-Thickening of the swim bladder can also be seen.

III-1-D-2- Definitive diagnosis:

III-1-D-2-a- Microscopical examination:

-The oocysts can be detected by microscopical examination (20-400 X) of the fecal samples, intestinal scrapings, and internal organs (using sucrose concentration and flotation technique).

-The oocysts are often distinguished only after sporulation, so, unsporulated oocysts can be seen after sporulation induction by keeping the suspected samples in the tap water in petridish or watch glass (specific diagnostic index)**.**

III-1-D-2-b- Histopathological examination:

-Histological preparations of intestine or internal organs are examined for detection of developmental stages of the coccidia inside the epithelial cells of the intestine or internal organs.

III-1-E- Prevention:

-Quarantine of the newly purchased fish
-Water filtration to avoid access of infective stage to cultured fishes.
- Drainage, drying and disinfection of the pond with quick lime before fish stocking.

III-1-F- Control:

-There is no therapeutic procedures known for piscine coccidiosis, however, sulfonamide coccidiostates used for higher vertebrates may be acceptable for therapy.
-Furazolidone in the food for 3 days or Furanace in the food for 6 days can be used.
-Hygienic disposal of dead fish.
- Drainage, drying and disinfection of the pond before fish restocking.

IV- Diseases caused by endoparasitic obligatory cnidosporans:

-They are "endoparasitic protozoa" parasitizing various tissues of the fish body either intracellularly or intercellularly.

-They are classified into two major groups, the **microsporidia** and **myxosporidia**.

-Microsporidia spores have sporoplasm and 1 polar capsule which contain a coiled polar tube (filament).

-Fish microsporidians are species of the genera, Pleistophora, Nosema, Glugea, Loma and Ichthyosporidium. **-Myxosporidia spores** have sporoplasm and two polar capsules, each one containing a coiled polar tube (filament).

-Fish myxosporidians are species of the genera Myxosoma, Henneguya and Myxobolus.

-Diseases caused by microsporidians and myxosporidians are **microsporidiosis** and **henneguyosis** and **whirling disease** respectively.

IV- 1- Diseases caused by microsporidians:

IV- 1- A. Microsporidiosis:

Definition:-

-It is a protozoal disease affects freshwater, brackish and marine fishes especially young fishes.

-The diseased fishes showed "white spots or nodules" in various parts of the fish body.

- Badly affected fishes become unfit for human consumption as musculature become jelly like in consistency.

-Fecundity of the infected fish is reduced.

-Mortalities occur "only" in heavy infection of vital organ.

IV- 1- A. a. Etiology:

-Different microsporidian species of genera Glugea, Pleistophora, Nosema, Loma and Ichthyosporidium.

-They are "intracellular protozoan" of tremendous reproductive capacity for production of large numbers of spores.

-The microsporidian spores are the smallest protozoal spores.

-Spores are ellipsoidal or egg shaped & the spore wall consists of a thin outer proteinaceaus layer (exospore) and a thick inner chitinous layer (endospore).

-The thick walled spore contains a sporoplasm, single or paired nucleus and coiled polar tube (polar filament).

-The tube serves for the injection of the sporoplasm into the host cell.

-This tube is inserted into the base of mushroom like polar cap at the anterior end of the spore.

-The polar cap is seen as red granule or dot under light microscope on staining with periodic acid- Schiff (PAS) as it is polysaccharides in nature.

-There is a large vacuole at the posterior end of the microsporidians spores.

IV- 1- A. b. Epizootiology:

-It is worldwide in distribution.

-It affects freshwater fish, brackish and marine fish either wild or cultured.

-Young fishes (fry and fingerlings) are more susceptible i.e. this disease spreads in the hatcheries.

-Microsporidiosis affects "a variety of hosts" other than fish because microsporidians are not host specific.

-The life cycle is direct i.e. need only one host.

-This disease is transmitted through ingestion of the microsporidian spores (infective stage) as contaminants of food or water.

-Autoinfection of the same host also occur.

-Microsporidian spores are resistant to environmental factors (can live for 1 year in water at 40ºC).

-Sources of infection are diseased fish, carriers, contaminated objects, food, water, reptiles, amphibians and aquatic birds.

IV- 1- A. c. Pathogenesis:

-After ingestion of the spore, in the intestinal tract, the polar tube emerge & pierce any host cell within its reach to inject the sporoplasm into it.

-The sporoplasm can develop directly in the epithelial cell of the intestine or more commonly it finds its way and enters a macrophage and thus reaches a final specific site in the body.

-Microsporidian spores are highly cell specific e.g. connective tissue cells, myocytes, neurocytes & so on.

-The sporoplasm reproduce by merogony (asexual reproduction) and sporogony (division for spore formation) until tremendous number of spores are produced within the host cell.

-In asexual reproduction, a great number of merozoites are produced.

-In sexual reproduction, spores are formed.

-The infection spreads within the infected tissue through transmission of meronts from one cell to another.

-The large number of spores enlarge the host cell and they remain in the hypertrophic cells (xenoma) of the host until death of the fish, or rupture of the cyst either internally or externally.

-After fish death and tissue disintegration, spore releasing occur.

-Autoinfection occurs when cysts rupture internally, while spores released in the water when cysts which present in the skin or musculature open.

-The type of the infected cell is difficult to recognize post-transformation to xenoma.

-In the aged xenomas, the cytoplasm and fragmented host cell nucleus disappear and xenoma becomes a bag of spores, while the infected cell undergoes atrophy & necrosis.

-Xenomas cause pressure atrophy to the surrounding tissue.

-Host tissue reaction directed toward isolation and elimination of the parasite.

-The host tissue reacts by granulomatous inflammatory reaction and proliferation of connective tissue around the infected cells.

-The efficiency of the host tissue reaction depends upon physiological conditions of the host and environmental conditions.

- When the defense response is good and intensity of infection is low, the granuloma undergoes involution where the migratory phagocytic cells, macrophages & fibroblasts ingest & digest spores and consequently gradual repair of the tissue lesions occur but the original function of the organ is not restored.

-When the defense response is weak and intensity of infection is high especially in vital organ, mortality usually happens.

IV- 1- A. d. Diagnosis:

IV- 1- A. d. 1. Presumptive diagnosis:

IV- 1- A. d. 1. a. Case history:

-High mortalities among young fish i.e. fry and fingerlings.

- Appearance of characteristic clinical signs.

-History of contact between susceptible fish & source of infection

IV- 1- A. d. 1. b. Clinical signs:

- Visible white nodules or cysts or spots appear as a bulge on the body.

-Discolored or grayish spots on the skin in case of subcutaneous tissue affection.

-The parasitic nodules may cause integument or body deformities.

-Also, affected fish undergoes emaciation & poor growth, so the productivity of cultured fish is reduced.

-The fecundity of the infected fish is reduced due to infection of male & female gonads leading to what is called "parasitic castration".

-Mortalities of adults occur only in heavy infection of vital organs.

-High mortalities among young fish.

IV- 1- A. d. 1. c. P.M lesions

- Visible white nodules or tumor-like masses on different fish organs.

-In fish with high intensity of infection, fish flesh develops a jelly-like consistency (milky white & creamy in texture) so, fish become unfit for human consumption.

IV- 1- A. d. 2. Definitive diagnosis:

-By microscopical examination, for detection of microsporidian spores in fresh tissue mounts which is prepared from the suspected lesions (whitish nodules). They are identified by large posterior vacuole with uniform size & shape.

-On staining with PAS, polar cap at the spore apex stains as intense red dot.

-The polar tube can be expelled by 2 % hydrogen peroxide (specific diagnostic index).

-In tissue sections, spores stain dark blue with Giemsa.

IV- 1- A. e. Prevention:

-Prevention is difficult due to direct transmission & longevity of spores.

-It depends upon avoiding contact between susceptible fish and sources of infection

-Quarantine of newly purchased fish.

-Obtaining fish diet from good source with healthy certificate.

-Water infiltration and covering water inlet with mesh to prevent entrance of wild fish and/or infective spores.

-Prevent fish-eating birds & aquatic animals from accessing the farm.

- Periodical drainage, drying & disinfection before fish stocking.

IV-1-A.f. Control:-

There is no therapy for the disease, but the disease can be reduced or eliminated by the following measures.

-Quarantine and restriction of fish movement.

-Hygienic disposal of the dead or sacrificed fish by deep burring or incineration are effective.

-Drainage, drying & disinfection before restock are essential.

-Disinfection depends upon creation of an alkaline condition in fish pond by calcium hydroxide which partially activated the spore i.e. filament extrusion occur, so they can die by calcium oxide or calcium cyanide.

-Use of chlorine is not successful due to presence of large amounts of organic matter in the water which reduce availability of chlorine.

-Ultraviolet rays can be used in disinfection of nets, utensils & water supplies contaminated with spores.

IV- 2- Diseases caused by Myxosporidians:

IV- 2- A. Henneguyosis (Henneguyan disease, Milky flesh disease, Blister disease, Adipose fin disease):

-Many cases of henneguyosis go unnoticed.

-Among cultured fish, it is characterized by debilitation and heavy mortalities.

IV- 2- A. a. Etiology:

-The disease is caused by Henneguya species.

-The henneguya spores are oval or fusiform in shape, but a few are round.

-They have two long whip-like caudal processes giving them a spermatozoan like appearance.

-The measurement of the spores does not include the caudal process.

- They have sporoplasm & polar capsules, each one contains 1 coiled polar tube or polar filament.

IV- 2- A. b. Epizootiology:

-World wide in distribution.

-Henneguyosis is widely distributed in freshwater, brackish and marine fish.

-cultured fish are more susceptible.

-Some henneguya species appear to be host specific and others are tissue specific.

-As in microsporidiosis.

IV- 2- A. c. Diagnosis:

IV- 2- A. c. 1. Presumptive diagnosis:

IV- 2- A. c. 1. a. Case history:

-Weight loss and high mortalities among cultured fish.

-Appearance of characteristic clinical signs.

-History of contact between susceptible fish & source of infection.

IV- 2- A. c. 1. b. Clinical signs and P.M. lesions:

- Visible or microscopic "opaque masses" in various parts of fish body e.g. skin, fins, gills intestine, liver, kidney, spleen, heart musculature, gall bladder and others.

-Weight loss.

-The fatality of the henneguya infection depends on the vitality of the organ involved.

IV- 2- A. c. 2. Definitive diagnosis:

-By detection of henneguya spores in microscopically examined wet mount preparations from the suspected lesions.

IV- 2- A. d& e. Prevention& control:-

As in microsporidiosis

IV- 2- B. Whirling disease (Black tail disease):

-It is chronic, debilitating and highly infectious disease of salmonids (appears to be specific for salmonids).

-The nomenclature of the disease by whirling disease describes the peculiar swimming activity of the diseased fish.

-The other name, black tail disease describes a characteristic clinical sign of the disease.

IV- 2- B. a. Etiology:

-The disease is caused by *Myxosoma cerebralis* spores.

-The myxosoma spores are oval or nearly circular in shape, each one contains two pyriform polar capsules.

-The wall of polar capsule consists of 2 layers, the inner layer is chitinous and the outer is proteinaceaus.

-A single barely discernible polar filament is coiled inside each of the polar capsules.

-A single sporoplasm is present within the spore at the opposite end from the polar capsules with a nucleus and glycogen vacuole (it is aggregated glycogen particles).

-A pronounced suture around the rim of the spore holds the two valves of the spore together.

-There is a transient mucous envelope around the spore which increases the buoyancy of the spore after the release of the spore so enhance "spread" of spores in aquatic environment.

IV- 2- B. b. Epizootiology:

-All species of salmonids are susceptible.

-It has been also reported in several other fish types, but up till now it is not recorded in Egypt.

-Young fishes are more susceptible than older ones of the same species.

-Whirling disease is transmitted by ingestion of the infective aged *Myxosoma cerebralis* spores.

-Transmission through abrasions in external body surface is suspected especially in young fish e.g. fry where it was noticed that they do not take food yet but become infected if they are within infected environment.

-The causative myxosporidian has a tropism for cartilaginous tissue of its host.

-Most spores remain within the cartilaginous tissues or trapped in bone until the fish dies.

-Disease cannot be transmitted directly from fish to fish where spores must spend a period lasting from few weeks to 6 months depending on the temperature either in dead fishes or in the bottom mud or debris of the pond to become infective.

-Transmission to the very young fishes in which nearly all hard tissues are cartilage is frequent because cartilage is a fertile substrate for sporulation, thus young fishes are usually more susceptible & more available to demonstrate deformed bodies.

-Incubation period (period between exposure to infective spore and appearance of sings of the disease) ranges from 2 weeks to 2 months according to the temperature & number of spores involved.

-The sources of the spores are the dead fishes or those carriers which survive the early stage of the disease and become a source of spores when they die.

-Piscivorous birds or aquatic animals may be involved in transmission cycle of the disease where spores can be spread in their feces.

-The tubificid worm can act as a source of infection.

-There is no indication that the causative agent of this disease can be transmitted through the eggs of the infected fish.

Pathogensis:

-After swallowing of the infective aged *Myxosoma cerebralis* spores, alkaline intestinal environment causes polar filament to extrude (emerge).

-The polar filaments emerge from the spores, then bierce intestinal epithelial cells of the fish and inject sporoplasm in them.

- Sporoplasm within intestinal epithelial cells is probably swept away by the blood stream or possibly the lymphatic system to the cartilage at any place in the body.

-When sporoplasm enters the cartilage at any place in the body, becomes a trophozoite and begins to reproduce forming multinucleate trophozoite by plasmotomy.

-Consequently a great number of spores are produced within the cartilage.

-According to the site of spore localization, different clinical abnormalities are induced.

IV- 2- B. c. Diagnosis:

IV- 2- B. c. 1. Presumptive diagnosis:

IV- 2- B. c. 1. a. Case history:

Fish species, fish age and characteristic clinical signs.

IV- 2- B. c. 1. b. Clinical signs:

-They vary depending upon fish age, site of spore localization and severity of infection.

-Light infection will usually lead to subclinical cases and fishes will become carriers, carrying spores for its life.

-In more heavily infection of young fish

- When sporulation occurs within the skull, usually alters equilibrium, so, the fish will swim erratically or frantically in circles and consequently, the disease is called whirling disease.
- When sporulation occurs within posterior vertebrae, they will alter caudal pigmentation through damage or pressure on the sympathetic nerves controlling pigmentation leading to the development of the characteristic black tail.
- The disease is highly fatal in young fishes with morbidity and mortality rates up to 100 %.

-Fish surviving the early stages of the disease develop

- Deformity in the head as sunken areas above the eyes.
- Permanently bent or open mouth
- Retraction of the operculum
- Deformed caudal area e.g. scoliosis (lateral bending) & lordiosis (dorsoventral bending) as a result of curvature of the vertebral column.

IV- 2- B. c. 2. Definitive diagnosis:

-It is carried out through detection of spores in the cartilage or bone of the suspected cases by tissue digestion method.

-In fresh smears, spores can be demonstrated by fluorescent antibody technique and they are identified by large posterior vacuole with uniform size & shape.

-Spores appear dark blue in histological sections of the skull, gill arches and vertebrae on staining with Giemsa stain.

IV- 2- B. d & e. Prevention& control:-

As in microsporidiosis

Metazoal diseases

I) Crustacean diseases

I- 1- Lernaeosis

I- 1- A. Etiology

♣The disease is caused by the adult fertilized parasitic females of the lernaedis (species of the genus *Lernaea*).

♣The most important Lernaea species is *L. cyprinacea*.

The adult females are:-

a) Long, slender, rod-shaped, non segmented (worm-shape) and greenish white to greyish brown in color.

b) They are large enough to be seen by naked eye, ranges from 9-22 mm.

c) It has a small semispherical cephalothorax containing the mouth and two pairs of symmetrical horn-shaped chitinous appendages called the cephalic process.

d) The anterior pair is digitiform and the posterior pair is T-shaped situated at right angles to the body.

e) The adult Lernaea female is named "anchor worm" depending on the shape of the cephalic process and absence of segmentation.

f) The cephalothorax is followed by elongated neck and trunk.

g) Near the posterior end of the body, there are paired egg sacs containing very numerous eggs.

♣**Males of Lernaea species** are free living, very smaller than females and have copepoid appearance with relatively short life span.

I- 1- B. Epizootiology

♣The egg sacs are shed as the eggs mature.

♣The eggs hatch and nauplii break out of the egg sacs.

♣The nauplii molt to become metanauplii and metanauplii form the first copepodite stage.

♣The first copepodite (infective stage) must find a host within three days or it will perish.

♣The first copepodite stage on a host's skin or gills continues to metamorphose. The second, third, fourth and fifth copepodite stages are formed.

♣Fifth copepodite stage undergoes sexual differentiation forming male and female individuals. Copulation occurs at the sixth copepodite stage and after mating, male dies.

♣The fertilized female continues to metamorphose, increases rapidly in length and burrows into the skin or gills of the host and becomes adult on a fish host.

♣The adult female becomes completely immobile after attachment to the host.

♣The fertilized attached female develops paired egg sacs.

♣Lernaeosis is widely distributed in Asia, Europe, North America and some African countries including Egypt.

♣It affects freshwater fish in warmwater and cool environments.

♣Water temperature is important to the life cycle of fish lernaeids.

♣Lernaea species cannot complete the life cycle at 15ºC or lower, but they can overwinter as eggs or larvae or adult females embedded in host tissue.

♣Cultured fish is more susceptible than wild ones.

♣Lernaeids are not at all host specific.

♣Transmission of lernaeosis can be through water supply, infested fish, amphibians and feathers of birds.

♣Lernaeids are sensitive to drying, salinity and low pH.

I- 1- C. Pathogenesis

♣The Lernaea females are true ectoparasites attach to the external surface of the fish body through embedding their cephalic processes in host tissues. This assists in holding the parasite in place during the remainder of its life.

♣Lernaeids are not site specific, so the adult female is embedded in host tissues either in eyes, gills, opercula, fins, skin, lips, buccal cavity or other body surfaces.

♣Harmful effect of parasitic females are due to their **feeding habit and attachment method.**

♣The Lernaea females ingest destroyed cells, tissue fluids as well as absorb whole blood.

♣Lernaea lesions are usually the portals of entry for opportunistic pathogens which may lead to fish loss.

♣At the point of attachment, the skin shows a typical inflammatory reaction (hyperemic, swollen) with fibrotic capsule.

♣The attachment site may undergo ulceration, focal necrosis and loss of scales.

♣The wounds caused by such parasites often develop into deep ulcer or even develop into fistula, penetrating the visceral cavity resulting in peritonitis, pushing into the intestines, liver or even brain causing death.

♣Starvation and death may be happened in case of infestation of the buccal cavity with large number of the parasitic females.

♣In addition, blindness and respiratory distress may occur on infestation of eyes &gills respectively.

♣The larval stages of Lernaea species feed on superficial mucous and tissue debris.

♣On the other hand, it was found that some fish might develop immunity as recovered fish are often more resistant to reinfestation with lernaeosis.

I- 1- D. Diagnosis

I- 1- D. a. Presumptive diagnosis:

I- 1- D. a. 1. Case history:

- Behaviour changes and clinical abnormalities (swollen & hyperemic areas with or without presence of visible long, slender and rod shaped object on the body external surface) of cultured fish during warm periods of the year.
- History of contact between susceptible fish and source of infection.

I- 1- D. a. 2. Clinical abnormalities:

♣They vary depending upon: The affected organ ,Infestation intensity (number of the parasites/fish) ,Fish age.

♣**The most common behaviour changes and clinical signs are:**

-Off food, flashing, respiratory distress, nervous manifestation.

- Sliminess (increase in mucous due to irritation).
- scales loss
- Swollen hyperemic areas or tumor like masses and/or greenish white long slender rod-shaped objects with paired appendages on the external surface of the fish body.
- Open ulcerating and haemorrhagic wounds may be also detected.
- Exophthalmia or even blindness.
- Emaciation.

I- 1- D. b. Definitive diagnosis:

- By detection of the large adult females by the naked eyes or by microscope

-Identification of the lernaeids is based on morphology of the adult females.

I- 1- E. Prevention & control

♣Prevention is the best control for crustacean diseases of fish because:

a) Crustaceans parasites in the water system are difficult to remove as they consider one of the zooplankton components.

b) Adult stages of crustaceans are no more affected by chemical treatment than the host, so attempts to eliminate the parasites may also damage the host. However, earlier life stages of crustaceans are usually more susceptible to chemical therapy.

♣For prevention of crustacean diseases, the following management practices must be done:

1- Quarantining the newly purchased fish for at least two weeks at 30ºC.

2- Plants, water and live food should be kept without fish contact for not less than 4 days.

3- Avoiding contact between cultured fish and sources of infestation (wild fish, amphibians, & aquatic birds) is essential.

4- Water flow should be increased to wash early life stages away before they can find a host.

5- Introduction of the planktonic copepod (mesocyclops) or monosex tilapia "*Oreochromis niloticus*" in the water is effective for lernaeosis prevention as they act as predators & feed on free swimming larval stages of Lernaea.

6- Drainage and drying of ponds before receiving new fish stock is essential for destroying all stages of the crustacean parasites.

7- Fish protection through immunization is a novel strategy.

♣For controlling of lernaeosis, various chemicals can be used:

1- Sodium chloride is recommended for euryhaline fish (can tolerate high salt concentration which is required to kill the parasites).

*It is effective against the free-swimming larval stages, not against the attached females.

2- Potassium permanganate is the only chemical capable of killing the adult females.

*However, it has a very low safety margin and its sensitivity increases in young & small fish.

*Its efficiency is lowered by increasing of organic load in the water and in direct sun light.

3- There are some organophosphate insecticides as metriphonate and dipterex can be used for lernaeosis treatment in non food fish.

*These pesticides are forbidden in cultured food fish for the attendant problems of the toxicity of them to both fish stock and farm workers, persistence of chemical residues in the environment. In addition, crustacean parasites are able to develop resistance to these compounds.

♣Recently, there is an effort for searching for an effective and safe alternatives.

♣Pine crude resin and its Steamed oil derivative were satisfactory and considered safe and natural cheap products for control lernaeosis.

♣The trials to control Lernaea infestation were directed toward the use of colophony either crude resin or steamed oil at different concentrations.

♣Colophony is a resin obtained from Pinus species trees and consists of about 90% resin acid (abietic acid), with the remainder consisting of neutral substances, oxidized terpentines and minor quantities of esters and anhydrides. ♣A very important aspect to be taken into consideration is the higher availability of the resin, which is produced in large commercial scale.

♣The application of crude colophony and its steamed oil in vivo on naturally infested fish gave promising results. It should be under-lined that the steamed oil derivative of colophony had a role in the healing process of the Lernaea lesions as the treated fish became Lernaea free and the hemorrhagic nodules disappeared rapidly than the lesions of fish treated with crude colophony.

I- 2- Ergasilosis

I- 2- A. Etiology

♣The disease is caused by mature fertilized parasitic females of the Ergasilids (species of the genus *Ergasilus*).

♣The most important species are *Ergasilus latus, E. mugilis and E. sarsi*.

♣They are true ectoparasites infesting gills and called "gill maggots".

♣They are small crustaceans ranges from 0.7-2 mm, cyclopoid in shape (i.e. body narrowing posteriorly) and white to yellow in color.

♣The adult females have a dorsoventrally flattened cephalothorax and a single median eye spot toward the anterior end.

♣The head has two pairs of segmented antennae, the first one is small and setated, while the second is large, slender, hook-like, as long as total body length terminates with a sickle-shaped powerful curved spines or claws serving as clasping organs used for attachment and pierce or encircle the gill filaments.

♣The three segmented abdomen terminates with a pair of anal lamella (furca) and two elongated whitish egg sacs.

I- 2- B. Epizootiology

♣The disease is worldwide in distribution.

♣It affects freshwater and brackish water fish (can withstand salinity up to 21 ppt).

♣Cultured fish are more susceptible than wild.

♣Sluggish or slow swimmers or diseased fish (cestodiosis) are the most susceptible ones.

♣The disease spreads in spring and more in summer where the increase in water temperature is associated with high prevalence.

♣Females spend a large part of their life cycle as free-living.

♣Only fertilized females are parasitic.

♣Males are free swimmers, non parasitic at all and after copulation, they die.

♣The eggs are laid, hatch & giving rise to nauplii, metanauplii, five copepodite stages and adults. Sexual differentiation is evident in the fourth copepodite stage.

♣The fertilized females of the fifth copepodite stages are considered the infective stages that seek out their host.

♣Overcrowdness and direct contact with infested fish are the most important factors for disease transmission.

I- 2- C. Pathogenesis

♣The mature fertilized females randomly attach to fish, then find the way to the gills i.e. site specific.

♣The damage of the gills in the ergasillosis is attributed to the feeding and attachment behaviours of the causative crustacean.

♣The attached females feed on the whole blood, mucous and epithelial cells. Extra-buccal digestive secretions of ergasilids cause lysis of tissues.

♣The parasitic ergasilids females attach to the gill filament with its large second antenna.

♣Such sickle-shaped powerful claws pierce or encircle the gill filaments and thrusting into them.

♣ Hemorrhages, thrombosis, ischemia, necrosis, hyperplasia, fusion of the gill filaments and disturbance of gill circulation occur as a result of destructive activity of the parasite.

♣Consequently, gills aquire the **marbling appearance** and affected fish show respiratory distress and high mortalities due to loss of gill surface which lead to reduction in oxygen supply as well as osmoregulatory failure.

♣In addition, the lesions caused by ergasilids become foci for secondary pathogens.

I- 2- D. Diagnosis
I- 2- D. a. Presumptive diagnosis:
I- 2- D. a. 1. Case history:

- High mortalities with respiratory distress in cultured fish in summer.
- History of contact between susceptible fish and source of infection.

I- 2- D. a. 2. Clinical signs:

- Anorexia
- Emaciation and respiratory distress are prominent in the diseased fish.
- Gills of the affected ones are frayed, slimy, nibbed with marbling appearance and have numerous white dots.

I- 2- D. b. Definitive diagnosis:

By microscopical examination of gill scrapings or suspected white dots. Identification of the ergasilids is based on morphology of the adult females.

I- 2- E. Prevention & control

♣As previously mentioned in lernaeosis.

♣Regarding chemical treatment, in addition to the mentioned chemicals, a mixture of **copper sulphate** and **ferric sulphate** in ratio of 5:2 can be used.

- It is effective in waters with high organic matter and at high temperatures.

I- 3- Argulosis (Fish lice)

I- 3- A. Etiology

♣The disease is caused by Argulids (species of the genus Argulus, fish lice).

♣The most important Argulus species are *Argulus Africans, A. japonicus, A. foliaceus.*

♣All larval stages until adulthood is reached as well as males & females cause the disease.

♣Argulus species are obligate ectoparasites on the external body surface of fish.

♣Both males and females are transparent, disc-shaped, have dorsoventrally flattened body, range from 5-15 mm (can be seen by naked eyes).

♣The anterior part constitutes the cephalothorax which is covered by dorsal carapace and there is two complex faceted eyes in the head.

♣The two maxillae modified into sucker-like organs and the second maxilla carries spines for attachment.

♣Also, the basal part of first and second antennae ends in a strong hook.

♣Other modified mouth parts form the stylet which is surrounded by a sheath and can be thrust out at will to pierce host tissue for feeding.

♣The stylet lies between the modified mandibles and slightly anterior to the eyes.

♣On the underside of the carapace, there are two laterally situated areas serving as respiratory organs to which four pairs of swimming legs constantly direct fresh water. By these legs, the parasite move in water & on host body.

♣The abdomen (called the caudal fin) is short& forming two rounded lobes i.e. medially notched.

♣There is no egg sacs on the females.

I- 3- B. Epizootiology

♣Argulosis is worldwide in distribution.

♣Freshwater fish, brackish water and marine fish are susceptible where fish lice can resist salinity up to 3-5 %.

♣Cultured fish are more susceptible than wild.

♣Smooth skinned fish are preferred than scaly ones.

♣This disease affects warm water fish when water temperature is high.

♣Argulus lives on the external surface of the host until adulthood is reached.

♣The fertilized female falls from the host for egg laying and males die soon after mating.

♣The females swim to aquatic plants and other submerged objects to which eggs are attached by a sticky mucous material.

♣The nauplius, metanauplius and first copepodite stages develop in the eggs.

♣After hatching, the metamorphic stages include the second through the seventh copepodite, sub adult and adult.

♣The second copepodite stage is the infective stage where it actively seeks a host. This stage has a body shape similar to the adult and if does not find the host within 2-3 days, it will die.

♣The disease is transmitted by direct and indirect contact. Sources of infestation include infested fish, contaminated water supply, living food ,amphibians, aquatic plants and contaminated objects.

♣Overcrowdness is the most important stress factor that favours disease transmission.

I- 3- C. Pathogenesis

♣The harmful effect of Argulids is attributed to feeding habit & method of attachment of the parasite.

♣The argulids during feeding pierce the host skin by the stylet and inject substances from the stylet glands into the underlying epidermis.

♣The injected material prevent the coagulation of blood and has cytolytic and paralyzing effects leading to severe inflammatory responses.

♣Fish lice feed only on the blood plasma, cannot feed on whole blood.

♣ As a result of their feeding activity, circular depressions, linear hemorrhages, ulcerations and scales loss occur.

♣The lesions of fish lice "parasite print" may provide a site for attacks by secondary pathogens which may lead to fish loss.

♣They can transmit some haemoflagellates through sucking of blood plasma.

♣The prolonged and strong attachment of the argulids cause mechanical damage to the host's skin.

♣Heavy argulids infestation can cause heavy mortalities especially in young fish due to the osmoregulatory failure and anemia.

I- 3- D. Diagnosis

I- 3- D. a. Presumptive diagnosis:

I- 3- D. a. 1. Case history:

- Cultured fish in warm water showed behaviour and clinical abnormalities.
- History of contact between susceptible fish and source of infection.

I- 3- D. a. 2. Clinical signs:

♣The affected fish appear

- Irritable & nervous with imbalanced swimming and flashing.

- Presence of circular red depressions on the manner of linear hemorrhages on the external body surface.
- Skin abraded areas, ulcerations, excess mucous or dry skin and loosened scales can be also detected.
- Transparent, broad, disc-shaped parasites can be seen by naked eyes.
- Argulids tend to present near the base of the fins. Also, they may occur in the walls of the oral & branchial cavities.

I- 3- D. b. Definitive diagnosis:

-By microscopical examination of the skin scraping or the suspected parasite.

-Identification of argulids is based on morphology of the adult individuals.

I- 3- E. Prevention & Control

♣In addition to the previously mentioned for prevention & controlling of lernaeosis:

-Submerged hard objects like stones, plants-etc. should be removed from the pond to avoid egg deposition.

-Introduction of mosquito fish (Gambosia), Anglefish, freshwater shrimp into fish ponds has good effect for prevention of this disease because they act as biological predator where they feed on the argulids' larvae.

-Immersing of wooden sticks in the pond will act as egg-trap because females will deposit their eggs on these sticks. Regular cleaning of these sticks will destroy great numbers of the argulids' eggs.

- Raising pH of water to 9.8 by using lime is useful for destruction of eggs.

II- Diseases caused by Helminthes:

II- 1- Trematodiosis:

-Diseases caused by bilaterally symmetrical dorsoventrally flattened worms (Trematodes)

II- I- A. a. Gill fluke disease,

(Dactylogyrosis, Cichlidogyrosis, Quadricanthosis).

II- 1- A. a. 1. Etiology:

-This disease is caused by species of the genera, Dactylogyrus, Cichlidogyrus and Quadricanthus.

-They are obligatory ectoparasites, parasitize gills primarily, but when increase in number, they may present on the skin.

-They are small (not more than 2.0 mm), but larger than gyrodactylus.

-They have 7 pairs of marginal hooks & 1 pair of median hooks on the opishaptor.

-Their anterior end (prohaptor) is divided into 4 cephalic lobes, having sticky & adhesive organs (cephalic glands) which provide an additional grip on the substrate during locomotion & feeding.

-On the anterior fourth of the body, there are 2 - 4 pigments (black eye spots).

-They have fused intestinal limbs.

-They are hermaphroditic, having both male and female genital organs & oviparous(no uterus).

-The ovaries are located in the front of the unpaired testes.

-They are dark because there is dense granular strands of yolk glands (vitellaria) occupying the whole body of the worm.

II- 1- A. a. 2. Epizootiology:

-They are worldwide in distribution, affect freshwater, brackish & marine fishes.

-Cultured fish are more affected than wild.

-This disease flourishes up with increasing temperature in summer and late spring.

-Young fishes are highly susceptible as age resistance occurs.

-Their presence in a farm is an indication of "poor husbandry".

-This disease is stress-related, where overcrowdness, poor water quality, malnutrition & physiological alteration of the host are responsible for increasing the intensity of infestation.

-This can be attributed to acceleration in the rate of worm reproduction on exposure to adverse environmental conditions especially the decrease in dissolved oxygen.

- Their presence in wild fish is an indication of pollution i.e. biological indicator for environmental pollution.

-Adults produce eggs, which are released, swept away by the water, hatch & free swimming larvae are liberated in the water searching for new host.

-If the infective stage don't find its host in 6 – 24 h., they will die.

-They have higher prevalence & lower infestation intensity than gyrodactylids.

-Gill flukes are transmitted by direct & indirect contact.

Pathogenesis:-

-They feed on tissue debris (mucous and epithelial cells)

- Small number of monogenetic trematodes causes little damage to their host, but large number of them causes excessive trauma or opening portals for opportunistics which in turn damage the host

II- 1- A. a. 3. Diagnosis:

II- 1- A. a. 3. i. Presumptive diagnosis:

II- 1- A. a. 3. i. 1. Case history:

Stress exposure, season & behaviour changes.

II- 1- A. a. 3. i. 2. Clinical signs:

-Respiratory distress & the opercula appear somewhat opened.

II- 1- A. a. 3. i. 3. Gross lesions:

-Marbling appearance of gills (areas of thickened mucous, hyperplasia, petechial hemorrhages & necrosis).

II- 1- A. a. 3. ii. Definitive diagnosis:

-By microscopical examination of wet mount preparation of gill scrapings.

-Identification by morphological characters of gill flukes.

II- 1- A. b. Skin fluke disease (Gyrodactylosis):

II- 1- A. b. 1. Etiology:

-This disease is caused by species of genus, Gyrodactylus.

- They are obligatory ectoparasites, parasitize skin primarily, but when increase in number, they may present on the gills.

-They are small, rarely over 0.4-0.1 mm.

-They have 8 pairs of marginal hooks & 1 pair of median hooks on the opishaptor.

-The anterior end (prohaptor) is divided to 2 large conical projections with adhesive glands as in dactylogyrus species.

-There is no eye spots.

-The intestinal limbs are separated & terminated with blind sacs.

-They are hermaphroditic, having both male and female genital organs and viviparous having V-shaped uterus.

-The unpaired testes lie in the front of the ovary.

- They are transparent because there is no yolk glands (vitellaria) .

-They have a copulatory organ on the ventral side of the body.

II- 1- A. b. 2. Epizootiology:

-This disease is worldwide in distribution, affect freshwater, brackish & marine fish.

-Cultured fish are affected at low temperature (14-17ºC).

-This disease flourishes up with decreasing temperature in winter & early spring.

-Both young & adult fish are susceptible.

-Cultured fish are more susceptible than wild ones.

-Their presence in a farm is an indication of "poor husbandry".

-Overcrowdness, poor water quality, malnutrition & physiological alteration of the host are responsible for increasing the intensity of infestation.

-Their presence in wild fish is an indication of "pollution" i.e. biological indicator for environmental pollution.

-They are viviparous, larval flukes developing in the uterus, then released & grow to adult on the same host as its parent.

-They have lower prevalence & higher intensity of infestation than dactylogyrids.

-Skin flukes are transmitted by direct contact.

Pathogenesis:-

As previously described in dactylogyrosis

II- 1- A. b. 3. Diagnosis:

-As in gill fluke disease, except in clinical signs, diseased fish showed signs of skin irritation (flashing).

-No gross lesions.

-Definitive diagnosis is by microscopical examination of wet mount preparation of skin scrapings.

-Identification by morphological characters of skin flukes.

II- 1- A. a&b 4. Prevention:

For dactylogyrosis and gyrodactylosis prevention;

- Quarantining the newly purchased fish

-Fish should kept under adequate environmental conditions.

-Keeping young fish away from adult.

-Prevention of wild fish from access to fish pond and strict hygienic measures are necessary.

- Water filteration.

- Drainage, drying and disinfection of ponds before receiving new fish stock is essential.

II- 1- A. a&b 5. Control:

-The best control procedures are careful management and balanced nutrition.

-Chemotherapy for these diseases is not usually satisfactory unless the primary cause of the increased fluke population is found and elevated.

-A number of chemotherapeutics can be used for therapy of gyrodatylosis and dactylogyrosis:-

- Formalin ,malachite green ,malachite green and formalin, methylene blue , potassium permanganate and sodium chloride.

-Elimination of the monogeans is usually not possible, only reduction of its population occur.

II- 1- B. Diseases caused by digenetic trematodes (digeneans):

-Digenean trematodes have a complex life cycle with several larval generations and need more than one host to develop into the adult in its primary host.

-They either use fish as a primary host (adult stage present in the fish) or as intermediate host (larval stage i.e. free metacercarial or encysted metacercaria present in the fish).

-Larval stage may be found in eye (parasitic eye or eye cataract or eye opacity or cataracta parasitica) or in the fish

skin, fins, musculature &visceral organ (black spot disease) or in the post branchial region attached to the branchiostegal musculature(yellow grub disease).

-

II- 1- B. a. Diseases caused by adult digenetic trematodes:

II- 1- B. a. 1. Fish schistosomiosis (Sanguinicoliosis, blood fluke disease, blood worm disease):

II- 1- B. a. 1. i. Etiology:

-This disease is caused by adult digenetic trematode of Sanguinicola species.

-Their predilection seat is the "circulatory system" especially heart chambers & large blood vessels in the gills, kidney & liver.

-Adult worm is lanceolate-shaped & characterized by X shaped intestine, very long oesophagus & butterfly shaped ovaries.

II- 1- B. a. 1. ii. Epizootiology:

-This disease is worldwide in distribution

- It affects freshwater fish as clarias, tilapia & carp species.

-Adult worms lay their eggs (they are hat shaped, appear as a triangle) into the blood, then passed along to the gills, where hatch and miracidia penetrate gill epithelium and are released into the water.

-The active miracidia enter the snails where they metamorphosed to sporocyst or redia which asexually produce cercariae.

-The active free swimming cercariae seek their primary host where they penetrate their integument and reach the underlying tissue or fish swallowed snails carrying cercaria.

- At its predilection seat, cercariae develop to adult worm.

II- 1- B. a. 1. iii. Diagnosis:

II- 1- B. a. 1. iii. a . Presumptive diagnosis:

II- 1- B. a. 1. iii. a. 1. Case history:

Presence of snails, fish species and clinical abnormalities.

II- 1- B. a. 1. iii. a. 2. Clinical abnormalities & post mortem lesions:

-In acute form, there is gill necrosis (marbling appearance) due to thrombosis formation as a result of occlusion of branchial capillaries by fluke eggs & severe hemorrhage due to burst out of the gills capillaries as a result of miracidia release.

-This form usually causes respiratory distress, anemia, growth retardation & heavy mortalities among young fish.

-In chronic form, there is necrosis in the renal tissue due to glomerular occlusion with eggs with subsequent ascitis, exophthalmia & scales erection. This form usually affects the older fish.

II- 1- B. a. 1. iii. b. Definitive diagnosis:

-Identification of eggs & adult worms by microscopic examination of gill & renal tissue post staining with carmine.

II- 1- B. a. 1. iiii. Control and prevention:

-Under cultural condition, snails elimination is essential for prevention & control.

- Best control is pond management (drainage, drying & disinfection).
- Di-N-butyltin oxide for getting rid of adult worm (Fish take it for 3 days in the diet) can be used.

-Under natural conditions, prevention & control are difficult & unpractical because destruction of intermediate host (snail) may be impossible or will lead to destruction of the aquatic environment.

II- 1- B. b. Diseases caused by larvae of digenetic trematodes (free and encysted metacercaria):

II- 1- B. b. 1. Eye cataract & Black spot diseases:

Etiology:-

-The causative agents of these diseases are the free metacercaria in family diplostomatidae (eye cataract or eye opacity or cataracta parasitica) and the encysted metacercaria of digenetic trematodes of the families Strigeidae & Heterophidae (black spot disease).

Epizootiology:-

- They are worldwide in distribution.

-They affect all fish species especially fresh water & brackish water fish, mostly Clarias, Tilapia & Mugil species.

-All digenetic trematodes are hermaphroditic, having both male and female genital organs & oviparous.

-Eggs of digenetic trematodes passing out with feces or urine of the primary host either hatch into miracidia in the water or are eaten by the mollusk (1^{st} intermediate host).

-The hatched miracidia actively seek its 1^{st} intermediate host i.e. snail.

-In snail, the miracidia metamorphoses into sporocyst or redia which asexually produce cercariae.

-The cercariae shed into the water & actively seek the 2^{nd} intermediate host & penetrate its integument to underlying tissues or fish eat snail carrying cercariae.

-The cercariae develop into metacercariae in the 2^{nd} intermediate host then go to its predilection seat in 2^{nd} intermediate host, remain free or encysted & when infested fish is swallowed by a primary host, the life cycle is completed.

-The primary host of digenetic trematodes which use fish as 2^{nd} intermediate host usually are piscivorous birds as herons, loons, gulls. Etc., and piscivorous mammals (human, dogs and cats).

-The digenetic trematodes involving fish are found primarily among wild ranging fish.

-They affect cultured fish when conditions were acceptable for completion of the life cycle i.e. presence of snails, fish & final host.

Pathogenesis:-

-The metacercaria of family diplostomatidae, reach eye through blood and free metacercaria causing eye cataract (cataracta parasitica).

-The metacercaria of families strigeidae & heterophidae are encysted in the skin, fins, musculature (flesh) and other internal organs and melanin pigments is deposited around

them by the host in skin and fins giving rise to the condition known as "black spot disease".

- Little pathological changes are seen in the tissue containing metacercarial cysts except in hyper-infection of any organ, functional damage may occur when active tissue is displaced by the encysted parasite

Diagnosis:-

1. Case history:

Presence of snails, piscivorous birds, fish eating mammals &clinical abnormalities.

2. Clinical signs:

-The most detected clinical signs:-

- Poor growth
- Surfacing &sluggish swimming movement near pond sides
- Respiratory distress in case of gill affection
- High mortalities especially among young fish
- Visible white to yellow spots in the musculature & visceral organs.
- Visible black spots in the skin and fins
- Whitening of the lens of the eye (cataracts) which may lead to vision loss & consequently weight retardation due to inability of fish to see food.

Definitive diagnosis:-

-These diseases are diagnosed by detection of free or encysted metacercaria in different body organs & by its developing to a mature stage in a suitable definitive host, the digenetic trematodes are identified.

Prevention & control:-

-No chemotherapy can be used for control of the metacercarial stage in the fish & prevention & control as previously mentioned in Sanguinicoliosis.

II- 1- B. b. 2. Yellow grub disease:

Etiology:-

-It is caused by larvae i.e. encysted metacercaria (metacercarial cysts) of Clinostomatidae species (as Complinatum and Tilapiae).

-They are present as visible small & round yellow cysts in the form of bunches of grapes in the post branchial region attached to the branchiostegal musculature.

-They resist freezing & salinity.

-On liberation of metacercaria, they moved by gliding movement.

-Adult digenetic trematodes (Clinostomum) present in the throat of the aquatic birds & characterized by presence of 2 suckers (oral & ventral) with separated intestine, testis & ovary.

Epizootiology:-

- It is world wide in distribution.

-The disease affects wild freshwater fishes, mostly older ones.

- It spreads in lake Naser fish especially Tilapias (prevalence about 70 %).

- Final hosts of Clinostomatidae species are aquatic birds and crocodiles.

-This disease has zoonotic importance as it may transmit to humans causing laryngopharyngitis on consuming raw or ill cooked fish, so evisceration and removal of heads of infested fish should be done.

- In this disease, man act as an aberrant host i.e. host in whom the larvae don't develop.

Diagnosis:-

1. Case history:

Wild fish, presence of snails, aquatic birds and crocodiles and clinical abnormalities.

2. Clinical abnormalities & post mortem lesions:

- In affected fish, most common clinical signs are respiratory manifestation, dislocation of the operculum with relatively large head & growth retardation.

- The most common post-mortem lesions are presence of visible pease-like, small & large yellow cysts like a bunch of grapes, each one 3-5 mm in diameter attached to the branchiostegal musculature with haemorrhagic area.

-Atrophied gills or even necrotic changes also seen.

-Number of metacercarial cysts vary & may reach 45/fish.

3- Definitive diagnosis:-

-The disease is easily diagnosed where the metacercarial cysts are easily recognized by naked eyes.

-Cysts must be opened & dissected then compressed between 2 slides with addition of few saline drops & examine under the binocular microscope for confirmative diagnosis.

-The adult worms of Clinostomatidae species are identified by its developing to a mature stage in a suitable definitive host.

Prevention & control:-

-Prevention & control depend upon elimination of snails & final host under culture condition.

-No therapy for treatment of metacercarial cyst in fish can be used.

- Under natural condition, it is impossible and impractical especially in large water bodies where control methods may upset the entire aquatic environment.

-In Egypt, evisceration of affected fish is done before marketing because fish with visible yellow cysts are usually rejected by the consumer.

Fish nematodiosis

Heart worm disease:-

Etiology:-

- It is caused by the third stage larvae (larva migrans) of amplicaecum nematodes.
- They about 3 cm in length so, can be seen by naked eyes.
- They attached to the sinus venosus of the heart.
- On severe infestation, they present in branchial cavity or extend to the abdominal cavity
- The larva migrans are non capsulated in the tilapia, while they are encapsulated in predatory fish as mode of defense mechanism

Epizootiology:-

- Aquatic birds, water snakes & crocodiles act as final host, crustaceans act as first intermediate host and fish serve as second intermediate host

- This disease mostly spread among tilapia, Lates niloticus & catfish in lake Nasr.

- Its prevalence in tilapia is about 30-70 %, while is 10-30 % in predatory fish.

- Density of infestation (number of worms/fish) is about 5-35/fish.

- Prevalence increases with increasing the fish size.

Diagnosis:-

1. Presumptive diagnosis:

A- Case history:

Presence of final hosts, wild fish and clinical abnormalities.

B- Clinical signs:

1- Nervous manifestation (due to pain as a result of their attachment to the heart chambers).

2- Slight abdominal distention

3- Anemia.

4- Emaciation.

5- Reduced vitality.

C- Post- mortem lesions:

♣ Detection of visible long, cylindrical white to creamy larvae with redness at their anterior end, attached to the sinus venosus of the heart & extend into branchial or abdominal cavity.

♣ Dislocation, compression & ulceration of internal organs.

D- Definitive diagnosis:

♣Identification of the causative larvae after their fixation with 5 % formalin is essential.

Prevention:-

♣Under natural conditions prevention is impossible & unpractical.

♣Under cultural conditions:-

1- Quarantining the newly purchased fish
2- Prevention of crustaceans from access to fish ponds through filtration of water inflow.
3- Prevention of access of aquatic birds, water snakes and reptiles to fish pond.
4-Periodical drainage, drying & disinfection.

Control:

♣Under natural condition, control is impossible & unpractical.

♣Under cultural conditions:-

1-Elimination of water snakes & aquatic birds from fish farm.

2- Drainage, drying & disinfection for elimination of first intermediate host are essential before restocking.

4- No therapy for encysted or free larvae.

Leechiosis

It is metazoal parasitic disease of fish caused by aquatic leeches.

Etiology:-

♣ Aquatic leeches, belong to phylum Annelida (ring worm), class Hirudinea.

♣They have long, slender, segmented, flexible bodies.

♣ they have 2 suckers, oral sucker for feeding and posterior one for attachment to the external body surface (skin, fins & gills) of fish.

♣They are hermaphrodites & have complete digestive tract.

Epizootiology:-

♣They are worldwide in distribution.

♣They affect freshwater, brackish & marine fishes.

♣Crowded cultured fish are more susceptible than wild ones. ♣Young fish & those have reduced vitality are highly susceptible.

♣Some of Aquatic leeches have no host specificity, and others have some degree of host specificity.

♣They spread in summer.

♣They have direct life cycle.

- Adults produce cocoons which are deposited on the aquatic vegetations, rocks & snails.

- Juveniles leeches hatch from cocoons.

Pathogenesis

♣Both mature and immature leeches are blood sucking

♣They are temporary or semi-permenant parasites.

♣They leave the host after one or successive blood meals.

♣They are more active in dark than in light.

♣Aquatic leeches feed on the blood of vertebrates & invertebrates.

♣Aquatic leeches cause considerable damage to their hosts due to:-

1- Blood sucking which cause reduction in host vitality.

2- Causing round reddish inflamed areas on the external surface of their host.

3- Opening portals of entry to secondary pathogens which increase susceptibility of fish to opportunistic pathogens.

4- Transmission of hematoprotozoans.

Diagnosis:

A- Presumptive Diagnosis:

1. Case history:-

Summer season, clinical abnormalities & presence of hard objects

2. Clinical signs:

♣ The attacked fish show:-

1- Restlessness & nervous swimming

2-Rubbing against hard objects.

3-Small rounded cut wounds (parasite print) are seen on fish skin.

4-Petechial hemorrhages & focal inflammations can be seen on fish skin.

5-The parasitic leeches can be seen by naked eyes as long, slender objects having segmented bodies on the external surface of the fish body.

Prevention:

♣The disease can be avoided through:-

1- Quarantining of live food &vegetations.

2- Getting rid off snails & Vegetations before fish stocking.

3-Filtration of water inflow with sand or gravel filter.

4-Drainage, drying, & disinfection of wet spots in ponds with quick lime are useful before fish stocking.

Control:

This can be done by application of:-

1- Masoten or metriphonate is added to the water in presence of fish for killing the worm, not the embryos in cocoons.

2- Sodium chloride can be used.

♣This is effective in removing freshwater leeches from fish as it paralyzes leeches, so drops off the fish.

♣Treated fish must be transported to clean pond.

3- Drainage & disinfection with chlorinated lime, then drying for 2 weeks post lime treatment to eliminate various life stages of the worm are essential.