مكتبة التقنية الصحية والطبية

قسم تحليلات

المرحلة الرابعة

(طفيليات/نظري)

العنوان: كلية التقنية الصحية والطبية / بغداد/ باب المعظم

Diagnostic parasitology class 4
Lecture one

Medical parasitology

Medical parasitology deals with the parasites, which cause human infections and the diseases they produce. It is broadly divided into 2 parts: **Protozoology** and Helminthology.

Parasites

Parasites are living organisms, which depend on a living host for their nourishment and survival. They multiply or undergo development in the host. The term '**parasite**' is usually applied to **Protozoa** (unicellular organisms) and **Helminths** (multicellular organisms).

Parasites can also be classified as:

Ectoparasite:

Ectoparasites inhabit only the body surface of the host without penetrating the tissue. Lice, ticks, and mites are examples of ectoparasites. The term **infestation** is often employed for parasitization with ectoparasites.

Endoparasite:

A parasite, which lives within the body of the host and is said to cause an infection is called an endoparasite. Most of the protozoan and helminthic parasites causing human disease are endoparasites.

Free-living parasite:

It refers to non parasitic stages of active existence, which live independent of the host, e.g. cystic stage of *Naegleria floweri.*

Endoparasites can further be classified as:

Obligate parasite:

The parasite, which cannot exist without a host, e.g. *Toxoplasma gondii* and *Plasmodium.*

Facultative parasite:

Organism which may either live as parasitic form or as free living form.

Accidental parasites:

Parasites, which infect an unusual host, are known as accidental parasites.

Echinococcus granulosus infects man accidentally, giving rise to hydatid cysts.

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Aberrant parasites:

Parasites, which infect a host where they cannot develop further, are known as aberrant or wandering parasites, e.g. *Toxocara canis* (dog roundworm) infecting humans.

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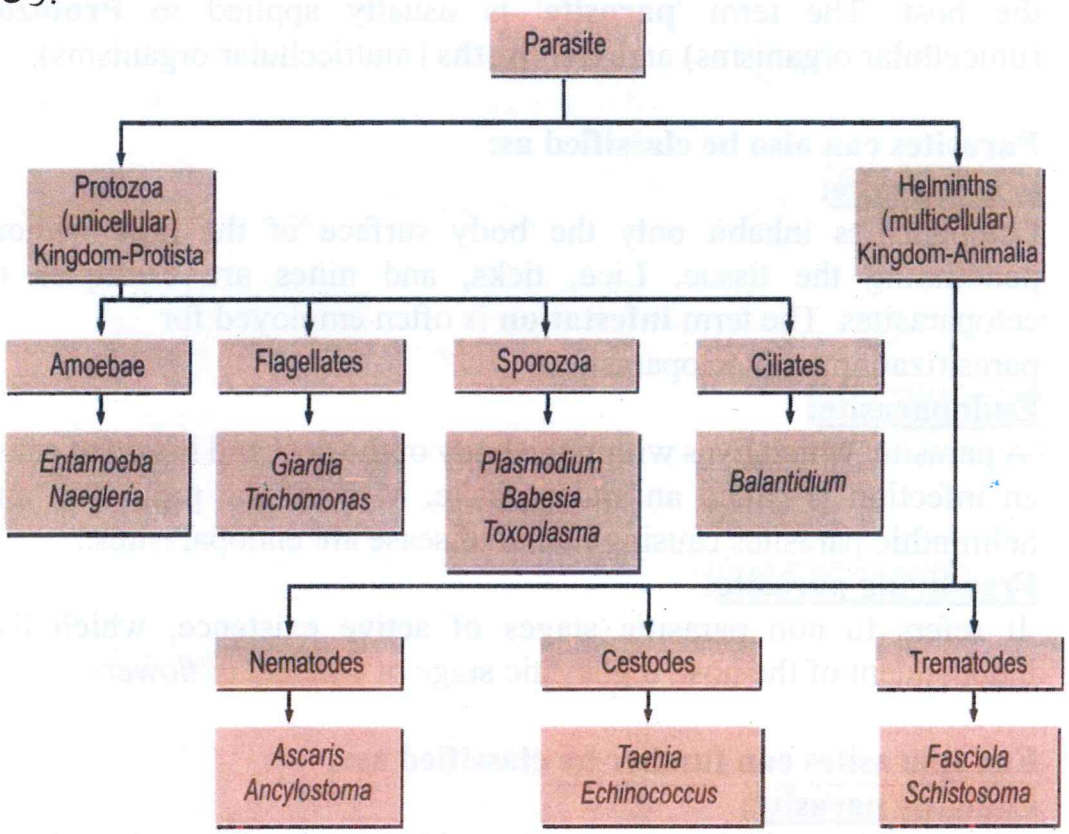


Types of parasitic host

Parasites having direct life cycle

Protozoa

- *Entamoeba histolytica*
- *Giardia lamblia*
- *Trichomonas vaginalis*
- *Balantidium coli*
- *Cryptosporidium parvum*
- *Cyclospora cayetanensis*
- *Isospora belli*
- *Microsporidia*

Helminths

- *Ascaris lumbricoides*
- *Enterobius vermicularis*
- *Trichuris trichiura*
- *Ancyclostoma duodenale*
- *Necator americanus*
- *Hymenolepis nana*

Parasites having indirect life cycle

Parasite	Definitive host	Intermediate host
Protozoa		
Plasmodium spp.	Female Anopheles mosquito	Man
Babesia	Tick	Man
Leishmania	Man, dog	Sandfly
Trypanosoma brucei	Man	Tsetse fly
Trypanosoma cruzi	Man	Triatomine bug
Toxoplasma gondii	Cat	Man
Cestodes		
Taenia solium	Man	Pig
Taenia saginata	Man	Cattle
Echinococcus granulosus	Dog	Man
Trematodes		
Fasciola hepatica	Man	Snail
Fasciolopsis buski	Man, pig	Snail
Schistosoma spp.	Man	Snail
Nematodes		
Trichinella spiralis	Man	Pig
Wuchereria bancrofti	Man	Mosquito
Brugia malayi	Man	Mosquito
Dracunculus medinensis	Man	Cyclops

Host-parasite Relationships

Host-parasite relationships are of following types

*Symbiosis تكافل

*Commensalism تغذية

*Parasitism. تطفل

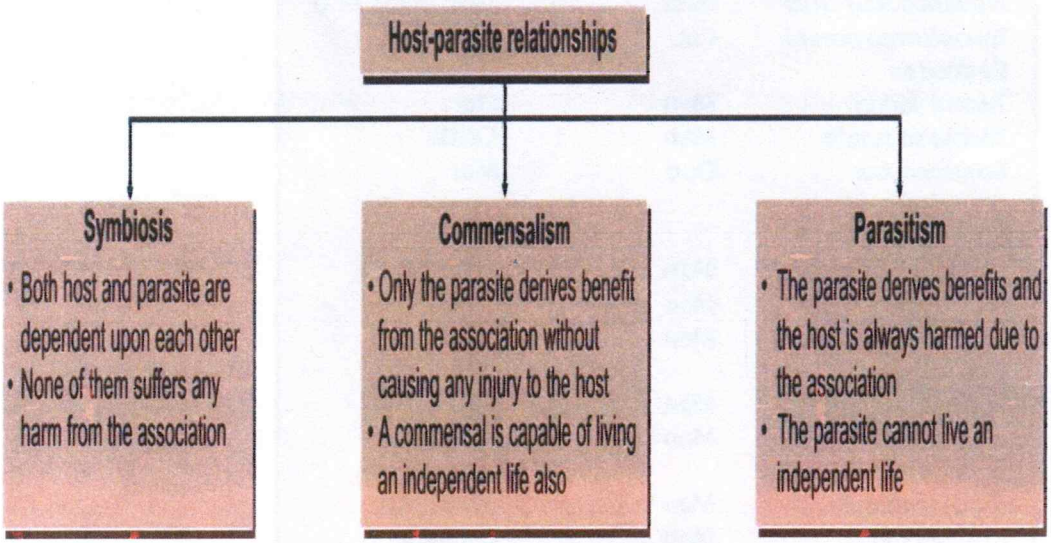
Life Cycle of Parasites

Direct life cycle:

When a parasite requires only single host to complete its development, it is called as direct life cycle, e.g. *Entamoeba histolytica* requires only a human host to complete its life cycle.

Indirect life cycle:

When a parasite requires 2 or more species of host to complete its development, the life cycle is called as indirect life cycle, e.g. malarial parasite requires both human host and mosquito to complete its life cycle.



Host-parasite relationships

Sources of Infection

Contaminated soil and water:

Soil polluted with embryonated eggs (roundworm, whipworm) may be ingested or infected larvae in soil, may penetrate exposed skin (hookworm).

Infective forms of parasites present in water may be ingested (cyst of amoeba and *Giardia*) Water containing the intermediate host may be swallowed (cyclops containing guinea worm larva Dracunculus Medinensis). Infected larvae in water may enter by penetrating exposed skin, (cercariae of schisotosomes)
€Free-living parasites in water may directly enter through vulnerable sites (*Naegleria* may enter through nasopharynx).

Food:

Ingestion of contaminated food or vegetables containing infective stage of parasite (amoebic cysts, *Toxoplasma* oocysts, *Echinococcus* eggs) Ingestion of raw or under-cooked meat harboring infective larvae (measly pork containing cysticercus cellulosae, the larval stage of *Taenia solium*).

Insect vectors:

A vector is an agent; usually an arthropod that transmits an infection from man to man or from other animals to man, e.g. female *Anopheles* is the vector of malarial parasite. Vectors can be:
€

Biological vectors:

The term biological vector refers to a vector, which not only assists in the transfer of parasites but the parasites undergo development or multiplication in their body as well. They are also called as **true vectors**. Example of true vectors are:
*Mosquito: Malaria, filariasis
*Sandflies: Kala-azar
*Tsetse flies: Sleeping sickness
* Reduviid bugs: Chagas' disease
*Ticks: Babesiosis.

Mechanical vectors:

The term mechanical vector refers to a vector, which assists in the transfer of parasitic form between hosts but is not essential in the life cycle of the parasite. Example of Mechanical vectors is: Housefly: amoebiasis.

Note:

In biological vectors, a certain period has to elapse after the parasite enters the vector, before it becomes infective. This is necessary because the vector can transmit the infection only after the parasite multiplies to a certain level or undergoes a developmental process in its body. This interval between the entry of the parasite into the vector and the time it takes to become capable of transmitting the infection is called the extrinsic incubation period.

Animals:

Domestic:

*Cow, e.g. *T. saginata*, Sarcocystis
*Pig, e.g. *T. solium, Trichinella spiralis*
*Dog, e.g. *Echinococcus granulosus*
*Cat, e.g. *Toxoplasma, Opisthrorochis.*

Wild:

* Wild game animals, e.g. trypanosomiasis
*Wild felines, e.g. *Paragonimus westermani*
*Fish, e.g. fish tapeworm
* Molluscs, e.g. liver flukes
*Copepods, e.g. guinea worm.

Other persons:

Which may be carriers of the parasite or patients, e.g. all anthroponotic. Infections, vertical transmission of congenital infections.

Self (autoinfection)

Finger-to-mouth transmission, e.g. pinworm internal re infection, e.g.

••*Strongyloides.*
••*Hymenolepis nana*
••*Enterobius vermicularis*
••*Taenia solium*
••*Strongyloides stercoralis*
••*Capillaria philippinensis*
••*Cryptosporidium parvum*

Lecture 2 Diagnostic parasitology

Habitat Large intestine

Mode of infection Contaminated food and water

Infected form **quadric nucleate** cyst

Precystic Stage

Trophozoites undergo encystment in the intestinal lumen.

Encystment does not occur in the tissues not in feces outside the body.

Before encystment, the trophozoite extrudes its food vacuoles and becomes round or oval, about 10–20.

μm in size. This is the precystic stage of the parasite

It contains a **large glycogen vacuole** and two **chromatid bars**.

It then secretes a highly retractile cyst wall around it and becomes cyst.

Cystic Stage

The cyst is spherical in shape about 10–20 μm in size.

The early cyst contains a single nucleus and two other structures—a mass of glycogen and 1–4 *chromatoid bodies or chromidial bars*, which are cigar-shaped refractile rods with rounded ends. The chromatoid bodies are so called because they stain with hematoxylin, like chromatin.

Life Cycle

E. histolytica passes its life cycle only in 1 host-man

Infective form: Mature quadrinucleate cyst passed in feces of convalscents and carriers. The cysts can remain viable under moist conditions for about 10 days.

Mode of transmission: Man acquires infection by swallowing food and water contaminated with cysts.

As the cyst wall is resistant to action of gastric juice, the cysts pass through the stomach undamaged and enter the small intestine.

Excystation: When the cyst reaches caecum or lower part of the ileum, due to the alkaline medium, the cyst wall is damaged by trypsin, leading to excystation.

The cytoplasm gets detached from the cyst wall and amoeboid movements appear causing a tear in the cyst wall, through which **quadrinucleate amoeba** is liberated. This stage is called the **metacyst** .

Metacystic trophozoites: The nuclei in the metacyst immediately undergo division to form **8 nuclei**, each of which gets surrounded by its own cytoplasm to become **8 small amoebulae** or **metacystic trophozoites**.

If exystation takes place in the small intestine, the metacystic trophozoites do not colonize there, but are carried to the caecum. The optimal habitat for the metacystic trophozoite is the submucosal tissue of caecum and colon, where they lodge in the glandular crypts and grow by binary fission. Some develop into precystic forms and cysts, which are passed in feces to repeat the cycle.

The entire life cycle is, thus completed in one host. In most of the cases, *E. histolytica* remains as a commensal in the large intestine without causing any ill effects. Such persons become carriers or asymptomatic cyst passers and are responsible for maintenance and spread of infection in the community. Sometimes, the infection may be activated and clinical disease ensues. Such latency and reactivation are the characteristics of amoebiasis.

Pathogenesis and Clinical Features

E. histolytica causes intestinal and extraintestinal amoebiasis.

Incubation period is highly variable. On an average, it ranges from 4 days to 4 months.

Amoebiasis can present in different forms and degree of severity, depending on the organ affected and the extent of damage caused.

Intestinal Amoebiasis

The lumen-dwelling amoebae do not cause any illnesss. They cause disease only when they invade the intestinal tissues. This happens only in about 10% of cases of infection,

the remaining 90% being asymptomatic.

Not all strains of *E. histolytica* are pathogenic or invasive. Differentiation between pathogenic and nonpathogenic strains can be made by susceptibility to complement-mediated lysis and phagocytic activity or by the use of genetic markers or monoclonal antibodies and zymodeme analysis.

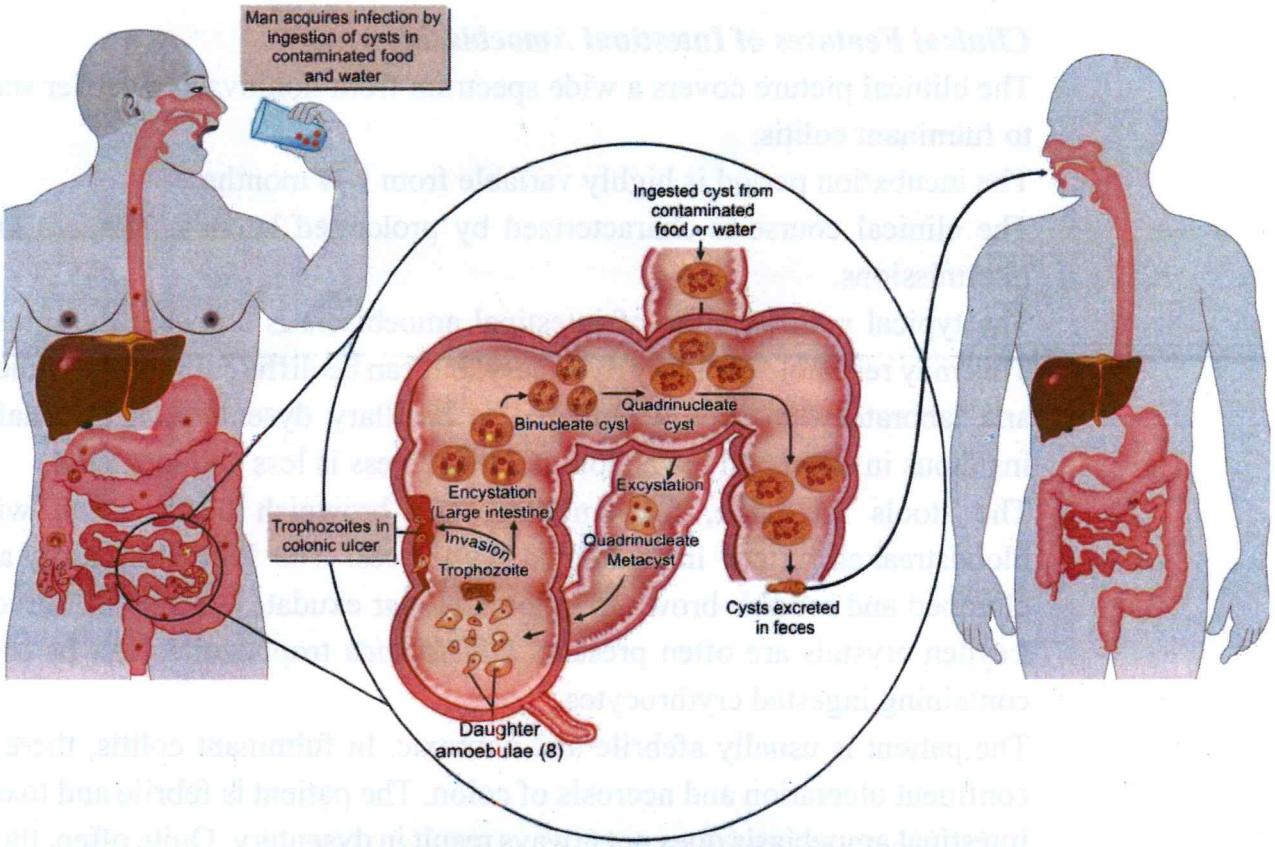
The metacystic trophozoites penetrate the columnar epithelial cells in the **crypts of Liberkühn** in the colon.

Penetration of the amoeba is facilitated by the motility of the trophozoites and the tissue lytic enzyme, **histolysin**, which damages the mucosal epithelium. Amoebic **lectin** another virulence factor mediates adherence.

Mucosal penetration by the amoeba produces discrete ulcers with pinhead center and raised edges. Sometimes, the invasion remains superfi cial and heals spontaneously. More often, the amoeba penetrates to submucosal layer and multiplies rapidly, causing lytic necrosis and thus forming an abscess. The abscess breaks down to form an ulcer.

Amoebic ulcer is the typical lesion seen in intestinal amoebiasis. The ulcers are **multiple** and are confined to the colon, being most numerous in the **caecum**

and next in the **sigmoidorectal region**. The intervening mucous membrane between the ulcers remains healthy. Occassionally, a granulomatous pseudotumoral growth may develop on the intestinal wall from a chronic ulcer. This amoebic granuloma or **amoeboma** may be mistaken for are malignant tumor.



LIFE CYCLE OF ENTAMOEBA HISTOLYTICA

Ulcers appear initially on the mucosa as raised nodules with pouting edges. They later break down discharging brownish necrotic material containing large numbers of
trophozoites.

The typical amoebic ulcer is **flask-shaped** in cross section, with mouth and neck being narrow and base large and rounded.

Multiple ulcers may coalesce to form large necrotic lesions with ragged and undermined edges and are covered with brownish slough.

The ulcers generally do not extend deeper than submucosal layer, but amoebae spread laterally in the submucosa causing extensive undermining and patchy mucosal loss. Amoebae are seen at the periphery of the lesions and extending into the surrounding healthy tissues. Occassionally, the ulcers may involve the muscular and serous coats of the colon, causing perforation and peritonitis. Blood vessel erosion may cause hemorrhage.

The superficial lesions generally heal without scarring, but the deep ulcers form scars which may lead to strictures, partial obstruction, and thickening of the gut wall.

Clinical Features of Intestinal Amoebiasis

The clinical picture covers a wide spectrum from noninvasive carrier state to fulminant colitis.

The incubation period is highly variable from 1–4 months.

The clinical course is characterized by prolonged latency, relapses and intermissions.

The typical manifestation of intestinal amoebiasis is amoebic dysentery. This may resemble bacillary dysentery, but can be differentiated on clinical and laboratory grounds. Compared to bacillary dysentery, it is usually insidious in onset and the abdominal tenderness is less and localized.

The stools are large, foul-smelling, and brownish black, often with bloodstreaked mucus intermingled with feces. The RBCs in stools are clumped and reddish-brown in color. Cellular exudate is scanty. Charcot-Leyden crystals are often present. *E.histolytica* trophozoites can be seen containing ingested erythrocytes.

The patient is usually afebrile and nontoxic. In fulminant colitis, there is confluent ulceration and necrosis of colon. The patient is febrile and toxic.

Intestinal amoebiasis does not always result in dysentery. Quite often, there may be only diarrhea or vague

abdominal symptoms popularly called '**uncomfortable belly**' or '**growling abdomen**.'

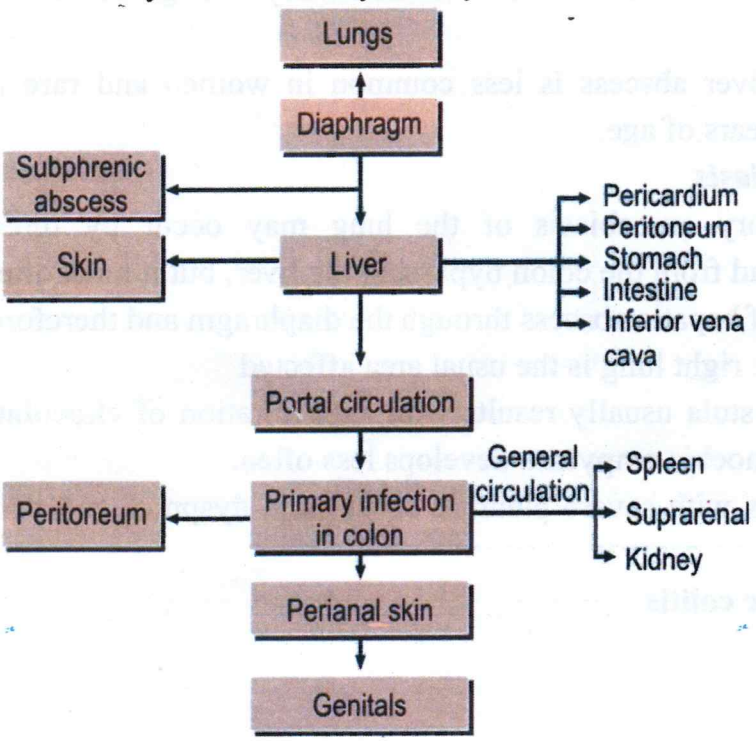
Chronic involvement of the caecum causes a condition simulating appendicitis.

Extraintestinal Amoebiasis

Hepatic Amoebiasis

Hepatic involvement is the most common extraintestinal complication of amoebiasis. Although trophozoites reach the liver in most cases of amoebic dysentery, only in a small
proportion do they manage to lodge and multiply there. In the tropics, about 2–10% of the individuals infected with *E. histolytica* suff er from hepatic complications.

The history of amoebic dysentery is absent in more than 50% of cases.



Several patients with amoebic colitis develop an enlarged tender liver without detectable impairment of liver function or fever. This acute hepatic involvement (**amoebic hepatitis**) may be due to repeated invasion by amoebae from an active colonic infection or to toxic substances from the colon reaching the liver.

It is probable that liver damage may not be caused directly by the amoebae, but by lysosomal enzymes and cytokines from the infl ammatory cells surrounding the trophozoites. In about 5–10% of persons with intestinal amoebiasis,

liver abscesses

The center of the abscess contains **thick chocolate brown pus (anchovy sauce pus),** which is liquefi ed necrotic liver tissue. It is bacteriologically sterile and free of amoeba. At the periphery, there is almost normal liver tissue, which contains invading amoeba.

Liver abscess may be multiple or more often solitary, usually located in the upper right lobe of the liver. Jaundice develops only when lesions are multiple or when they press on the biliary tract.

Untreated abscesses tend to rupture into the adjacent tissues through the diaphragm into the lung or pleural cavity, pericardium, peritoneal cavity, stomach, intestine, or inferior vena cava or externally through abdominal wall and skin.

The incidence of liver abscess is less common in women and rare in children under 10 years of age.

Pulmonary Amoebiasis

Very rarely, primary amoebiasis of the lung may occur by direct hematogenous spread from the colon bypassing the liver, but it most often follows extension of hepatic abscess through the diaphragm and therefore, the lower part of the right lung is the usual area affected

Hepatobronchial fi stula usually results with expectoration of **chocolate brown sputum**. Amoebic empyema develops less often.

The patient presents with severe pleuritic chest pain, dyspnea, and non-productive cough.

Fulminant amoebic colitis

Toxic megacolon

Perianal ulceration

Amoeboma

Extraintestinal amoebiasis

Amoebic hepatitis

Amoebic liver abscess

Amoebic appendicitis and peritonitis

Pulmonary amoebiasis

Cerebral amoebiasis

Splenic abscess

Cutaneous amoebiasis

Genitourinary amoebiasis

Metastatic Amoebiasis

Involvement of distant organs is by hemato genous spread and through lymphatics. Abscesses in kidney, brain, spleen, and adrenals have been noticed. Spread to brain leads to severe destruction of brain tissue and is fatal.

Cutaneous Amoebiasis

It occurs by direct extension around anus, colostomy site, or discharging sinuses from amoebic abscesses. Extensive gangrenous destruction of the skin occurs. The lesion may
be mistaken for condyloma or epithelioma.

Genitourinary Amoebiasis

The prepuce and glans are aff ected in penile amoebiasis which is acquired through anal intercourse. Similar lesions in females may occur on vulva, vagina, or cervix by spread
from perineum. The destructive ulcerative lesions resemble carcinoma.

L3 Diagnostic parasitology

Laboratory Diagnosis

Diagnosis of Intestinal Amoebiasis

Stool examination

Intestinal amoebiasis has to be differentiated from bacillary dysentery. The stool should be collected into a_wide mouth container and examined without delay. It should be inspected macroscopically as well as microscopically,

Macroscopic Appearance: The stool is foul-smelling, copious, semi-liquid, brownish black in color, and intermingled with blood and mucus. It does not adhere to the container.

Microscopic Appearance: Saline preparation

The cellular exudate is scanty and consists of only the nuclear masses (pyknotic bodies) of a few pus cells, epithelial cells, and macrophages. The RBCs are in clumps and yellow or brown red in color. Charcot-Leyden crystals are often present. These are diamond-shaped, clear and refractile crystals.

Actively motile trophozoites throwing pseudopodia can be demonstrated in freshly-passed stool. Presence of ingested RBCs clinches the identity of *E. histolytica*. Nucleus is not visible but a faint outline may be detected.

€ Cyst has a smooth and thin cell wall and contains round refractile chromatoid bars.

For the demonstration of cysts or dead trophozoites, stained preparations may be required for the study of the nuclear character. Iodine-stained preparation is commonly employed for this purpose. The trophozoite of *E. histolytica* stains yellow to light brown. Nucleus is clearly visible with a central karyosome. The cyloplam of the cystic stage shows
smooth and hyaline appearance. Nuclear chromatin and karysome appear bright yellow Glycogen masses stain golden brown and chromatoid bars are not stained.Trichrome stain is useful to demonstrate intracellular features of both trophozoites and cysts. Since excretion of cysts in the stool is often intermittent, at least 3 consecutive specimens should be examined.

Mucosal Scrapings

Scraping obtained by sigmoidoscopy is often contributory. Examination method includes a direct wet mount and iron hemotoxylin and immunofluroscent staining.

Stool Culture

Stool culture is a sensitive method in diagnosing chronic and asymptomatic intestinal amoebiasis.

Media used for stool culture

€ Boeck and Drbohlav media

€ NIH polygenic media
€ Craig's medium
€ Nelson's medium
€ Robinson's medium

Serodiagnosis

Serological tests become positive only in invasive amoebiasis.

Various serological tests done include— **Indirect hemagglutination (IHA) test:** serum with antibody titer of 1:256 or more by IHA is diagnostic of amoebic hepatitis.

Latex agglutination test.

Enzyme-linked immunosorbent assay (ELISA):

Commercially available tests that use ELISAs to detect *Entamoeba* antigens are less expensive and more easily performed and are being used with increasing frequency. Greater sensitivity than microscopy and the ability to detect *E. histiolytica* specifically are claimed by some of the leading kits, representing significant advantages over microscopy.

Molecular Diagnosis

Recently, DNA probes and Radio immunoassay have been used to detect *E. histolytica* in stool. It is a rapid and specific method.

Diagnosis of Extraintestinal Amoebiasis

Microscopy

Microscopic examination of pus aspirated from liver abscess may demonstrate trophozoite of *E.histolytica* in less than 20 percent cases. In case of liver abscess, when diagnostic aspiration is done, the pus obtained from the center of the abscess may not contain amoeba as they are confined to the periphery. The fluid draining after a day or two is more likely to contain the trophozoite. Aspirates from the margins of the abscess would also show the trophozoites. Cysts are never seen in extraintestinal lesions.

Liver biopsy

Trophozoite of *E.histolytica* may be demostrated in liver biopsy specimen, in case of hepatic amoebiasis or amoebic hepatitis.

Serological test

Serological test, are of immense value in the diagnosis of hepatitis amoebiasis.

Craig (1928) was the first to report a **complement fixation test** in amoebiasis. Subsequently a number of different serological tests have been developed including

indirect haemagglutination (IHA), latex agglutination (LA), gel diffusion precipitation (GDP), cellulose acetate membrane precipitation (CAP) test, counter current immunoelectrophoresis (CIE) and enzyme linked immunosorbent assay (ELISA).

***Naegleria fowleri*:**

History and Distribution

N. fowleri is named after Fowler, who along with Carter described it first from Australia in 1965.

****N. fowleri*** is a heat-loving (**thermophilic**) amoeba that thrives in warm water at low oxygen tension and is commonly found in warm freshwater (e.g. lakes, rivers, and springs) and soil.

* It is worldwide in distribution.

*In the last 10 years from 2002 to 2011, 32 infections were reported in the US, and in India, a total of 17 cases have been reported so far.

Morphology

N.fowleri occurs in 3 forms:

1- Cyst

2- Amoeboid trophozoite form

3- Flagellate trophozoite form

Trophozoite Stage

The trophozoites occur in 2 forms, **amoeboid and flagellate**.

Amoeboid form

The amoeboid form is about 10–20 μm, showing rounded pseudopodia (**lobopodia**), a spherical nucleus with big endosome, and pulsating vacuoles. With electron microscopy, vacuole appear to be densely granular in contrast to highly vacuolated body of amoeba and are called as **amoebostomes**. They are used for engulfing RBCs and WBCs and vary in number, depending on the species. Amoeboid form is the feeding, growing, and replicating form of the parasite, seen on the surface of vegetation, mud, and water. It is the invasive stage of the parasite and the infective form of the parasite.

Cyst Stage

Trophozoites encyst due to unfavorable conditions like food deprivation, dessication, cold temperature, etc. The cyst is 7–10 μm in diameter and has a smooth double wall. They are the resting or the dormant form and can resist unfavorable conditions, such as drying and chlorine up to 50 ppm. The trophozoites can withstand moderate heat (45°C), but die at chlorine levels of 2 ppm and salinity of 0.7%. Cysts and fl agellate forms of *N. fowleri* have never been found in tissues of cerebrospinal fluid (CSF).

Life Cycle

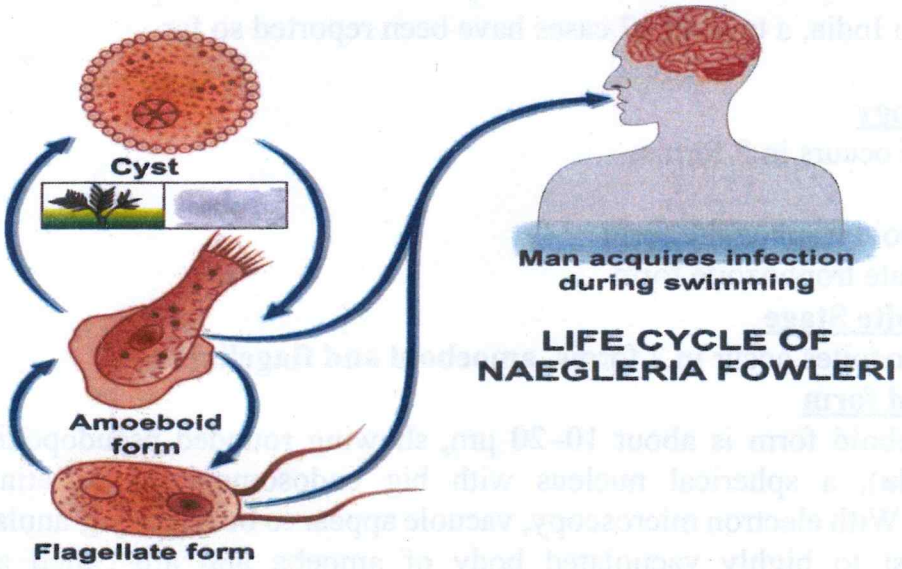
Typically, infection occurs when people go swimming or diving in warm freshwater river or ponds and poorlymaintained swimming pools or nasal

irrigation using contaminated tap water. The life cycle of *N. fowleri* is completed in the external environment.

*The amoeboid form of trophozoite multiplies by binary fission.

*Under unfavorable conditions, it forms a cyst and which undergoes excystation in favorable conditions.

*Flagellate form of trophozoite helps in the spread of *N. fowleri* to new water bodies. Since the amoeboid form is the invasive stage, hence, the flagellate forms revert to amoeboid forms to become infective to man.



Pathogenecity and Clinical Features

Patients are mostly previously healthy young adults or children.

*Human infection comes from water containing the amoebae and usually follows swimming or diving in ponds.

*The amoebae invade the nasal mucosa and pass through the olfactory nerve branches in the cribri form plate into the meninges, and brain to initiate an acute purulent meningitis and encephalitis, **called as primary amoebic meningo encephalitis (PAM)**.

*The incubation period varies from 2 days to 2 weeks.

*In the incubation period, the patient experiences anosmia.

*The disease advances rapidly, causing fever, headache, vomiting, stiff neck, ataxia, seizure, and coma.

*Cranial nerve palsies, especially of the third, fourth, and sixth nerves have also been documented.

*The disease almost always ends fatally within a week (average 5 days).

Laboratory Diagnosis

The diagnosis of PAM is based on the finding of motile *Naegleria* trophozoites in wet mounts of freshly-obtained CSF.

Cerebrospinal Fluid Examination

The CSF is cloudy to purulent, with prominent neutrophilic leucocytosis, elevated protein, and low glucose, resembling pyogenic meningitis.

*Wet film examination of CSF may show trophozoites.

*Cysts are not found in CSF or brain.

*At autopsy, trophozoites can be demonstrated in brain histologically by immunofluroscent staining.

Culture

N. fowleri can be grown in several kinds of liquid axenic media or non-nutrient agar plates coated with *Escherichia coli.* Both trophozoites and cysts occur in culture.

Molecular Diagnosis

Newer tests based on polymerase chain reaction (PCR) technology are being developed.

Treatment

The drug of choice is amphotericin-B intravenously. It can also be instilled directly into the brain.

*Treatment combining miconazole and sulfadiazine has shown limited success, only when administered early.

*More than 95% cases of PAM are fatal despite of treatment.

Lecture 4

Acanthamoeba Species

A. culbertsoni (formerly, *Hartmanella culbertsoni*) is the species most often responsible for human infection but other species like *A. polyphagia*, *A. castalleni,* and *A. astromyx* have also been reported.

Distribution

This is an opportunistic protozoan pathogen found worldwide in the environment in water and soil.

*Approximately, 400 cases have been reported worldwide.

Morphology

Acanthamoeba exists as active trophozoite form and a resistant cystic form.

*The trophozoite is large, 20–50 μm in size and characterized by spine-like pseudopodia (**acanthopodia**).

*It differs from *Naegleria* in not having a flagellate stage and in forming cysts in tissues.

*The polygonal double-walled cysts are highly resistant.

*The cysts are present in all types of environment, all over the world.

Life Cycle

*Both trophozoites and cysts are infective.

*Human beings acquire by inhalation of cyst or trophozoite, ingestion of cysts, or through traumatized skin or eyes.