Ministry of Higher Education & Scientific Research
Al-Rasheed University College
Department of Medical Laboratory Technique

وزارة التعليم العالي والبحث العلمي
كلية الرشيد الجامعة
قسم تقنيات المختبرات الطبية

حقيبة تعليمية

بعنوان:

الطفيليات الطبية

إعداد
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المقدمة

يتسم البرنامج التعليمي لمقرر الطفيليات الطبية بتدريس الطلبة انواع الطفيليات والصفات العامة لها ودورة حياتها وتشخيصها ويتدرب الطالب خلال هذه الفترة على اكتساب المعلومات والمهارات على معرفة العينات الخاصة بكل طفيلي والقدرة على تشخيص انواع الطفيليات والتمييز بينها بالإضافة الى كتابة نتائج المشاهدات المجهرية .

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وصف المقرر الدراسي

HIGHER EDUCATION PERFORMANCE REVIEW: PROGRAMME REVIEW

COURSE SPECIFICATION

This Course Specification provides a concise summary of the main features of the course and the learning outcomes that a typical student might reasonably be expected to achieve anddemonstrate if he/she takes full advantage of the learning opportunities that are provided. It should be cross-referenced with the programme specification.

1. Teaching Institution	Al-rasheed University College
2. University Department/Centre	Medical Laboratory Techniques
3. Course title/code	Medical Parasitology / MePa422
4. Programme(s) to which itcontributes	Medical laboratory technique
5. Modes of Attendance offered	Online learning & face to face learning
6. Semester/Year	Semester1&2/ 4th year class/2022-2023
7. Number of hours tuition (total)	60
8. Date of production/revision of this specification	10/9/2022
9. Aims of the Course	
Introduce the student to the classification of parasites, the diseases they cause, and the molecular laboratory tests through which parasites can be diagnosed.	

10· A. Learning Outcomes, Teaching ,Learning and Assessment Methode
B. General and Transferable Skills (other skills relevant to employability and personal development)
C. Subject-specific skills Training to use the equipment making reports Research work
Teaching and Learning Methods The discussion daily exams Guiding students to some websites related to the scientific subject Field visits to educational laboratories
Assessment methods Daily and monthly exams Making reports The discussion
C. Thinking Skills Develop the student's ability to work with devices Develop the student's ability to use modern laboratory equipment and techniques Develop the student's ability to dialogue and debate Develop the student's ability to learn electronically
Teaching and Learning Methods Adapting the student to daily activities and duties Allocate a percentage of the grade for daily exams

Assessment methods
Daily and monthly exams The student's commitment to attend the laboratory and follow up on everything new The speed in the student's completion of research and reports is an indication of seriousness and responsibility

A- Knowledge and Understanding

1. Gaining experience in diagnosing parasites
2. Gaining experience in the work of laboratory equipment
3. Dealing with various advanced laboratory analyzes

11. Course Structure					
Week	Hours	ILOs	Unit/Module or TopicTitle	Teaching Method	Assessment Method
1-2	4	Classification of parasite	Recent classification of parasite * Systematic grouping of parasites* General terms used in parasitology	Video lecture + lab	Daily exam
3-5	4	Handling of laboratory samples for diagnosis	Strategies for diagnosis of parasitic infection Collection and transport of specimens for * enteric pathogens Factors interfering for all types of stool * collection	Video lecture + lab	Daily exam
6-7	4	Stool diagnosis	Examination of stool sample a) Macroscopic examination of stool b) Microscopic examination of wet mounts	Video lecture + lab	Daily exam
8	4	pigmentation	Preparation of solutions for wet mount; the advantages and disadvantages solution: * Saline solution * Iodine solutions * Eosin solution	Video lecture + lab	Daily exam
9	4	static pigmentation	Preparation of preservatives and fixatives for mounted slides * Formalin solution (5-7%) * PVA (Polyvinyle alcohol) as fixative * Schaudinns fixativ	Video lecture + lab	Daily exam
10	4	Common diagnosis of parasites	Laboratory diagnosis of enteric protozoa * The routine methods used in laboratory diagnosis	Video lecture + lab	Daily exam
11	4	Concentrated methods of diagnosis	Concentration methods; types, purpose to use concentration methodes	Video lecture + lab	Daily exam
12	4	Advanced Serological Diagnostics	Application of immunological methods in the diagnosis of parasite in general * Detection of antibodies in serum of patients with enteric protozoa (ELISA) * Detection of antigens in stool specimen of patients with enteric protozoa (ELISA)	Video lecture + lab	Daily exam
13	4	Differentiate between primitives and their forms	Differentiation of pathogenic *Entamoebahistolytica* and the morphologically identical non pathogenic*Entamoebadispar* using immunological assays	Video lecture + lab	Daily exam

14	4	Molecular diagnosis of parasites	The application of molecular assays in the diagnosis ofparasites	Video lecture + lab	Daily exam
15	4	free living firsts	.Naegleriafowleri&Acanthamoeba spp Morphology, habitat, mode of infection, infective stage, life cycle and laboratory diagnosis	Video lecture + lab	Daily exam
16	4	flagellate	Tissue flagellates e.gGenusTrypanosoma&GenusLeishmania Laboratory diagnosis; routine methods, immunological Assays and molecular assays	Video lecture + lab	Daily exam
17	4	Baghdad pill and methods of prevention	Properties of ideal vaccines.leishmania Vaccine in trail	Video lecture + lab	Daily exam
18	4	Intestinal coccidian	Intestinal coccidian e.g Cryptosporidium parvum Morphology, habitat, mode of infection, infective stage, lifecycle and laboratory diagnosis with special emphasis on Ziehl-Neelsen technique	Video lecture + lab	Daily exam
20-21	4	Toxoplasmosis	Extra-intestinal coccidian e.g.ToxoplasmagondiiBrief lecture on morphology, habitat, modes of infection,infective stages, life cycle	Video lecture + lab	Daily exam
22-23	4	Advanced laboratory diagnosis	Methods of laboratorydiagnosis includes: Direct detection of the parasite; Serological methods& Molecular assays	Video lecture + lab	Daily exam
24	4	Genus Plasmodium	Genus Plasmodium; Terms used in malaria& Life cycle	Video lecture + lab	Daily exam
25	4	Rapid diagnosis of parasites and pigmentation	Methods of laboratory diagnosis include: - Preparation and detection of parasite in thick and thin blood Smears Preparation of Geimsa and leishman stains - Quantitative Buffy Coat (QBC) test - Non microscopic test - Rapid Diagnostic Tests (RDTs -	Video lecture + lab	Daily exam
26	4	Introduction to Helminths	Introduction to Helminths Classification of helminthes into: Phylum Platyhelminths which includes; Class Cestoda& Class Trematoda	Video lecture + lab	Daily exam

27	4	Bovine and pig worms	Genus Taenia including Taeniasaginata&T. soluim Morphology, habitat, mode of infection, infective stage, life cycle and laboratory diagnosis;differentiate between both species in laboratory	Video lecture + lab	Daily exam
28	4		EchinococcusgranulosusShort notes on the parasite with special emphasis on the methods of diagnosis (detection of certain Ag)	Video lecture + lab	Daily exam
29	4	liver worms	Genus Schistosoma in general with emphasis on the species endemic in Iraq Schistosomahaematobium the use of special technique in the examination of urine sample (filtration by Schisto-kit) as direct method and immunoblot as indirect method	Video lecture + lab	Daily exam

Required reading: · CORE TEXTS · COURSE MATERIALS · OTHER	Textbook of Human Parasitology Advances in Parasitology 2015
Special requirements (include forexample workshops, periodicals,IT software, websites)	Websites
Community-based facilities (include for example, guest Lectures , internship,field studies)	Lectures Field studies

إرشادات للطلبة

- الرغبة والحماس للتعليم
- كن مشاركاً في جميع الأنشطة
- احترم أفكار المدرس والزملاء
- أنقد أفكار المدرس والزملاء بأدب إن كانت هناك حاجة.
- احرص على استثمار الوقت
- تقبل الدور الذي يسند إليك في المجموعة
- حفز أفراد مجموعتك في المشاركة في النشاطات
- احرص على بناء علاقات طيبة مع المدرس والزملاء أثناء المحاضرة
- احرص على ما تعلمته في المحاضرة وطبقه في الميدان .
- ركز ذهنك بالتعليم واحرص على التطبيق المباشر
- تغلق الموبايل قبل الشروع بالمحاضرة

الوحدة الاولى - المحاضرة الأولى - الزمن: 90 دقيقة

أهداف المحاضرة الاولى:

يتوقع في نهاية الجلسة أن يكون الطالب قادراً على:

1- Discuss the various types of parasites and hosts.
2- Explain the relationship between a parasite and the host and their effects.
3- Discuss in detail the classification of medically important parasites.

موضوعات المحاضرة الاولى:

1- Recent classification of parasite
2- Systematic grouping of parasites
3- General terms used in parasitology

الأساليب والأنشطة والوسائل التعليمية

م	الأساليب والأنشطة التدريبية	الوسائل التدريبية
1	• محاضرة • مناقشة • سؤال وجواب • نشاط (1/1/1) نشاط	• جهاز حاسوب • جهاز عرض • سبورة • اوراق واقلام

خطة إجراءات تنفيذ المحاضرة الاولى

الوحدة	المحاضرة	الإجراءات	الزمن بالدقيقة
الأولى	الأولى	التعريف بالبرنامج وأهدافه وأهميته	90 دقيقة
		القاء المحاضرة مستخدما جهاز العرض والسبورة	
		طرح بعض الاسئلة خلال القاء المحاضرة	
		نشاط (1/1/1) تمرين تعريف وتعداد	

المادة العلمية:

Diagnostic parasitology
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:

1- 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 patriotization with ectoparasites.

2- 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.

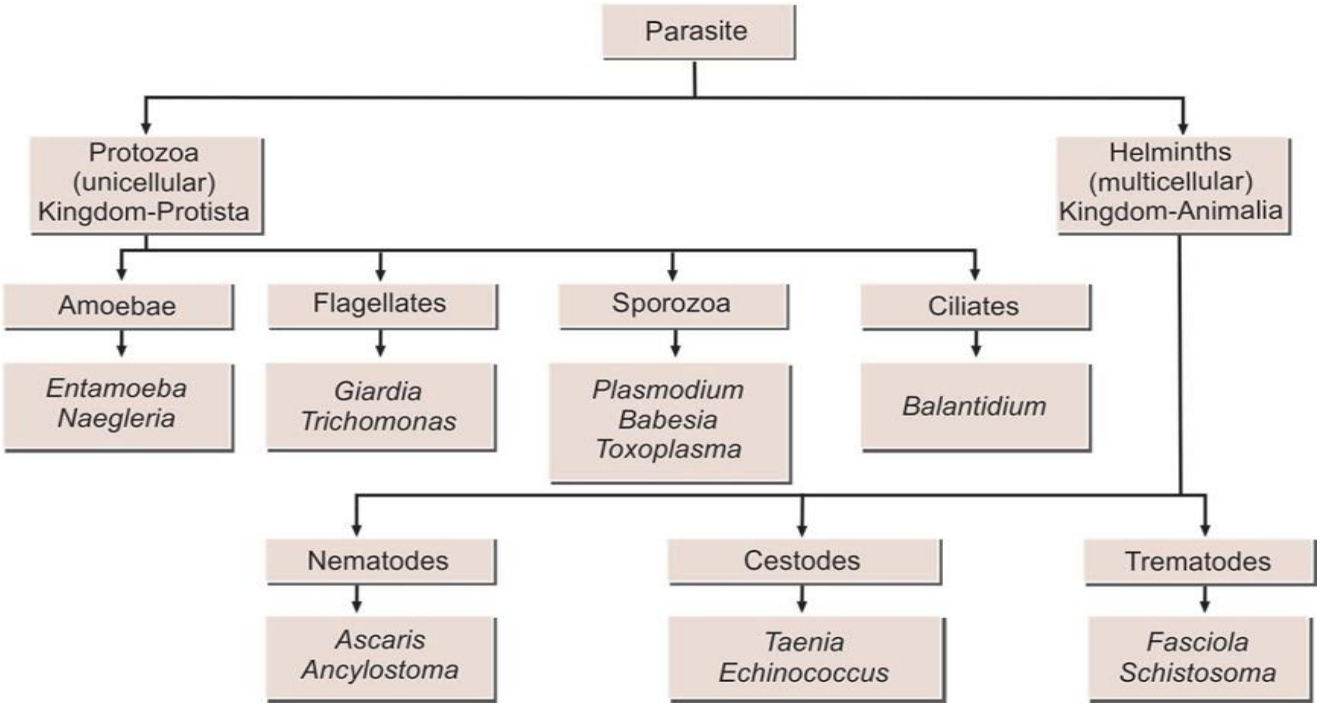
3-Free-living parasite: It refers to non-parasitic stages of active existence, which live independent of the host, e.g., cystic stage of Naegleria flowery. Endoparasites can further be classified as:

4-Obligate parasite: The parasite, which cannot exist without a host, e.g., Toxoplasma gondii and Plasmodium.

5-Facultative parasite: Organism which may either live as parasitic form or as free-living form.

6-Accidental parasites: Parasites, which infect an unusual host, are known as accidental parasites. Echinococcus granulosus infects man accidentally, giving rise to hydatid cysts.

7- 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.



Host: Host is defined as an organism, which harbors the parasite and provides nourishment and shelter to latter and is relatively larger than the parasite.

The host may be of the following types:

1- Definitive host: The host, in which the adult parasite lives and undergoes sexual reproduction is called the definitive host, e.g., mosquito acts as definitive host in malaria. *The definitive host may be a human or any other living being. However, in majority of human parasitic infections, man is the definitive host (e.g., filarial, roundworm, hookworm).

2- Intermediate host: The host, in which the larval stage of the parasite lives or asexual multiplication takes place, is called the intermediate host. In some parasites, 2 different intermediate hosts may be required to complete different larval stages. These are known as first and second intermediate hosts, respectively.

3- Paratenic host: A host, in which larval stage of the parasite remains viable without further development is referred as a paratenic host. Such host transmits the infection to another host.

4-Reservoir host: In an endemic area, a parasitic infection is continuously kept up by the presence of a host, which harbors the parasite and acts as an important source of infection to other susceptible hosts, e.g., dog is the reservoir host of hydatid disease.
5- Accidental host: The host, in which the parasite is not usually found, e.g., man is an accidental host for cystic echinococcosis.

Parasites with man as intermediate or secondary host
Plasmodium spp.
Babesia spp.
Toxoplasma gondii
Echinococcus granulosus
Echinococcus multilocularis
Taenia solium
Spirometra spp.

Zoonosis: The word zoonosis was introduced by Rudolf Virchow in 1880 to include the diseases shared in nature by man and animals.
Defined zoonosis as: Those diseases and infections, which are naturally transmitted between vertebrate animals and man".
It is of following types:
* Protozoal zoonoses, e.g., toxoplasmosis, leishmaniasis, balantidiasis, and cryptosporodiasis
*Helminthic zoonoses, e.g., hydatid disease, taeniasis
* Anthropozoonoses: Infections transmitted to man from lower vertebrate animals, e.g., cystic echinococcosis
* Zooanthroponoses: Infections transmitted from man to lower vertebrate animals, e.g., human tuberculosis to cattle.

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
*Commensalism
*Parasitism

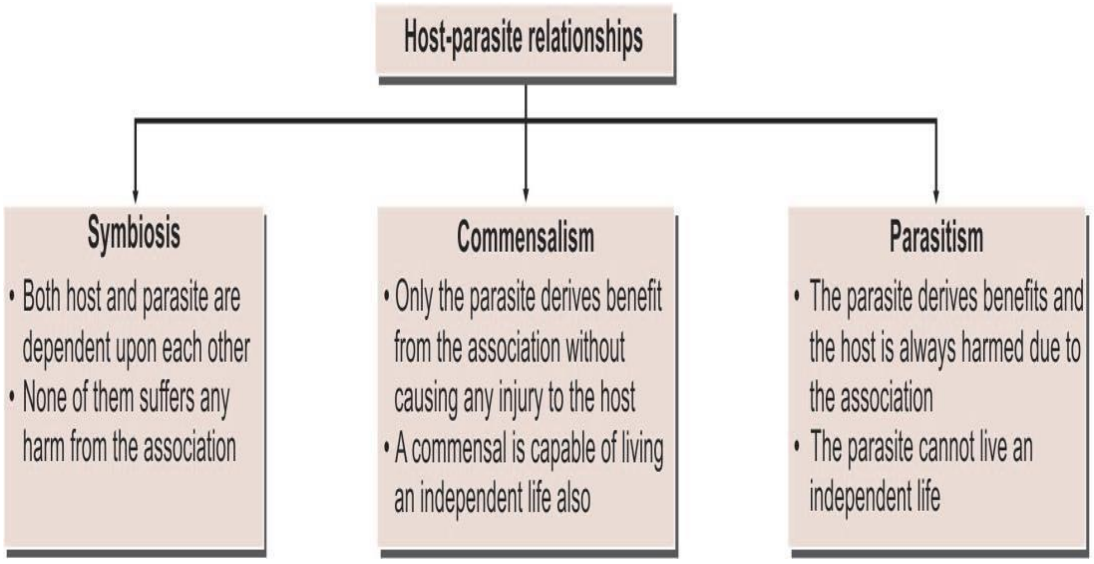
.

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.



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.

نشاط (1/1/1)

Q- Define the Parasite and Enumerate different kinds of Parasite.

الوحدة الثانية - المحاضرة الأولى - الزمن: 90 دقيقة

أهداف المحاضرة الاولى:

يتوقع في نهاية الجلسة أن يكون الطالب قادراً على:

1-Enumerate the correct sampling methods.
2- Enumerate the types of samples we need to detect parasites.

موضوعات المحاضرة الاولى:

1- Strategies for diagnosis of parasitic infection.

الأساليب والأنشطة والوسائل التعليمية

م	الأساليب والأنشطة التدريبية	الوسائل التدريبية
1	• محاضرة • مناقشة • سؤال وجواب • نشاط (1/1/2) نشاط	• جهاز حاسوب • جهاز عرض • سبورة • اوراق واقلام

خطة إجراءات تنفيذ المحاضرة الاولى

الوحدة	المحاضرة	الإجراءات	الزمن بالدقيقة
الثانية	الأولى	مراجعة سريعة للمحاضرة السابقة	90 دقيقة
		القاء المحاضرة مستخدما جهاز العرض والسبورة	
		طرح بعض الاسئلة خلال القاء المحاضرة	
		نشاط (1/1/2) تمرين تعداد	

المادة العلمية:

Sample collection

Patient is asked to pass stool in a clean container.*

Stool should be collected in a sterilized, wide mouthed container.*

Stool portion containing mucus, blood, etc. is to be collected.*

Should be uncontaminated with urine or any other body secretions.*

*> 2 gm is required.

*Properly named and always a fresh sample should de tested.

* Liquid stool to be examined within ½ hour.

*Solid stool to be examined within 1 hour.

*If delayed store in a refrigerator.

*3 samples of stool within 10 days to exclude false negatives.

*Formalin is the best preservative. It kills the bacteria but preserves the protozoa and helminths.

*For culture no preservative to be used.

Samples we need for detection about the parasites are:

*Stool (E. histolytica, G. lamblia, …ect.)

* Urine (S. heamatobium,… etc)

* Blood (Plasmmodium, leishmania spp., Trypanosoma)

*Sputum (Larval stages of Ascaris, Strongyloides)

*Biopsies :(Direct microscopic examination of muscle (Trichinella spiralis) or intestinal / bladder mucosa (Schistosoma eggs, Entamoeba).

*Aspirates and Biopsies: (for Giardia lamblia and Strongyloides stercoralis).

*Abscess aspirates - usually for extra-intestinal amoebiasis (liver aspiration)

* Anal Swabs: (Enterobius vermicularis & other helminth eggs can be seen)

* Genital Specimens :(Trichomonas vaginalis - vaginal, urethral, prostatic exudates, looking for motile organisms).

Methods for diagnosis parasitic samples

- Examined fresh stool (direct).
- Examined fresh urine (direct).
- Immunological methods.
- Molecular methods.
- Culture methods.
- Histological examination (bone marrow)

نشاط (1/1/2)

Q\ Enumerate the Samples we need for detection about the parasites?

الوحدة الثانية - المحاضرة الثانية - الزمن: 90 دقيقة

أهداف المحاضرة الثانية:

يتوقع في نهاية الجلسة أن يكون الطالب قادراً على:

1. Enumeration types of examination in stool
2. Knowing the normal characteristics of stool

موضوعات المحاضرة الثانية:

1- Collection and transport of specimens for enteric pathogens
2- Factors interfering for all types of stool collection

الأساليب والأنشطة والوسائل التعليمية

م	الأساليب والأنشطة التدريبية	الوسائل التدريبية
1	• محاضرة • عرض فيديو توضيحي • سؤال وجواب • نشاط (1/2/2) نشاط	• جهاز حاسوب • جهاز عرض • سبورة • اوراق واقلام

خطة إجراءات تنفيذ المحاضرة الثانيه

الوحدة	المحاضرة	الإجراءات	الزمن بالدقيقة
الثانية	الثانية	طرح أسئلة تتعلق بالمحاضرة السابقة	90 دقيقة
		القاء المحاضرة مستخدما جهاز العرض والسبورة	
		طرح بعض الاسئلة خلال القاء المحاضرة	
		نشاط (1/2/2) فراغات	

المادة العلمية:

Definition

*Human feces are called as stool
*Faeces / Feces is plural of latin term faex meaning RESIDUE.

* It is the waste residue of indigestible materials of the digestive tract expelled through the anus during defecation.
*Meconium is newborn's first feces. SCATOLOGY or CAPROLOGY is the study of feces.

Composition

- ¾ Water, ¼ Solid
- Undigested and Unabsorbed food
- Intestinal secretions, Mucous
- Bile pigments and Salts
- Bacteria and Inorganic material
- Epithelial cells, Leukocytes

Precaution Before Collection

- Patient should avoid the following things for at least 48 hrs. before collection of stools
- Meneral oils, bismuth, non-absorbable antidiarroeal drugs, antimalarial drugs, antibiotics, etc
- Avoid iron containing drugs, meat, fish etc for at least 48hrs. Before stool for occult blood
- In constipated patients use only non-residual purgative.

COLLECTION (Universal precautions)

- Stool should be collected in a sterilized, wide mouthed container.
- Loose/last/portion containing mucus, blood etc is to be collected in a wide mouthed bottle.
- Should be uncontaminated with urine or any other body secretions.
- Properly named and always a fresh sample should be tested.
- Liquid stool to be examined within ½ hour
- Solid stool to be examined within 1 hour.
- If delayed store in a refrigerator.

TYPES OF EXAMINATION

- MACROSCOPIC EXAMINATION:

color, volume, consistency, odour, blood, mucus, pus, and adult helminths.

- CHEMICAL EXAMINATION:

reactions, occult blood, fat, carbohydrate, protein, etc

- MICROSCOPIC EXAMINATION:

remnants of food, pus cells, macrophages, RBCs, crystals, bacteria, yeasts, molds, protozoa, helminths.

- STOOL CULTURE

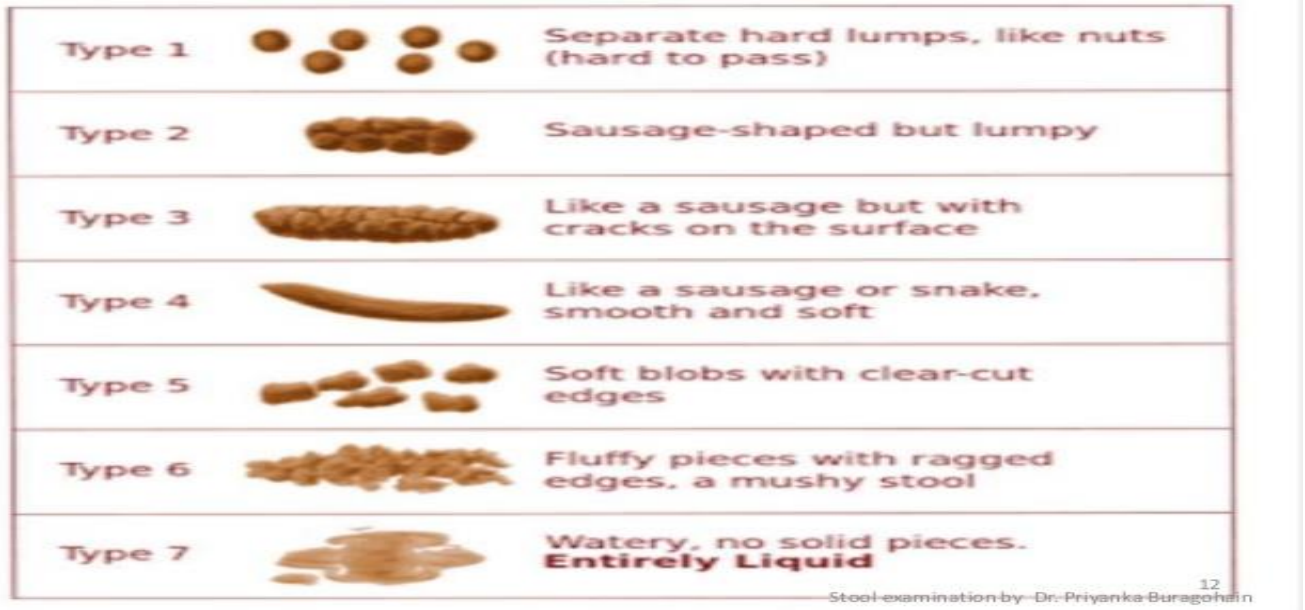
MACROSCOPIC EXAMINATION

*Amount: Normal is 150 g to 200 g/day and increased in steatorrhoea, diarrhoea, indigestion of carbohydrate.

*Color of stool: Human fecal matter is normally yellowish brown in colour which results from a combination of bile and bilirubin.

***Consistency or form:**

- Normal is soft but formed
  - Excessively hard/scybala- habitual constipation
  - Flattened or ribbon like-intake of excess of mineral oil, carcinoma of rectum, stricture of rectum
  - Soft, mushy, liquid and voluminous- diarrhea, intake of purgatives
  - Small numerous, largely mucus and blood with small amount of stool- dysenteries
  - Rice watery without fecal matter- Cholera



*Odour of stool: Normal odour of the stool is aromatic due to INDOLE and SKETOLE are the substances that produce normal odour formed by Intestinal bacterial fermentation and putrefaction.
Increased: A foul odour is caused by excessive protein and degradation of undigested protein, and excessive carbohydrate intake. Sickly sweet odour is produced by undigested Lactose.Sour rancid: fatty acid in milk indigestion (in children and adults), normal in infants. Putrid: severe diarrhoea of malignancy, gangrenous dysentry.

Reaction

- Normal is neutral
- Ph varies from 6.9 to 7.2
- pH is dependent on bacterial fermentation and putrefaction in the bowel.
- Alkaline – excess protein ingestion
- Acidic – excess carbohydrate ingestion

Mucus

- Small quantity of mucin is normal
- Small quantity – faeces from small gut
- Excessive quantity – infection of intestine
- Entirely mucus with little or no faeces and streaks of blood- dysentery, ileo colitis, intussusception

Blood

- Absent in normal faeces
- Formed stool with streaks of blood – lesion in sigmoid colon, rectum or anal canal
- Liquid stool with bright red blood, pus and mucus- bacillary dysentery, ulcerative colitis
- Semi formed stool with deep tarry black blood- melena
- Loose stool with deep cherry red blood- melena

نشاط(1/2/2)

Q\ fill in the blanks

1- The Amount Normal of stool is ……..
2- Color normal of stool is ……..
3- The Consistency or form of stool is ……

الوحدة الثانية - المحاضرة الثالثة - الزمن: 90 دقيقة

أهداف المحاضرة الثالثة:

يتوقع في نهاية الجلسة أن يكون الطالب قادراً على:

1-Making a slide to examine the stool
2- Examination of stool and writing an examination report for Macroscopic and microscopic examination

موضوعات المحاضرة الثالثة:

Examination of stool sample
a) Macroscopic examination of stool
b) microscopic examination of wet mounts

الأساليب والأنشطة والوسائل التعليمية

م	الأساليب والأنشطة التدريبية	الوسائل التدريبية
1	• محاضرة • مناقشة • سؤال وجواب • نشاط (1/3/2) نشاط	• جهاز حاسوب • جهاز عرض • سبورة • اوراق واقلام

خطة إجراءات تنفيذ المحاضرة الثالثه

الوحدة	المحاضرة	الإجراءات	الزمن بالدقيقة
الثانية	الثالثة	القاء المحاضرة مستخدما جهاز العرض والسبورة	90 دقيقة
		طرح بعض الاسئلة خلال القاء المحاضرة	
		نشاط (1/3/2) تمرين تعداد	

المادة العلمية:

Examination and collection of the stool (feces):
Examination of stool becomes necessary if gastrointestinal, symptoms diarrhea, dysentery is present. Most of parasites which inhabiting the gastrointestinal tract are found in the stool.

General stool examination(G.S.E)

Examination of stool sample (GSE) include: -

A. Macroscopic examination (by naked eye)
B. Microscopic examination (by microscope)

Macroscopic examination (by naked eye):

1- Consistency: normal feces is fresh, dense and semisolid, but abnormal stool sample is: solid (formed), watery (liquid), mucoid , bloody ,bloody with mucus.
2- Color: normal stool is (brown or little dark brown). But the other colors are abnormal like: yellow, black, green, red.
3- Presence of blood: the normal stool must be without blood.
4- Presence of mucus: little mucus found in normal stool but it is increased in pathogenic cases.
5- Presence of stones: you can observe it by using wooden stick.
6- Presence of parasites: whole worm like ascaris, or segments of worms.
7- Presence of foreign bodies.
8- Presence of food particles.

Microscopic examination (by microscope):

1- Parasitic findings:
   A- protozoa (trophozoite and cyst).
   B- Whole worm or segment (proglotted) of worm.
   C- Ova (eggs) of worms.
   D- Larva of worms.

2- Non- parasitic findings:
   A- bacteria: e.g. bacilli
   B- air bubbles
   C- fat droplets
   D- muscle fibers
   E- animal cells
   F- red blood cell (RBC)

G- pus cell
H- fungi
I- stones

Preparation of slide fresh stool:

1- take clean slide and put one drop of (normal saline) on one side and one drop of iodine solution on another side.
2- Take small amount of stool by mean of wood stick and first mix well with the drop of normal saline and then with the iodine solution.
3- Cover it with cover slide.
4- Examination in under microscope (zigzag line) using 10X and 40X objective lens.

نشاط (1/3/2)

Q\ Enumerate the Microscopic examination of stool only parasite finding

الوحدة الثانية - المحاضرة الرابعة - الزمن: 90 دقيقة

أهداف المحاضرة الرابعة:

يتوقع في نهاية الجلسة أن يكون الطالب قادراً على:

1- Enumerate of Types of solutions used in smear
2- What are the advantages and disadvantages of each solution:

موضوعات المحاضرة الرابعة:

- Types of solutions used in smear the advantages and disadvantages of each solution:
- Saline solution
- Iodine solutions
- *Eosin solution

الأساليب والأنشطة والوسائل التعليمية

م	الأساليب والأنشطة التدريبية	الوسائل التدريبية
1	• محاضرة • مناقشة • سؤال وجواب • نشاط (1/4/2) نشاط	• جهاز حاسوب • جهاز عرض • سبورة • اوراق واقلام

خطة إجراءات تنفيذ المحاضرة الرابعه

الوحدة	المحاضرة	الإجراءات	الزمن بالدقيقة
الثانية	الرابعة	القاء المحاضرة مستخدما مجموعة مواد تعريفية	90 دقيقة
		طرح بعض الاسئلة خلال القاء المحاضرة	
		نشاط (1/4/2) تمرين تعداد	

المادة العلمية:

The solutions that used in parasitic preparation include:

*Buffers
*Stains
*Preservatives
*Fixatives
*Liquid Media

1. **Normal Saline Solution (NSS)**

It is commonly used in various laboratory procedures like in the preparation of red cells Suspension for the cross match, for preparing dilutions of Reagents, for stool examinations, to make the dilutions in serological tests, diagnostic tests etc.

There are commercially prepared Normal saline solution available in the market but it can easily be prepared manually in the laboratory whenever required. The normal saline solution is simply the 0.85% Sodium chloride (NaCl) solution which can be prepared in the laboratory by dissolving the calculated amount of Sodium chloride crystals in the required quantity of Distilled water. The Normal saline solution is prepared as follows……

CALCULATION FOR NORMAL SALINE SOLUTION…

⇒ A normal saline solution is the 0.85% sodium chloride solution.

⇒That means 0.85 gm of sodium chloride in 100 ml distilled water.

⇒ For preparing 1 L normal saline solution we require,

W1 / V1 = W2 / V2 W1 = Required quantity of sodium chloride for 100 ml.

NS = 0.85 gm

V1 = Required volume for 0.85 gm sodium chloride to make

NS = 100 ml

W2 = Required Quantity of Sodium chloride to make the desired quantity of Normal Saline solution

V2 = 1000 ml (or desired quantity of Normal saline to be prepared)

0.85 gm / 100 ml = W2 gm / 1000 ml

0.85 gm × 1000 ml = W2 × 100 ml

W2 = 0.85 × 1000 ml / 100 ml

W2 = 8.5 gm

PROCEDURE FOR NORMAL SALINE SOLUTION….

⇒ Weigh 8.5 gm of Sodium chloride (NaCl) with the help of weighing scale.

⇒ Now take 500 ml of distilled water in the volumetric flask or in a Beaker and to this add 8.5 gm NaCl.
⇒ Swirl the flask gently to mix the contents or stir in case you are making the solution in a beaker with the help of stirrer.
⇒ When NaCl dissolves completely then add distilled water and make the final volume 1 liter.
⇒ Insert an air-tight stopper into the mouth of the volumetric flask and shake it gently to make the solution homogeneous or if you are using a beaker stir the solution well using a stirrer.
⇒ Now Autoclave the prepared normal saline solution at 121 °C, 15 psi pressure for 15 minutes. This will sterilize the solution and made it the Laboratory grade Normal saline solution which can be used in pathological as well as microbiological analysis.

2- Lugo's Iodine:

Reagents:
Potassium iodide (Kl) 10 gm*
Iodine crystals 5 gm*
* Distilled water 100 ml

Lugo's 5% iodine solution Procedure

1.Dissolve potassium iodide in 20-30 ml distilled water;
2.Slowly add iodine crystals to get a homogeneous solution;
3.Add the remaining water, mix well and filter.
4.Store in a tightly sealed brown bottle. The solution is stable for several weeks.
Advantage
* Can be classed as having a low acute toxicity . Potassium Iodide is not considered hazardous.
* Benefit in the preparation of MIF
* Stock for logo's iodine in another concentration

نشاط (1/4/2)

Q\ Enumerate of Types of solutions used in smear

الوحدة الثانية- المحاضرة الخامسة - الزمن: 90 دقيقة

أهداف المحاضرة الخامسة:

يتوقع في نهاية الجلسة أن يكون الطالب قادراً على:

1-The most important methods of preserving fecal samples.
2- Ingredients Polyvinyl Alcohol (PVA):
3- what is it Advantages and Disadvantages of PVA

موضوعات المحاضرة الخامسة:

- Preparation of preservatives and fixatives for mounted slides
- Formalin solution (5-7%)
- PVA (Polyvinyle alcohol) as fixative
- Schaudinns fixative

الأساليب والأنشطة والوسائل التعليمية

م	الأساليب والأنشطة التدريبية	الوسائل التدريبية
1	• محاضرة • مناقشة • سؤال وجواب • نشاط (1/5/2) نشاط	• جهاز حاسوب • جهاز عرض • سبورة • اوراق واقلام

خطة إجراءات تنفيذ المحاضرة الخامسة

الوحدة	المحاضرة	الإجراءات	الزمن بالدقيقة
الثانية	الخامسة	يقوم طالب بشرح سريع للمحاضرة السابقة	90 دقيقة
		القاء المحاضرة مستخدما جهاز العرض والسبورة	
		طرح بعض الاسئلة خلال القاء المحاضرة	
		نشاط (1/5/2) تعداد	

المادة العلمية:

Preservation methods for fecal specimens

- Preservation allows fecal samples to be examined after a delay in delivery or postage or testing.
- Many methods for the preservation of stool samples and permanent staining procedures.
- The most common fixatives are:
  - Polyvinyl Alcohol(PVA).
  - Formalin.
  - Merthiolate Iodine Formalin(MIF).
  - Sodium acetate Acetic acid Formalin (SAF).

The preservatives used have different effect on the various stages of the parasites

Buffered formalin (10%)

Composition

-Sodium phosphate, monobasic (NaH_2PO_4) 4.0 g

-Sodium phosphate, dibasic (Na_2HPO_4) 6.5 g

-Commercial formalin (37%-40%) 100 ml

- Distilled water or saline 900 ml

Procedure

1- -Dissolve the salts in distilled water
2- Add formalin and mix well.
3- -Store in a sealed container
4- Label the bottle Formalin &write the date. store on a shelf or in a cabinate. The solution will remain good for two years or more.

Advantages:

*Easy to prepare
*It has a long shelf life & commercially available.
* Suitable for concentration procedure (sedimentation techniques)
*Good preservation of morphology of helminth eggs, larvae, protozoan cysts, and coccidia
*Materials can be preserved for several years.
*Suitable for acid-fast, safranin, and chromotrope stains
*Compatible with immunoassay kits and epifluorescence microscopy
*Can be used for concentration techniques (sedimentation techniques)

*Neutral formalin (buffered with sodium phosphate) helps maintain organism morphology with prolonged storage.

The major disadvantage of formalin

- That permanent staining procedures can't be performed from formalin preserved stool samples.
- Formalin is corrosive & poisonous.
- Inadequate preservation of morphology of protozoan trophozoites.
- Can interfere with PCR, especially after extended fixation time.

Polyvinyl Alcohol(PVA):

Composition

Schaudinn's stock solution	93.5 ml
Glycerol	1.5 ml
Glacial acetic acid	5.0 ml
Polyvinyl alcohol	5 g

Advantages:

- ✓ Fixative for almost purpose.
- ✓ Has a long shelf life (months to years) in tightly sealed containers at room temperature.
- ✓ The preservation of the two stages of protozoa(trophozoite and cyst) is excellent, and also suitable for helminthes eggs and larvae.
- ✓ Compatible with immunoassay kits and UV fluorescence microscopy.
- ✓ The PVA is a plastic resin that serves as adhesive for the stool material, When the stool-PVA mixture is spread onto the glass slide, it adheres because of the PVA component.
- ✓ The greatest advantage of this fixative is that a permanent stain can be prepared from stool specimen preserved by PVA, giving excellent result with trichrome staining.
- ✓ Commercially available from a number of sources

Disadvantages:

- ❖ Contains mercury compounds (Schaudinn’s fixative), which is highly toxic to both man and the environment, and must be disposed of as toxic waste.
- ❖ Concentration methods can’t performed from the specimen preserved by PVA.
- ❖ Difficult to prepare in the laboratory.

- ❖ Some organisms (Trichuris trichiura eggs, Giardia lamblia cysts, Isospora belli oocysts) are not concentrated as well from PVA , and morphology of some ova and larvae may be distorted.
- ❖ Can interfere with PCR, especially after extended fixation time.

fixative used for the preservation of stool samples an overview of the advantages and disadvantages:

	Formalin	PVA
Toxicity	+/-	+++ (duo to Hg)
Shelf life	Long (months)	Long (months/years)
Preparation	Easy	Difficult
Quality of fixation	Egg: ++	Egg: ++
	Cyst: ++	Cyst: +++
	Troph's: +/-	Troph's: +++
Formalin ether concentration	Possible	Not possible
Permanent stained smear	Not possible	Only Trichrome

Schaudinn's fixative

Saturated solution of mercuric chloride (HgCl2)

Procedure

- Dissolve 10 g HgCl2 in 100 ml warm (not boiling) distilled water;
- Leave to cool (mercuric chloride crystals deposit);
- Filter off the clear supernatant;
- Store in a sealed glass bottle until use.
- Label the bottle (it is very important).

نشاط (1/5/2)

Q\ what is it Advantages and Disadvantages of PVA?

الوحدة الثانية - المحاضرة السادسة - الزمن: 90 دقيقة

أهداف المحاضرة السادسة:

يتوقع في نهاية الجلسة أن يكون الطالب قادراً على:

1- What is it the purpose to use concentration methods
2- Enumerate the types of Concentration.
3- what is it the material and methods used of Sedimentation &Floatation

موضوعات المحاضرة السادسة:

- Concentration methods
- Types
- Purpose to use concentration methods

الأساليب والأنشطة والوسائل التعليمية

م	الأساليب والأنشطة التدريبية	الوسائل التدريبية
1	• محاضرة • مناقشة • سؤال وجواب • نشاط (1/6/2) نشاط	• جهاز حاسوب • جهاز عرض • سبورة • اوراق واقلام

خطة إجراءات تنفيذ المحاضرة السادسه

الوحدة	المحاضرة	الإجراءات	الزمن بالدقيقة
الثانية	السادسة	القاء المحاضرة مستخدما جهاز العرض والسبورة	90 دقيقة
		طرح بعض الاسئلة خلال القاء المحاضرة	
		نشاط (1/6/2) تمرين فراغات	

المادة العلمية:

Concentration procedures

A routine part of complete stool examination for parasites Allows detection of small numbers of parasites that may be missed by using only a direct wet smear Designed to separate protozoan organisms & helminths eggs & larvae from fecal debris by centrifugation and/or differences in specific gravity.

There are two types of concentration procedures:

1- Sedimentation

Concentration procedures are used to concentrate eggs, larvae and protozoan cysts to increase the sensitivity for parasite detection in stool samples, specially in cases of mild infection.

All type of eggs and cysts can be recovered by sedimentation. Parasites settle down more rapidly by centrifugation.

Larger food particles can be removed prior to centrifugation by filtering through a sieve with a pore size enough to retain those particles. The efficiency of detection is increased by adding formalin for fixation & preservation of parasites, and ethyl acetate to remove organic material especially fat.

Materials:

- 10% formalin
- Ethyl acetate
- Centrifuge tubes
- Centrifuge stand
- Funnel
- Gauze
- Spatula
- Pipettes
- Microscope slides & coverslips

Method:

Step 1:

Emulsify 0.5-1 g of stool in 7 ml of 10% formalin in a tube.

Step 2:

Pour the stool emulsion onto a double layer of gauze in a funnel and collect in a beaker

Step 3:

Wash the stool through the gauze using formalin. This washes the parasites through but filters out the larger pieces of debris.

Step 4:

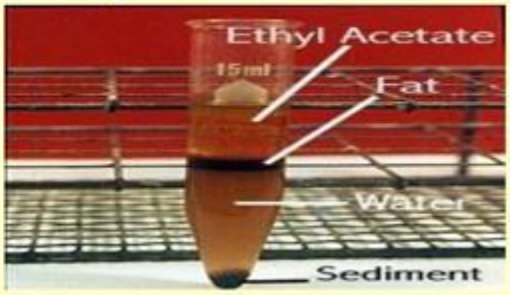
Pour the filtrate into a 15 ml centrifuge tube and add 3 ml of ethyl acetate, mix well (~1 min) by hand.

Step 5:

Centrifuge the mixture for 5 minutes at 500 g (~2000rpm).

Four layers are formed in the tube after centrifugation

(Formol-ethyl acetate concentration method)



Step 7:

Mix the sediment well

Step 6:

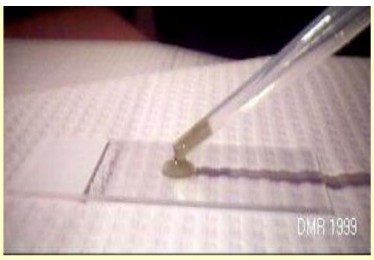
Loosen the fatty plug at the top of the tube with an applicator stick and invert the tube to discard the supernatant.

* Few drops should be kept with the sediment.

Step 9:

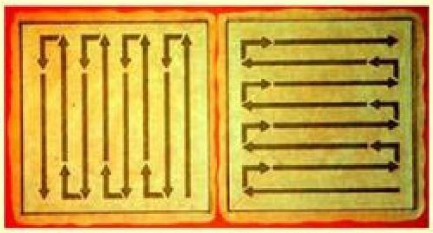
Place one edge of the coverslip on the drop and carefully pull the drop along. When the coverslip is in position gently let it drop.

Step 8:



Place a drop on the slide.

Step 11:



- Scan the entire coverslip systematically as illustrated using the 10X objective.
- If you suspect cysts or trophozoites, use higher magnification.
- 1/3 of the slide should be scanned using a higher magnification.

Step 10:

If there is a thick and thin area, the cysts and ova will generally be found in the thick portion of the mount.

2-Floatation

Floatation procedure Permits the separation of protozoa cysts & oocysts, and certain helminthes eggs & larvae through the use of a liquid with a high specific gravity. Parasites are recovered in the surface film, and the debris remains in the

bottom of the tube.

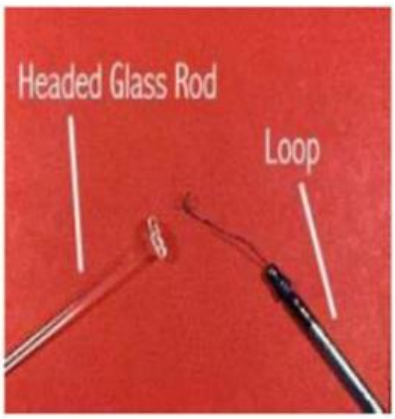
Disadvantages:

- Some helminthes eggs (operculated eggs and/or very dense eggs such as unfertilized Ascaris eggs) do not concentrate well with the floatation method.
- Protozoan cysts become distorted and difficult to identify

Floatation Method

1. Emulsify 2 to 3 grams of feces (a size of a grape) into 5 ml of ZnSO4 solution in a dish.
2. Push the emulsion through the strainer into a 15 ml centrifuge tube then fill the tube with zinc sulfate solution.
3. Centrifuge for 2 min at 1500 - 2000 rpm.
4. Using a headed-rod or loop, remove a sample from solution surface & place on a microscope slide.
5. Add a drop of iodine to stain cysts & ova and don the examination.

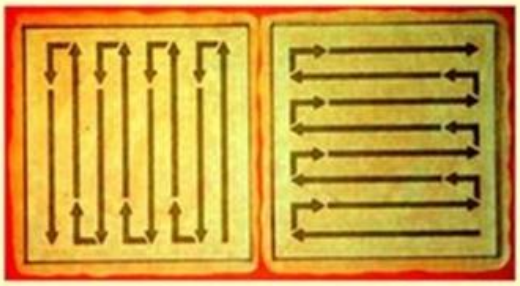
Zinc Sulfate Floatation Method



Step 10:

If there is a thick and thin area, the cysts and ova will generally be found in the thick portion of the mount.

Step 11:



- Scan the entire coverslip systematically as illustrated using the 10X objective.
- If you suspect cysts or trophozoites, use higher magnification.
- 1/3 of the slide should be scanned using a higher magnification.

نشاط (1/6/2)

Q\ fill in the blanks

1- **the main material in Sedimentation Concentration of Stool is …….**
2- **the main material in Floatation Concentration of Stool is ……..**

الوحدة الثالثة - المحاضرة الأولى - الزمن: 90 دقيقة

أهداف المحاضرة الاولى:

يتوقع في نهاية الجلسة أن يكون الطالب قادراً على:

1- Discuss the pathogenesis and clinical aspects of infections.
2- Describe the general epidemiological aspects and transmission patterns of diseases caused by *Entamoeba histolytica*.
3- Identify the methods and procedures of laboratory diagnosis of pathogenic *Entamoeba histolytica* in clinical specimens.

موضوعات المحاضرة الاولى:

1- Differentiation of pathogenic
2- *Entamoeba histolytica* and the morphologically identical non-pathogenic
3- using immunological assays

الأساليب والأنشطة والوسائل التعليمية

م	الأساليب والأنشطة التدريبية	الوسائل التدريبية
1	• محاضرة • مناقشة • سؤال وجواب • نشاط (1/1/3) نشاط	• جهاز حاسوب • جهاز عرض • سبورة • اوراق واقلام

خطة إجراءات تنفيذ المحاضرة الاولى

الوحدة	المحاضرة	الإجراءات	الزمن بالدقيقة
الأولى	الأولى	القاء المحاضرة مستخدما جهاز العرض والسبورة	90 دقيقة
		طرح بعض الاسئلة خلال القاء المحاضرة	
		نشاط (1/1/3) تمرين رسم	

المادة العلمية:

Pathogenic amoeba

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 undergodivision 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.

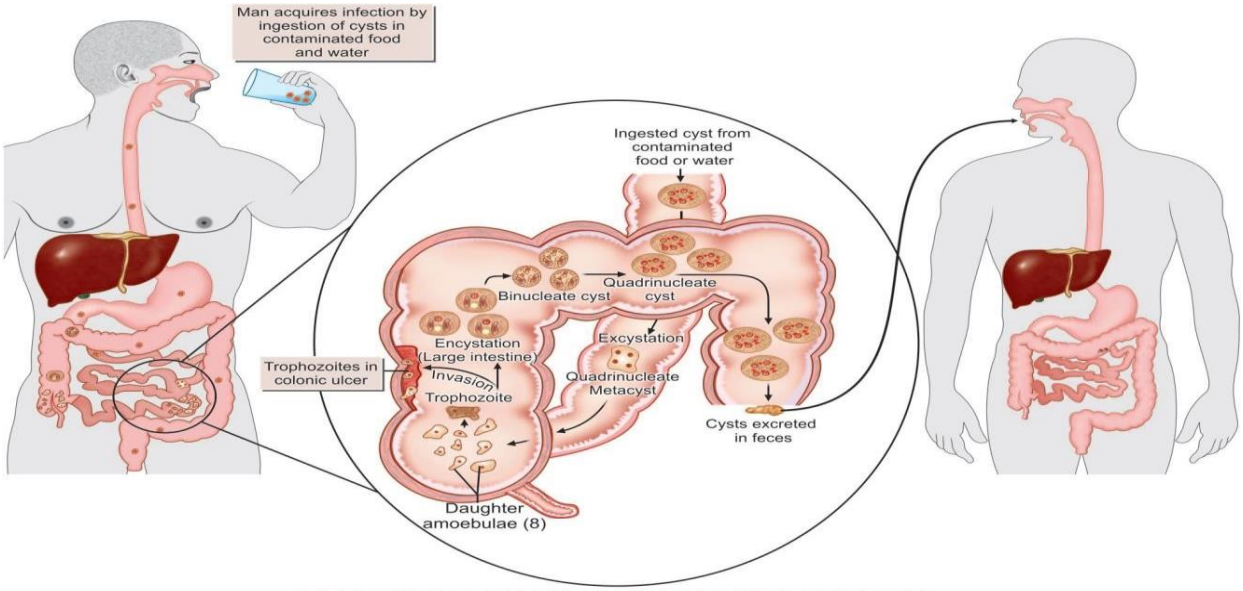
Not all strains of E. histolytica are pathogenic or invasive. Differentiation between pathogenic and nonpathogenic strains can be made by susceptibility to complement-mediated lysis and phagocytic activity or by the use of genetic markers or monoclonal antibodies and zymodeme analysis.

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

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.

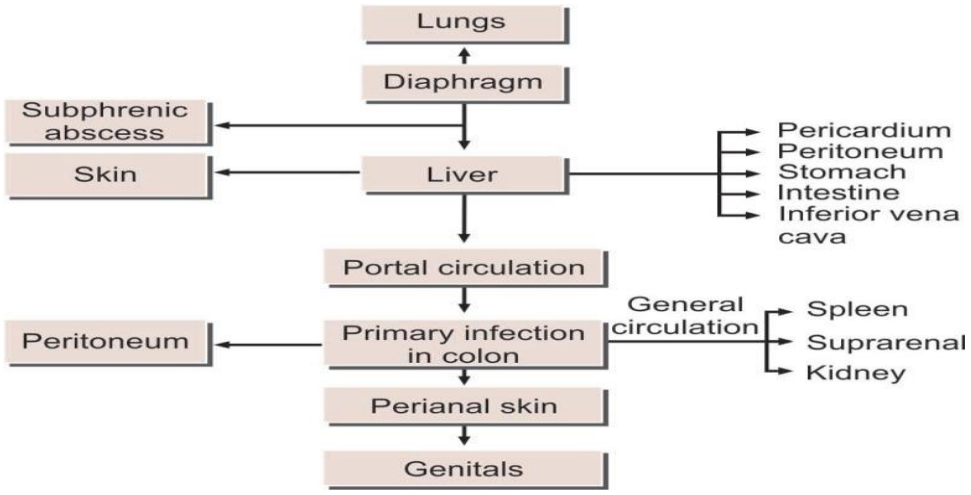
The patient is usually afebrile and nontoxic. In fulminant colitis, there is confluent ulceration and necrosis of colon. The patient is febrile and toxic. Intestinal amoebiasis does not always result in dysentery. Quite often, there may be only diarrhea or vague abdominal symptoms popularly called 'uncomfortable 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.

نشاط (1/1/3)

Q\ Draw the life cycle of *Entamoeba histolytica*

الوحدة الثالثة - المحاضرة الثانية- الزمن: 90 دقيقة

أهداف المحاضرة الثانية:

يتوقع في نهاية الجلسة أن يكون الطالب قادراً على:

1- Enumerate and explain morphology of Acanthamoeba
2- List the disease symptoms of Acanthamoeba.
3- diagnosis of Acanthamoeba.
4- Comparison between Naegleriafowleri & Acanthamoeba.

موضوعات المحاضرة الثانية:

1- Naegleriafowleri&Acanthamoeba spp
2- Morphology, habitat, mode of infection, infective stage
3- life cycle and laboratory diagnosis

الأساليب والأنشطة والوسائل التعليمية

م	الأساليب والأنشطة التدريبية	الوسائل التدريبية
1	• محاضرة • مناقشة • سؤال وجواب • نشاط (1/2/3) نشاط	• جهاز حاسوب • جهاز عرض • سبورة • اوراق واقلام

خطة إجراءات تنفيذ المحاضرة الثانيه

الوحدة	المحاضرة	الإجراءات	الزمن بالدقيقة
الثالثة	الثانية	يقوم طالب برسم احد اطوار الاميبا للمحاضرة السابقة	90 دقيقة
		القاء المحاضرة مستخدما جهاز العرض والسبورة	
		طرح بعض الاسئلة خلال القاء المحاضرة	
		نشاط (1/2/3) تمرين مقارنة	

المادة العلمية:

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.

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.

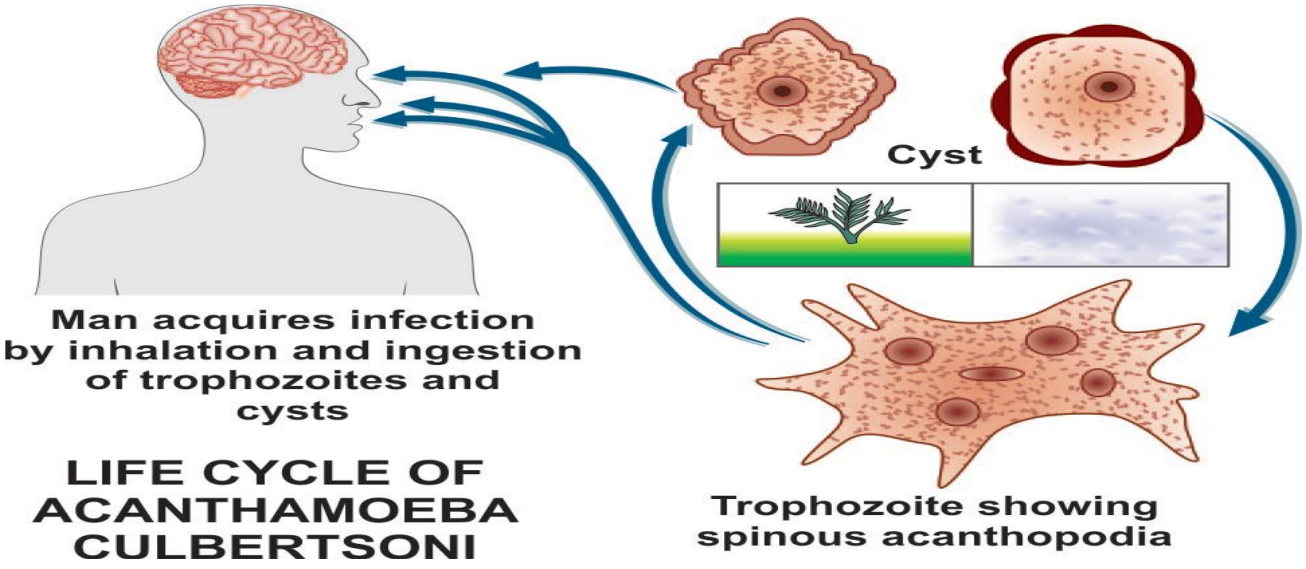
*The cysts are present in all types of environment, all over the world.

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.

After inhalation of aerosol or dust containing trophozoites and cysts, the trophozoites reach the lungs and from there, they invade the central nervous system through the blood stream, producing granulomatous amoebic encephalitis (GAE).



Pathogenesis and Clinical Features

*Infection usually occurs in patients with immunodeficiency, diabetes, malignancies, malnutrition, systemic lupus erythematosus (SLE), or alcoholism.

*The parasite spreads hemotogenously into central nervous system. Subsequent invasion of the connective tissue and induction of pro-inflammatory responses lead to neuronal damage that can be fatal within days.

*A postmortem biopsy reveals severe edema and hemorrhagic necrosis.

Clinical Disease

It presents chiefly as 2 chronic conditions keratitis and encephalitis.

*Acanthamoeba keratitis: An infection of the eye that typically occurs in healthy persons and develops from the entry of the amoebic cyst through abrasions on the cornea. Majority of such cases have been associated with the use of contact lenses. The picture resembles that of severe herpetic keratitis with a slow relapsing course, but the eye is severely painful in the amoebic infection. Unilateral photophobia, excessive tearing, redness and foreign

body sensation are the earliest signs and symptoms; disease is bilateral in some contact lens users. Keratitis and uveitis can result in permanent visual impairment or blindness.

*Granulomatous amoebic encephalitis (GAE): It is a serious infection of the brain and spinal cord that typically occurs in persons with a compromised immune system.

€ GAE is believed to follow inhalation of the dried cysts. The incubation period is long and the evolution of the illness is slow. Clinical picture is that of intracranial space-occupying lesions with seizures, pareses, and mental deterioration.

*Disseminated infection: In immunocomprised states like acquired immunodeficiency syndrome (AIDS), a widespread infection can affect skin, lungs, sinuses, and other organs independently or in combination.

Laboratory Diagnosis

*Diagnosis of amoebic keratitis is made by demonstration of the cyst in corneal scrapings by wet mount, histology and culture. Growth can be obtained from corneal scrapings inoculated on nutrient agar, overlaid with live or dead Escherichia coli and incubated at 30°C.

*Diagnosis of GAE is made by demonstration of trophozoites and cysts in brain biopsy, culture, and immofluroscence microscopy using monoclonal antibodies. CSF shows lymphocytic pleocytosis, slightly elevated protein levels, and normal or slightly decreased glucose levels.

CT scan of brain provides inconclusive findings.

Treatment

In acanthamoeba keratitis, current therapy involves topical administration of biguanide or chlorhexadine with or without diamidine agent. In severe cases, where vision is threatened,

penetrating keratoplasty can be done. No effective treatment is available for GAE. Multidrug combinations including pentamidine, sulfadiazine, rifampicin, and fluconazole are being used with limited success.

	Naegleria	*Acanthamoeba*
Disease	Primary amoebic meningoencephalitis (PAM)	Granulomatous amoebic encephalitis (GAE) and keratitis
Portal of entry	Nose	Upper Respiratory tract (?), cornea
Clinical course	Acute	Subacute or chronic
Pathogenecity	Acute suppurative inflammation	Granulomatous inflammation
Morphological forms	3 stages: trophozoite, cyst and flagellate form	2 stages: trophozoite and cyst flagellate form absent
Trophozoite	10–20 µm, with a single pseudopodia	20–50 µm, with spine-like pseudopodia
Cyst	7–10 µm, round with smooth wall	15–25 µm, polygonal double-walled with wrinkled surface
Nuclear division	By promitosis, nucleolus divides, nuclear membrance persists	Nuclear membrance dissolves
WBC in CSF	Predominantly neutrophils	Predominantly lymphocytes

نشاط (1/2/3)

Q\ Comparison between Naegleriafowleri & Acanthamoeba

الوحدة الرابعة - المحاضرة الأولى - الزمن: 90 دقيقة

أهداف المحاضرة الاولى:

يتوقع في نهاية الجلسة أن يكون الطالب قادراً على:

1- Define the flagellates
2- Enumerate groups of flagellates
3- Diagnosis of *Trypanosoma*

موضوعات المحاضرة الاولى:

1- Tissue flagellates
2- Genus *Trypanosoma*
3- Laboratory diagnosis; routine methods, immunological Assays and molecular

الأساليب والأنشطة والوسائل التعليمية

م	الأساليب والأنشطة التدريبية	الوسائل التدريبية
1	• محاضرة • مناقشة • سؤال وجواب • نشاط (1/1/4) نشاط	• جهاز حاسوب • جهاز عرض • سبورة • اوراق واقلام

خطة إجراءات تنفيذ المحاضرة الاولى

الوحدة	المحاضرة	الإجراءات	الزمن بالدقيقة
الرابعة	الأولى	القاء المحاضرة مستخدما جهاز العرض والسبورة	90 دقيقة
		طرح بعض الاسئلة خلال القاء المحاضرة	
		نشاط (1/1/4) تمرين تقرير	

المادة العلمية:

Flagellates

Flagellates are unicellular microorganisms. Their locomotion is by lashing a tail-like appendage called a flagellum or flagella and reproduction is by simple binary fission.

There are three groups of flagellates:

1- Luminal flagellates: Giardia lamblia

2- Hemoflagellates: Trypanosoma species. Leishmania species.

3- Genital flagellates: Trichomonas vaginalis

***Trypanosoma Brucei Gambiense* (West African Trypanosomiasis)**

History and Distribution

Trypanosomiasis is believed to have been existing in tropical

Habitat

Trypanosomes live in man and other vertebrate host. They are essentially a parasite of connective tissue, where they multiply rapidly and then invade regional lymph nodes, blood, and finally may involve central nervous system

Morphology

Vertebrate Forms

In the blood of vertebrate host, T. brucei gambiense exists as trypomastigote form, which is highly pleomorphic

It occurs as a long slender form, a stumpy short broad form with attenuated or absent flagellum, and an intermediate form

*The trypomastigotes are about 15–40 μm long and 1.5– 3.5 μm broad

¾¾In fresh blood fi lms, trypomastigotes are seen as colorless, spindleshaped bodies that move rapidly, spinning around the red cells

*In smears stained with Giemsa or other Romanowsky's stain, the cytoplasm appears pale blue and the nucleus appears red. The kinetoplast appears as a deep red dot and volutin

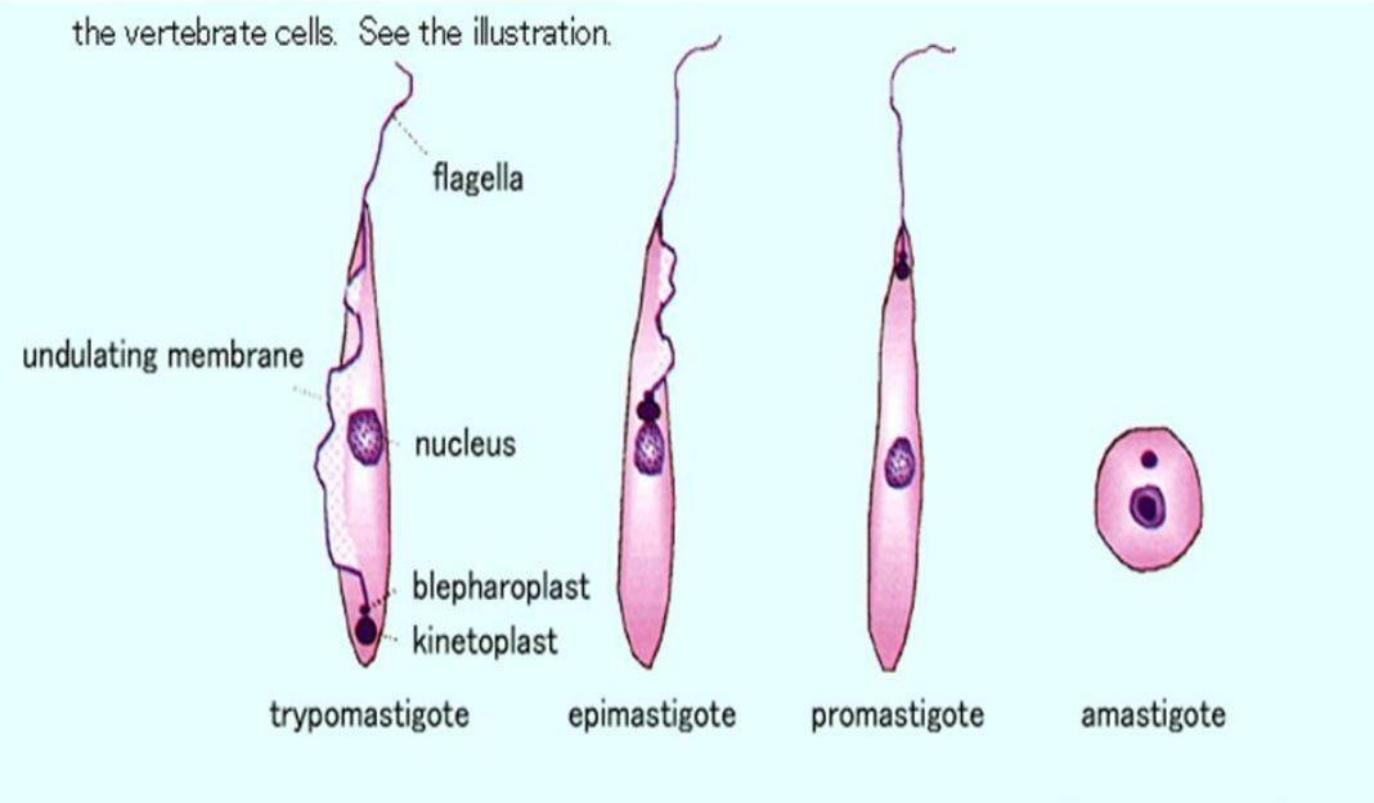
granules stain deep blue. The undulating membrane appears pale blue and the flagellum red.

Insect Forms

:In insects, it occurs in 2 forms

Epimastigotes

Metacyclic trypomastigote forms



Antigenic Variation

Trypanosomes exhibit unique antigenic variation of their glycoproteins

There is a cyclical fluctuation in the trypanosomes in the blood of infected vertebrates after .every 7–10 days

Each successive wave represents a variant antigenic type (VAT) of trypomastigote possessing variant surface specific antigens (VSSA) or variant surface glycoprotein (VSG) coat antigen.

It is estimated that a single trypanosome may have as many as 1,000 or more VSG genes that help to evade immune response. Besides this, trypanosomes have other mechanisms also that help them to evade host immune responses

Life Cycle

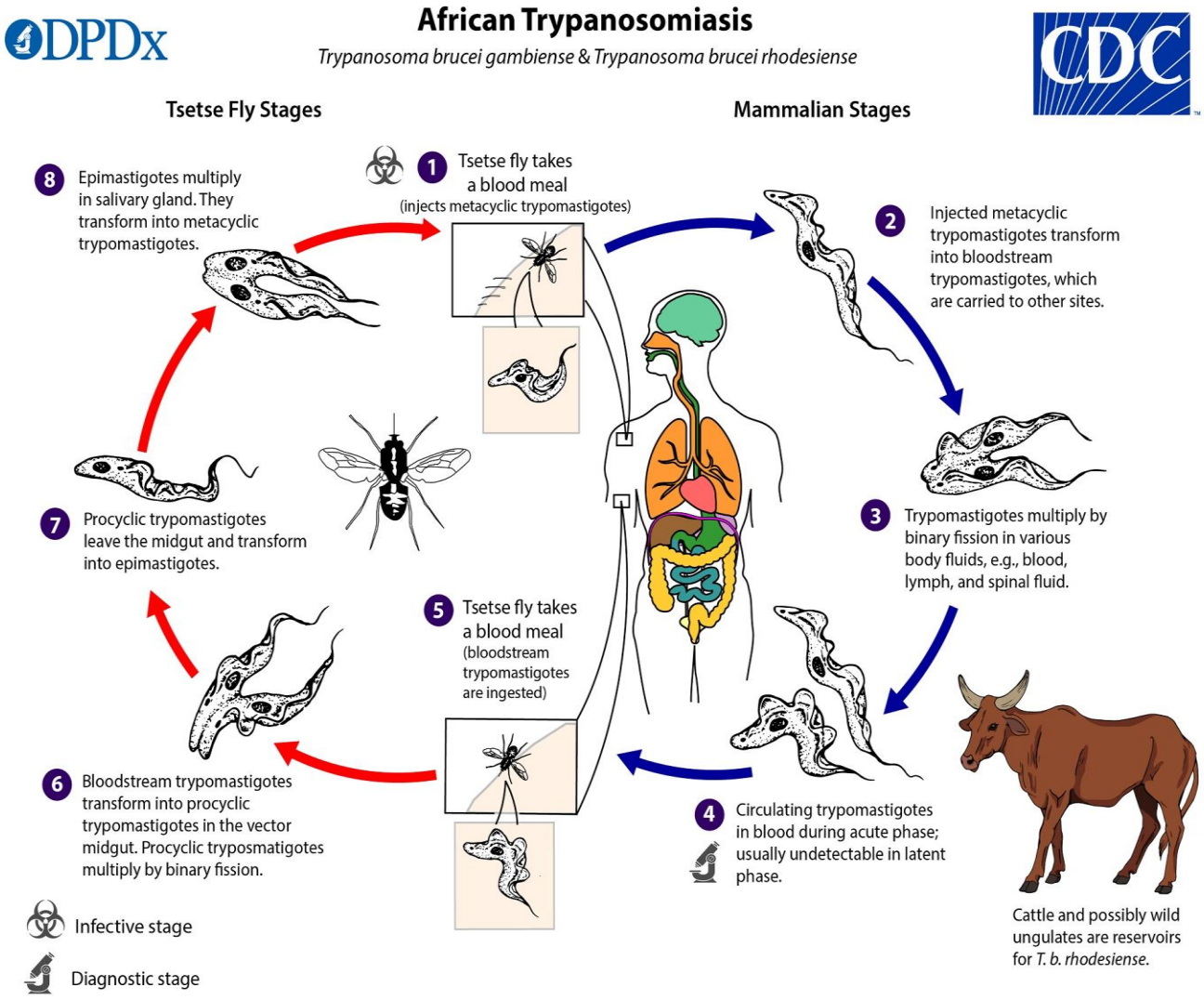
.T. brucei gambiense passes its life cycle in 2 hosts

Vertebrate host: Man, game animals, and other domestic animals

Invertebrate host: Tsetse fly.

Both male and female tsetse fly of Glossina species (G. palpalis) are capable of transmitting the disease to humans. These flies' dwell on the banks of shaded streams, wooded savanna, and agricultural areas.

Infective form: Metacyclic trypomastigote forms are infective to humans.



Mode of transmission:

*By bite of tsetse fly

*Congenital transmission has also been recorded.

Reservoirs: Man is the only reservoir host, although pigs and others domestic animals can act as chronic asymptomatic carriers of the parasite.

Development in Man and Other Vertebrate Hosts.

*Metacyclic stage (infective form) of trypomastigotes is inoculated into a man (definitive host) through skin when an infected tsetse fly takes a blood meal.

The parasite transforms into slender forms that multiply asexually for 1–2 days before entering the peripheral blood and lymphatic circulation.

*These become ‘stumpy’ via intermediate forms and enter the blood stream.
In chronic infection, the parasite invades the central nervous system.
*Trypomastigotes (short plumpy form) are ingested by tsetse fly (male or female) during blood meal.

Development in Tsetse Fly

*In the midgut of the fly, short stumpy trypomastigotes develop into long, slender forms and multiply.
*After 2–3 weeks, they migrate to the salivary glands, where they develop into epimastigotes, which multiply and fill the cavity of the gland and eventually transform into the infective metacyclic trypomastigotes.
*Development of the infective stage within the tsetse fly requires 25–50 days (extrinsic incubation period).
*Thereafter, the fly remains infective throughout its life of about 6 months.

Pathogenecity and Clinical Features

T. brucei gambiense causes African trypanosomiasis (West African sleeping sickness).
The illness is chronic and can persist for many years.
*There is an initial period of parasitemia, following which parasite is localized predominatly in the lymph nodes.
*A painless chancre (trypanosomal chancre) appears on
skin at the site of bite by tsetse fly, followed by intermittent fever, chills, rash, anemia, weight loss, and headache.
*Systemic trypanosomiasis without central nervous system involvement is referred to as stage I disease.
In this stage, there is hepatosplenomegaly and lymphadenopathy, particularly in the posterior cervical region (Winterbottom’s sign).

*Myocarditis develops frequently in patients with stage I disease and is especially common in T. brucei rhodesiense infections.
*Hematological manifestations seen in stage I include anemia, moderate leucocytosis, and thrombocytopenia. High levels of immunoglobulins mainly immunoglobulin IgM are a constant feature.
*Stage II disease involves invasion of central nervous system. With the invasion of central nervous system, which occurs after several months, the ‘sleeping sickness’ starts. This is marked by increasing headache, mental dullness, apathy, and day time sleepiness. The patient falls into profound coma followed by death from asthenia. Histopathology shows chronic meningoencephalitis.

The meanings are heavily infiltrated with lymphocytes, plasma cells, and morula cells, which are atypical plasma cells containing mulberry-shaped masses of IgA. Brain vessels show perivascular cuffi ng. This is followedby infiltration of the brain and spinal cord, neuronal degeneration, and microglial proliferation.
*Abnormalities in cerebrospinal fl uid include raised intracranial pressure, pleocytosis, and raised total protein concentrations.

نشاط (1/1/4)

Q\Write a report on a parasite *Trypanosoma Brucei Gambiense*

الوحدة الرابعة - المحاضرة الثانية - الزمن: 90 دقيقة

أهداف المحاضرة الثانية:

يتوقع في نهاية الجلسة أن يكون الطالب قادراً على:

1- Understanding History and Distribution of *Leishmania*
2- Enumarate of Morphology of *Leishmania*
3- Draw the life cycle of *Leishmania Donovani*

موضوعات المحاضرة الثانية:

1- Genus *Leishmania*
2- History and Distribution
3- Morphology and life cycle
4- Mode of transmission

الأساليب والأنشطة والوسائل التعليمية

م	الأساليب والأنشطة التدريبية	الوسائل التدريبية
1	• محاضرة • مناقشة • سؤال وجواب • نشاط (1/2/4) نشاط	• جهاز حاسوب • جهاز عرض • سبورة • اوراق واقلام

خطة إجراءات تنفيذ المحاضرة الثانية

الوحدة	المحاضرة	الإجراءات	الزمن بالدقيقة
الرابعة	الثانية	القاء المحاضرة مستخدما جهاز العرض والسبورة	90 دقيقة
		طرح بعض الاسئلة خلال القاء المحاضرة	
		نشاط (1/2/4) تمرين تعريف وتعداد	

المادة العلمية:

Leishmania

General Characteristics

The genus Leishmania is named after Sir William Leishman, who discovered the flagellate protozoa causing Kala azar, the Indian visceral leishmaniasis.

*All members of the genus Leishmania are obligate intracellular parasites that pass their life cycle in 2hosts: the mammalian host and the insect vector, female sand fly.

*In humans and other mammalian hosts, they multiply within macrophages, in which they occur exclusively in the amastigote form, having an ovoid body containing a nucleus and kinetoplast.

*Spindle shaped body and a single flagellum arising from anterior end.

*Leishmaniasis has an immense geographical distribution in the tropics and subtropics of the world, extending through most of the Central and South America, part of North America, Central and Southeast Asia, India, China, the Mediterranean region, and Africa.

*The disease affects the low socioeconomic group of people. Overcrowding, poor ventilation, and collection of organic material inside house facilitate its transmission.

Across the tropics, 3 different diseases are caused by various species of genus Leishmania. These are:

Visceral leishmaniasis: The species L. donovani complex infecting internal organs (liver, spleen, and bone marrow) of human is the causative parasite.

Cutaneous leishmaniasis: The species L. tropica complex, L. aethiopica, L. major and L. Mexicana complex are the causative parasite.

Mucocutaneous leishmaniasis: It is caused by the L. braziliensis complex.

Classification

The genus Leishmania includes a number of diff erent varieties and subspecies, which differ in several features such as antigenic structure, isoenzymes, and other biochemical characteristics, growth properties, host specifi city, etc. Leishmania species can also be classified on the basis of geographical distribution. The various manifestations of leishmaniasis and Leishmania species causing them.

Classification of *Leishmania* based on Geographical Distribution

Old world leishmaniasis	New world leishmaniasis
Leishmania donovani	*Leishmania braziliensis complex*
Leishmania infantum	*Leishmania mexicana complex*
Leishmania tropica	*Leishmania chagasi*
Leishmania major	*Leishmania peruviana*
Leishmania aethiopica	

Species	Disease	Geographical distribution	Vector	Reservoir	Transmission
Leishmania donovani	Visceral leishmaniasis (Kala-azar or dumdum fever)	Middle East, Africa, and Indian Subcontinent	*Phlebotomus argentipes, Phlebotomus orientalis*	Humans	Anthroponotic, occasionally zoonotic
Leishmania infantum	Visceral leishmaniasis, cutaneous leishmaniasis	Mediterranean Coast, Middle East, and China.	*Phlebotomus perniciousus, Phlebotomus ariasi, Phlebotomus papatasi*	Dog, fox, jackal, and wolf	Zoonotic
Leishmania chagasi	Visceral leishmaniasis	Tropical South America	*Lutzomyra longipalpis*	Fox and wild canines	Zoonotic
Leishmania tropica	Cutaneous Leishmaniasis (oriental sore, Baghdad boil)	Middle East and Central Asia	*Phlebotomus sergenti*	Humans	Anthroponotic
Leishmania major	Cutaneous leishmaniasis	Africa, Indian Subcontinent, and Central Asia	*Phlebotomus papatasi, Phlebotomus duboscqi*	Gerbil	Zoonotic
Leishmania aethiopica	Cutaneous and diffuse cutaneous leishmaniasis	Ethiopia and Kenya	*Phlebotomus longipes Phlebotomus pedifer*	Hydraxes	Zoonotic
Leishmania braziliensis complex	Mucocutaneous leishmaniasis (Espundia)	Tropical South America	*Lutzomyra umbratilis*	Forest rodents and peridomestic animals	Zoonotic
Leishmania mexicana complex	Mucocutaneous leishmaniasis (Chiclero's ulcer)	Central America and Amazon basin	*Lutzomyra olmeca, Lutzomyra flairscutellata*	Forest rodents and marsupials	Zoonotic

Old World Leishmaniasis
Leishmania Donovani

L. donovani causes visceral leishmaniasis or Kala-azar. It also causes the condition, Post Kala-azar Dermal Leishmaniasis (PKDL).

History and Distribution

Sir William Leishman in 1900, observed the parasite in spleen smears of a soldier who died of 'Dumdum fever' or Kala azar contracted at Dum Dum, Calcutta. Leishman reported this finding from London in 1903. In the same year, Donovan also reported the same parasite in spleen smears of patients from Madras. The name Leishmania donovani was, therefore given to this parasite. The amastigote forms of the parasite as seen in smears from patients are called Leishman Donovan (LD) bodies.

*__Visceral leishmaniasis or Kalaazar__

Is a major public health problem in many parts of world. According to the World Health Organization (WHO), a total of 5,00,000 cases of visceral leishmaniasis occur every year. Of these new cases, 90% are found in the Indian subcontinent and Sudan and Brazil. The disease occurs in endemic, epidemic, or sporadic forms. Major epidemics of the disease are currently found in India, Brazil, and Sudan.

*India, beginning in the mid1970s, assumed epidemic proportions in 1977 and involved over 1,10,000 cases in humans. Initially, the disease was confi ned to Bihar (Muzaff arpur, Samastipur, Vaishali, and Sitamarhi). Since then, the cases are increasing and involving newer areas. The epidemic extended to West Bengal and fi rst outbreak occurred in 1980 in Malda district.

*At present, the disease has established its endemicity in 31 districts in Bihar, 11 districts in West Bengal, 5 districts in Jharkhand, and 3 districts in Uttar Pardesh. Sporadic cases have been reported from Tamil Nadu, Maharashtra, Karnataka, and Andhra Pradesh.

Habitat

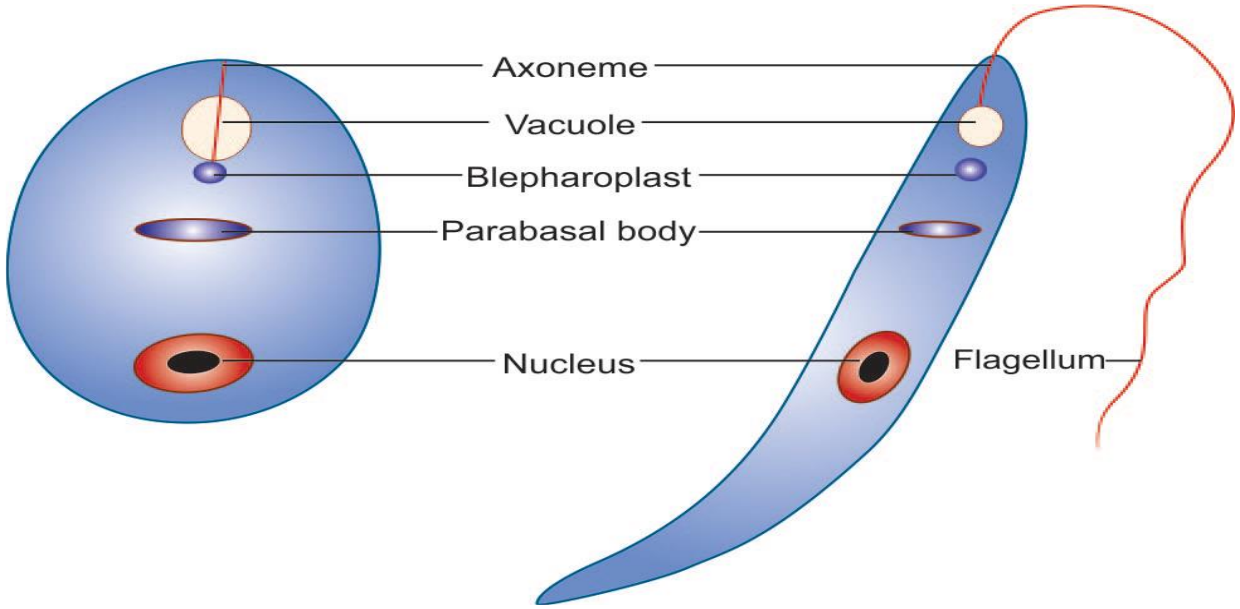
The amastigote (LD body) of L. donovani is found in the reticuloendothelial system. They are found mostly within the macrophages in the spleen, liver, bone marrow and less often in other locations such as skin, intestinal mucosa, and mesenteric lymph nodes.

Morphology

The parasite exists in 2 forms mammals.

*__Promastigote__: form: in the sandfl y and in artifi cial culture.

*__Amastigote__: The amastigote form (LD body) is an ovoid or rounded cell, about 2–4 μm in size.
*It is typically intracellular, being found inside macrophages, monocytes, neutrophils, or endothelial cells.
*They are also known as LD bodies.
*Smears stained with Leishman, Giemsa, or Wright's stain show a pale blue cytoplasm enclosed by a limiting membrane.
*The large oval nucleus is stained red. Lying at the right angles to nucleus, is the red or purple stained kinetoplast.
*In well stained preparations, the kineloplast can be seen consisting of a parabasal body and a dot like blepharoplast with a delicate thread connecting the two. The axoneme arising from the blepharoplast extends to the anterior tip of the cell.
*Alongside the kinetoplast a clear unstained vacuole can be seen.
*Flagellum is absent. Promastigote. It is a flagellar stage and is present in insect vector, sand fly and in cultures.
*The promastigotes, which are intitially short, oval or pear-shaped forms, subsequently become long spindle shaped cells, 15–25 μm in length and 1.5–3.5 μm in breadth. A single nucleus is situated at the center. The kinetoplast lies transversely near the anterior end.
*The flagellum is single, delicate, and measures 15–28 μm.
*Giemsa or Leishman-stained films show pale blue cytoplasm with a pink nucleus and bright red kinetoplast.



Morphology of *Leishmania donovani*.

A. Amastigote (LD body)

B. Promastigote

*A vacuole is present near the root of the flagellum.

*There is no undulating membrane.

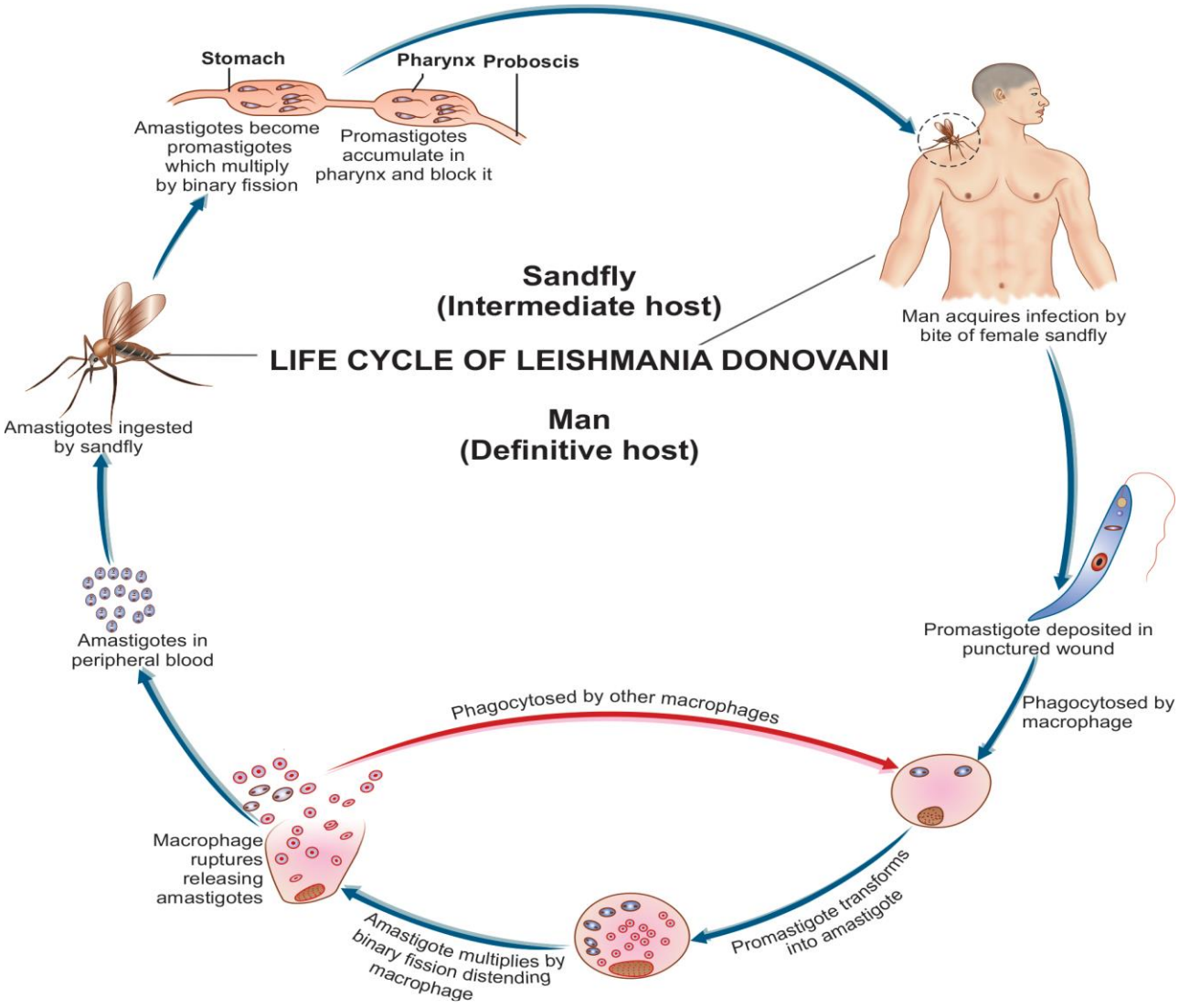
*Promastigote forms, which develop in artificial cultures, have the same morphology as in the sandfly.

Life Cycle

L. donovani completes its life cycle in 2 hosts. Definitive host: Man, dog, and other mammals.

Vector: Female sandfly (Phlebotomus species).

Infective form: Promastigote form present in midgut of female sandfly.



Mode of transmission:

Humans acquire by bite of an infected female sandfly. It can also be transmitted vertically from mother to fetus, by blood transfusion, and accidental inoculation in the laboratory.

Incubation period: Usually 2–6 months, occasionally it may be as short as 10 days or as long as 2 years.

*The sandfly regurgitates the promastigotes in the wound caused by its proboscis.

*These are engulfed by the cells of reticuloendothelial system (macrophages, monocytes, and polymorphonuclear leucocytes) and change into amastigote (LD body) within the cells.
*The amastigote multiplies by binary fission, producing numerous daughter cells that distend the macrophage and rupture it. The liberated daughter cells are in turn, phagocytosed by other macrophages and histiocytes. Small number of LD bodies can be found in peripheral blood inside neutrophils or monocytes.
*When a vector sandfly feeds on an infected person, the amastigotes present in peripheral blood and tissue fluids enter the insect along with its blood meal. In the midgut (stomach) of the sandfly, the amastigote elongates and develops into the promastigote form.
*The promastigote multiples by longitudinal binary fission and reaches enormous numbers. They may be seen as large rosettes with their flagella entangled.
*In the sandfly, they migrate from the midgut to the pharynx and hypostome, where they accumulate and block the passage.
*Such blocked sandflies have difficulty in sucking blood. When they bite a person and attempt to suck blood, plugs of adherent parasites may get dislodged from wound. It takes about 10 days for the promastigotes to reach adequate numbers after ingestion of the amastigotes, so as to block the buccal cavity and pharynx of the sandfly. This is, therefore, the duration of extrinsic incubation period.
This period is also synchronous with the gonadotropic cycle of the vector, so that amastigotes ingested during a single blood meal, are ready to be transmitted when the sandfly takes the next blood meal after its eggs have been laid.

نشاط (1/2/4)

Q1/Complete the table

Parasite	**causing disease**
1- Leishmania Donovani	
2- Leishmania tropica	

الوحدة الرابعة - المحاضرة الثالثة - الزمن: 90 دقيقة

أهداف المحاضرة الثالثة:

يتوقع في نهاية الجلسة أن يكون الطالب قادراً على:

1- Enumerate of Pathogenicity of *Leishmania*
2- Enumerate of Laboratory diagnostic of *Leishmania*
3- Enumerate of molecular diagnostic of *Leishmania*

موضوعات المحاضرة الثالثة:

1- Genus *Leishmania*
2- Pathogenicity
3- Laboratory Diagnosis
4- Molecular diagnosis

الأساليب والأنشطة والوسائل التعليمية

م	الأساليب والأنشطة التدريبية	الوسائل التدريبية
1	• محاضرة • مناقشة • سؤال وجواب • نشاط (1/3/4) نشاط	• جهاز حاسوب • جهاز عرض • سبورة • اوراق واقلام

خطة إجراءات تنفيذ المحاضرة الثالثة

الوحدة	المحاضرة	الإجراءات	الزمن بالدقيقة
الرابعة	الثالثة	القاء المحاضرة مستخدما جهاز العرض والسبورة	90 دقيقة
		طرح بعض الاسئلة خلال القاء المحاضرة	
		نشاط (1/3/4) تمرين تعداد	

المادة العلمية:

Pathogenicity *Leishmania donovani*

L. donovani causes visceral leishmaniasis or kala-azar.

*Kala-azar is a reticuloendotheliosis resulting from the invasion of reticuloendothelial system by L. donovani.

*The parasitized macrophages disseminate the infection to all parts of the body.

*In the spleen, liver, and bone marrow particularly, the amastigotes multiply enormously in the fixed macrophages to produce a 'blockade' of the reticuloendothelial system. This leads to a marked proliferation and destruction of reticuloendothelial tissue in these organs.

***Spleen:**

* The spleen is the most affected organ. It is grossly enlarged and the capsule is thickened due to perisplenitis.

* Spleen is soft and friable and cuts easily due to absence of fibrosis.

* The cut section is red or chocolate in color due to the dilated and engorged vascular spaces.

* The trabeculae are thin and atrophic.

* Microscopically, the reticulum cells are greatly increased in numbers and are loaded with LD bodies.

* Lymphocytic infiltration is scanty, but plasma cells are numerous.

***Liver:**

* The liver is enlarged.

*The Kupffer cells and vascular endothelial cells are heavily parasitized, but hepatocytes are not affected.

* Liver function is, therefore, not seriously affected, although prothrombin production is commonly decreased.

*The sinusoidal capillaries are dilated and engorged.

* Some degree of fatty degeneration is seen. The cut surface may show a 'nutmeg' appearance.

***Bone marrow:**

*The bone marrow is heavily infiltrated with parasitized macrophages, which may crowd the hematopoietic tissues.

Peripheral lymphnodes and lymphoid tissues of the nasopharynx and intestine are hypertrophic, although this is not seen in Indian cases.

*Severe anemia with hemoglobin levels of 5–10 g/dL may occur in Kala-azar, as a result of infiltration of the bone marrow as well as by the increased destruction of erythrocytes

due to hypersplenism. Autoantibodies to red cells may contribute to hemolysis. Leucopenia with marked neutropenia and thrombocytopenia are frequently seen. Antibodies against WBCs and platelets suggest an autoimmune basis for the pancytopenia observed in Kala-azar.

Clinical Features of Kala-Azar

*The onset is typically insidious. The clinical illness begins with fever, which may be continuous, remittent, or irregular.
*Splenomegaly starts early and is progressive and massive.
*Hepatomegaly and lymphadenopathy also occur but are not so prominent.
*Skin becomes dry, rough, and darkly pigmented (hence, the name Kala-azar).
*The hair becomes thin and brittle.
*Cachexia with marked anemia, emaciation, and loss of weight is seen.
*Epistaxis and bleeding from gums are common.
*Most untreated patients die in about 2 years, due to some inter current disease such as dysentery, diarrhea, and tuberculosis.

Post Kala-azar Dermal Leishmaniasis

About 3–10% cases of patients of visceral leishmaniasis in endemic areas develop PKDL, about a year or 2 after recovery from the systemic illness.
*PKDL is seen mainly in India and East Africa and not seen elsewhere. The Indian and African diseases differ in several aspects; important features of PKDL in these two regions.
*PKDL is a non-ulcerative lesion of skin. The lesions are of 3 types.
Depigmented macules: These commonly appear on the trunk and extremities and resemble tuberculoid leprosy.

Erythematous patches: These are distributed on the face in 'butterfly distribution'. Nodular lesion: Both of the above-mentioned lesions may develop into painless yellowish pink no ulcerating granulomatous nodules.
*The parasite can be demonstrated in the lesions.

Laboratory Diagnosis

Laboratory diagnosis of Kalaazar depends upon direct and indirect evidences.
Microscopy

*Demonstration of amastigotes in smears of tissue aspirates is the gold standard for diagnosis of visceral leishmaniasis. For microscopic demonstration of the parasite, the materials collected are:

* Peripheral blood
* Bone marrow
* Splenic aspirate
* Enlarged lymph node.

*The smears are stained by Leishman, Giemsa, or Wright's stains and examined under oil immersion objective.

*Amastigote parasite can be seen within the macrophages, often in large numbers. A few extracellular forms can also be seen.

Peripheral blood smear:

* Peripheral blood contains the amastigotes present inside circulating monocytes and less often in neutrophils, but the numbers are so scanty that a direct blood smear may not show them.
* Chances of detecting them are somewhat improved by examination of a thick blood film.
*It is best to examine Buffy coat smear, although ven these are not often found positive.
* Buffy coat smears show a diurnal periodicity, more smears being positive when collected during the day than at night.

Bone marrow aspirate:

* Bone marrow aspirate is the most common diagnostic specimen collected.
*Generally, the sternal marrow is aspirated by uncturing the sternum at the level of the 2nd or 3rd intercostal space, using a sternal puncture needle.
* This consists of a short stout needle with a stylet. It has a movable guard, which is fixed at 1–2 cm from the tip, depending on the thickness of the chest wall over the sternum.
* After disinfecting and anesthetizing the skin, the needle is introduced into the sternal marrow and about 0.5 mL of marrow fluid is aspirated using a syringe. The puncture wound is sealed with celloidin or tincture benzoin. Bone marrow samples can also be obtained by puncturing the iliac crest.

Splenic aspirates:

* Splenic aspirates are richer in parasites and therefore, are more valuable for diagnosis.
*But the procedure can sometimes cause dangerous bleeding and therefore, should be done carefully and only when a marrow examination is inconclusive.

Lymph node aspirates:

*Lymph node apsirates are not useful in the diagnosis of Indian Kala-azar, although it is employed in visceral leishmaniasis in some other countries.

Comparison of aspiration biopsies

Although splenic aspiration is the most sensitive method (98% positive), one marrow puncture (50–85%, positive) is a safer procedure when compared to spleen puncture, as there is risk of hemorrhage in splenic puncture particularly in patients with advanced stage of disease with soft enlarged spleen. Splenic aspiration is contraindicated in patients with prolonged prothrombin time or if platelet count is less than 40,000/mm3. Liver biopsy is also not a safe procedure and carries a risk of hemorrhage. Lymph node aspiration is positive in 65% of cases of African Kala-azar, but not useful in cases of Indian Kala-azar.

Culture

Different tissue materials or blood are cultured on NNN medium. This is a rabbit blood agar slope consisting of 2 parts of salt agar and 1 part of defibrinated rabbit blood. The material is inoculated into the water of condensation and culture is incubated at 22°–24°C for 1–4 weeks. At the end of each week, a drop of culture fl uid is examined for promastigotes under high power objective or phase contrast illumination.

Other biphasic medium, like Schneider's drosophila tissue culture medium with added fetal calf serum can also be used.

Animal inoculation

Animal inoculation is not used for routine diagnosis.

When necessary, Chinese golden hamster is the animal employed.

*The material is inoculated intraperitoneally or intradermally into the skin of nose and feet.

*The inoculated animals are kept at 23°–26°C.

*In positive cases, the amastigote can be demonstrated in smears taken from ulcers or nodules developing at the sites of inoculation or from the spleen.

*Animal inoculation is a very sensitive method, but takes several weeks to become positive.

Indirect Evidences

Serodiagnosis

*Detection of antigen: The concentration of antigen in the serum or other body fluids is very low. ELISA and PCR have been developed for detection of leishmanial antigen.

Detection of antibodies: CFT was the first serological test used to detect serum antibodies in visceral leishmaniasis. The antigen originally used, was prepared from human tubercle bacillus by Witebsky, Kliengenstein, and Kuhn (hence, called WKK antigen). CFT using WKK antigen becomes positive early in the disease, within weeks of infection. Positive reaction also occurs in other conditions, including tuberculosis, leprosy, and tropical eosinophilia.

*Specific leishmanial antigens prepared from cultures have been used in a number of tests to demonstrate specific antibodies. These tests include:

*Indirect immunofluroscent antibody test (IFAT)

*Counter immunoelectrophoresis (CIEP)

LD body in spleen smear of experimentally infected animal (Giemsa stain)

A specific rapid immunchromatographic dipstick (ICT) method for antibody has been developed using a recombinant leishmanial antigen rk 39 consisting of 39 amino acids conserved in kinesin region of L. infantum. The sensitivity of the test is 98% and specificity is 90%.

Molecular diagnosis

A number of molecular diagnosis methods have been developed, which help in species identification of Leishmania. The methods include Western blot and PCR. The use of PCR is confined to specialized laboratories and is yet to be used for routine diagnosis of visceral leishmaniasis in endemic areas.

>**Leishmanin skin test (Montenegro test):**

* It is delayed hypersensitivity test.
* This was first discovered by Montenegro in South America and hence, named after him.
* 0.1 mL of killed promastigote suspension (106 washed promastigotes/mL) is injected intradermally on the dorsoventral aspect of forearm.
* Positive result is indicated by an induration and erythema of 5 mm or more after 48–72 hours.
* Positive result indicates prior exposure to leishmanial parasite.
* In active Kala-azar, this test is negative and becomes positive usually 6–8 weeks after cure from the disease.

>Blood picture

*Complete blood count shows normocytic normochromic anemia and thrombocytopenia.

*Leucocyte count reveals leucopenia accompanied by a relative increase of lymphocytes and monocytes. Eosinophil granulocytes are absent. During the course of disease, there is a progressive diminution of leucocyte count falling to 1,000/mm3 of blood or even below that.

*The ratio of leucocyte to erythrocyte is greatly altered and may be about 1:200 to 1:100 (normal 1:750).
*Serum shows hypergammaglobulinemia and a reversal of the albumin: globulin ratio.
*Liver function tests show mild elevations.
Diagnosis of PKDL
*The nodular lesions are biopsied and amastigote forms are demonstrated in stained sections.
*The biopsy material can be cultured or animal inoculation can be done.
*Immunodiagnosis has no role in the diagnosis of PKDL.

نشاط (1/3/4)

Q\Enumerate of molecular diagnostic methods with a simple explanation of each one

الوحدة الخامسة - المحاضرة الأولى - الزمن: 90 دقيقة

أهداف المحاضرة الاولى:

يتوقع في نهاية الجلسة أن يكون الطالب قادراً على:

1- Explain the morphology of *Cryptosporidium parvum*
2- Draw the life cycle of *Cryptosporidium parvum*

موضوعات المحاضرة الاولى:

- Intestinal coccidian e.g., *Cryptosporidium parvum*
- Morpholog
- Habitat, mode of infection, infective stage
- Life cycle
- Laboratory diagnosis

الأساليب والأنشطة والوسائل التعليمية

م	الأساليب والأنشطة التدريبية	الوسائل التدريبية
1	• محاضرة • مناقشة • سؤال وجواب • نشاط (1/1/5) نشاط	• جهاز حاسوب • جهاز عرض • سبورة • اوراق واقلام

خطة إجراءات تنفيذ المحاضرة الاولى

الوحدة	المحاضرة	الإجراءات	الزمن بالدقيقة
الأولى	الأولى	القاء المحاضرة مستخدما جهاز العرض والسبورة	90 دقيقة
		طرح بعض الاسئلة خلال القاء المحاضرة	
		نشاط (1/1/5) تمرين صح وخطأ	

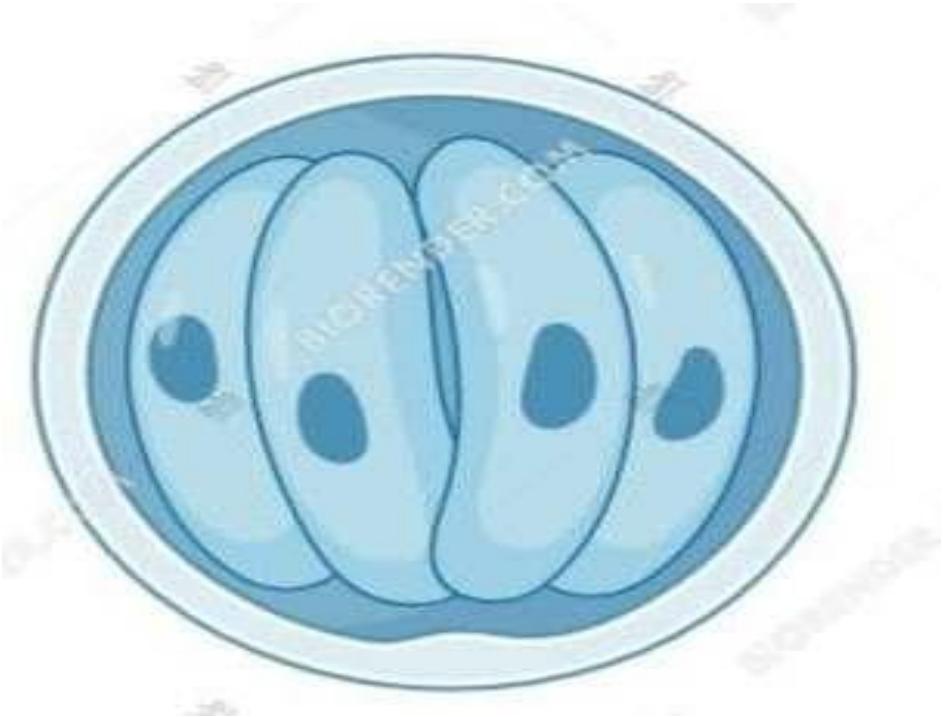
المادة العلمية:

Cryptosporidium parvum

Morphology

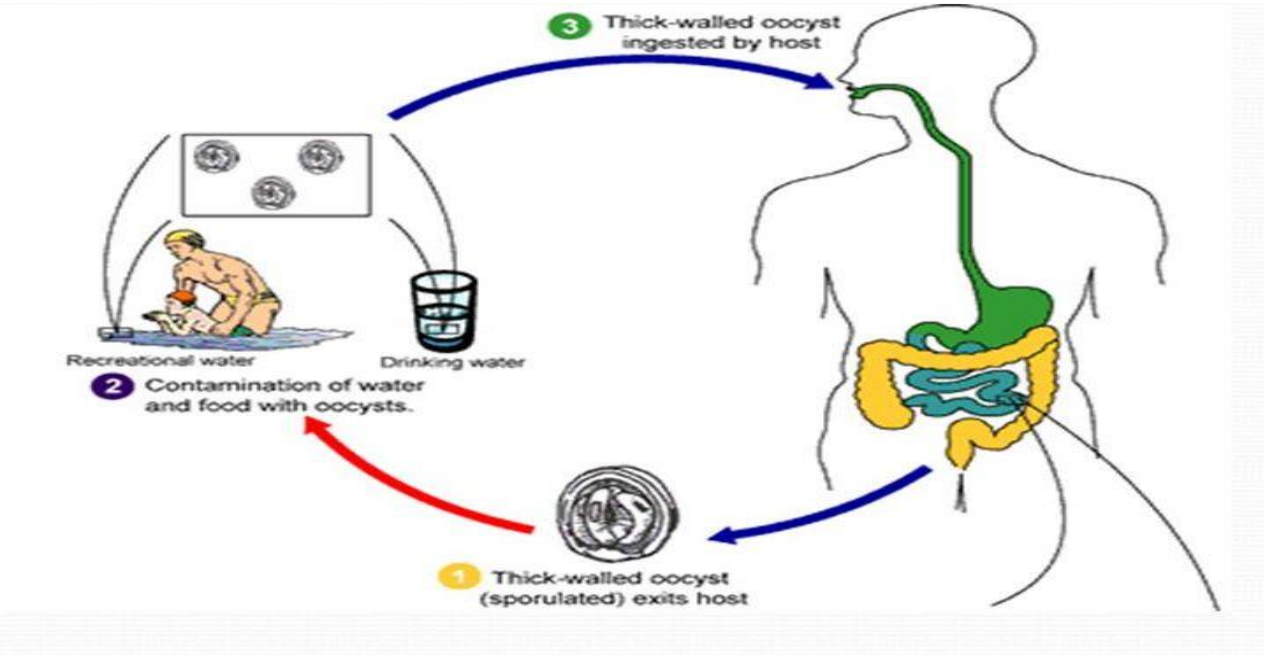
Oocyst

Rounded 4-6um in size, contains 4 sporozoites Two types of oocysts: Thick-walled oocyst (Has double wall membrane) commonly excreted with feces and thin-walled oocyst (20%) involved in internal autoinfection.



Life cycle

- The parasite completes its life cycle, sexual and asexual phases in a single host
- Definitive host: human.
- Reservoirs: Man, cattle, cat, and dog.
- Mode of transmission: Man acquires infection by: Ingestion of food and water contaminated with feces containing oocysts Autoinfection.
- Infective stage: oocysts
- Habitat: in the small intestine. It may also be found in stomach, appendix, colon and rectum
- Disease: cryptosporidiosis



Pathogenesis

Cryptosporidium causes diarrhea worldwide, for large outbreaks of diarrhea caused by Cryptosporidium are attributed to inadequate purification of drinking water. The disease in immunocompromised patients presents primarily as a watery, non-bloody diarrhea causing large fluid loss. Symptoms persist for long periods in immunocompromised patients, whereas self-limited in immunocompetent individuals. Although immunocompromised patients usually do not die of cryptosporidiosis, the fluid loss and malnutrition are severely debilitating

Diagnosis

by finding oocysts in fecal smears.

نشاط (1/1/5)

Q/ Write True or false

1- **The Definitive host of *Cryptosporidium parvum* is pigs**

2- **The Life cycle of *Cryptosporidium parvum* in two host**

الوحدة السادسة - المحاضرة الأولى - الزمن: 90 دقيقة

أهداف المحاضرة الاولى:

يتوقع في نهاية الجلسة أن يكون الطالب قادراً على:

1- Enumerate the morphology of *toxoplasma gondii*
2- Knowing How a person gets infected with a parasite *toxoplasma gondii*
3- Enumerate the diagnosis of *toxoplasma gondii*
4- What are the hosts of *toxoplasma gondii*

موضوعات المحاضرة الاولى:

* Extra-intestinal coccidian e.g., *Toxoplasma gondii*
*morphology
*life cycle
*Pathogenesis
*Diagnosis

الأساليب والأنشطة والوسائل التعليمية

م	الأساليب والأنشطة التدريبية	الوسائل التدريبية
1	• محاضرة • مناقشة • سؤال وجواب • نشاط (1/1/6) نشاط	• جهاز حاسوب • جهاز عرض • سبورة • اوراق واقلام

خطة إجراءات تنفيذ المحاضرة الاولى

الوحدة	المحاضرة	الإجراءات	الزمن بالدقيقة
السادسة	الأولى	القاء المحاضرة مستخدما جهاز العرض والسبورة	90 دقيقة
		طرح بعض الاسئلة خلال القاء المحاضرة	
		نشاط (1/1/6) تمرين تعداد	

المادة العلمية:

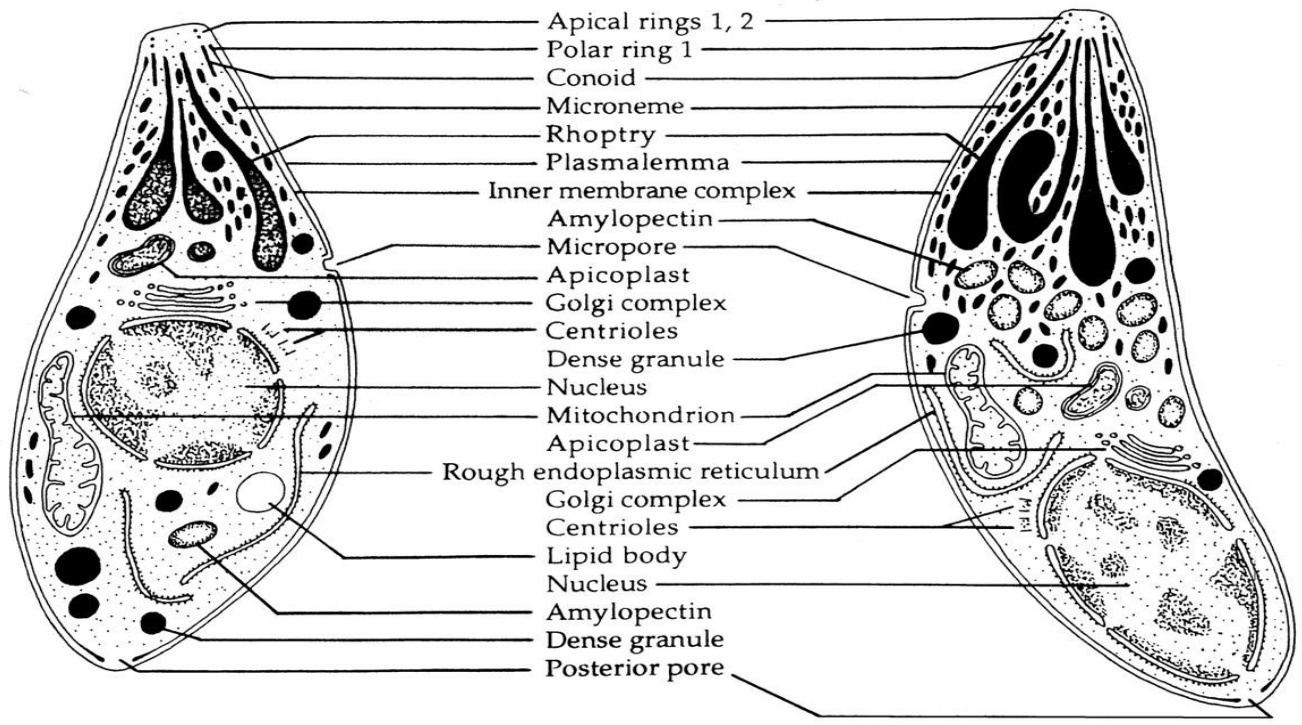
Toxoplasma gondii

Morphology

toxoplasma gondii occurs in 3 forms:

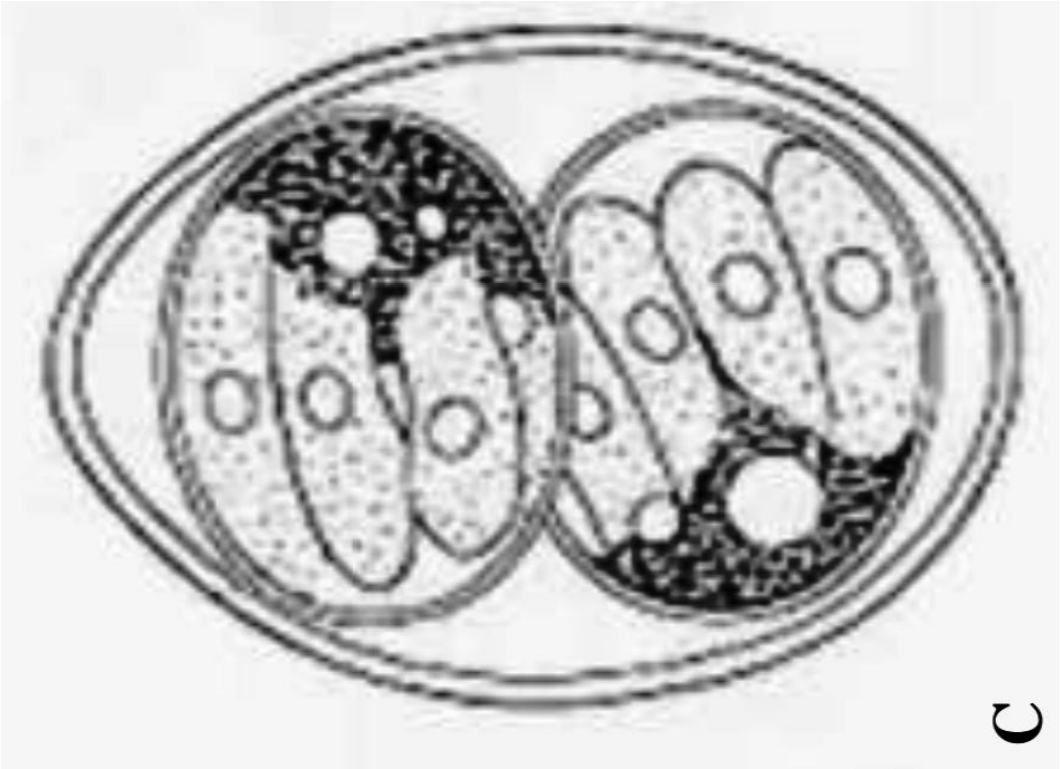
A. Bradyzoite (tissue syst)
- ❖ Crescent shaped
- ❖ Center nucleus
- ❖ Slow growing stage
- ❖ Chronic phase of infection

B. Tachyzoite (Trophozoite)
- ❖ Crescent shaped
- ❖ Nucleus is near the rounded end
- ❖ Rapidly growing stage
- ❖ Acute phase of infection



C. Oocyst

- ❖ Oval shape
- ❖ Contains 2 sporocysts each contains 4 sporozoites



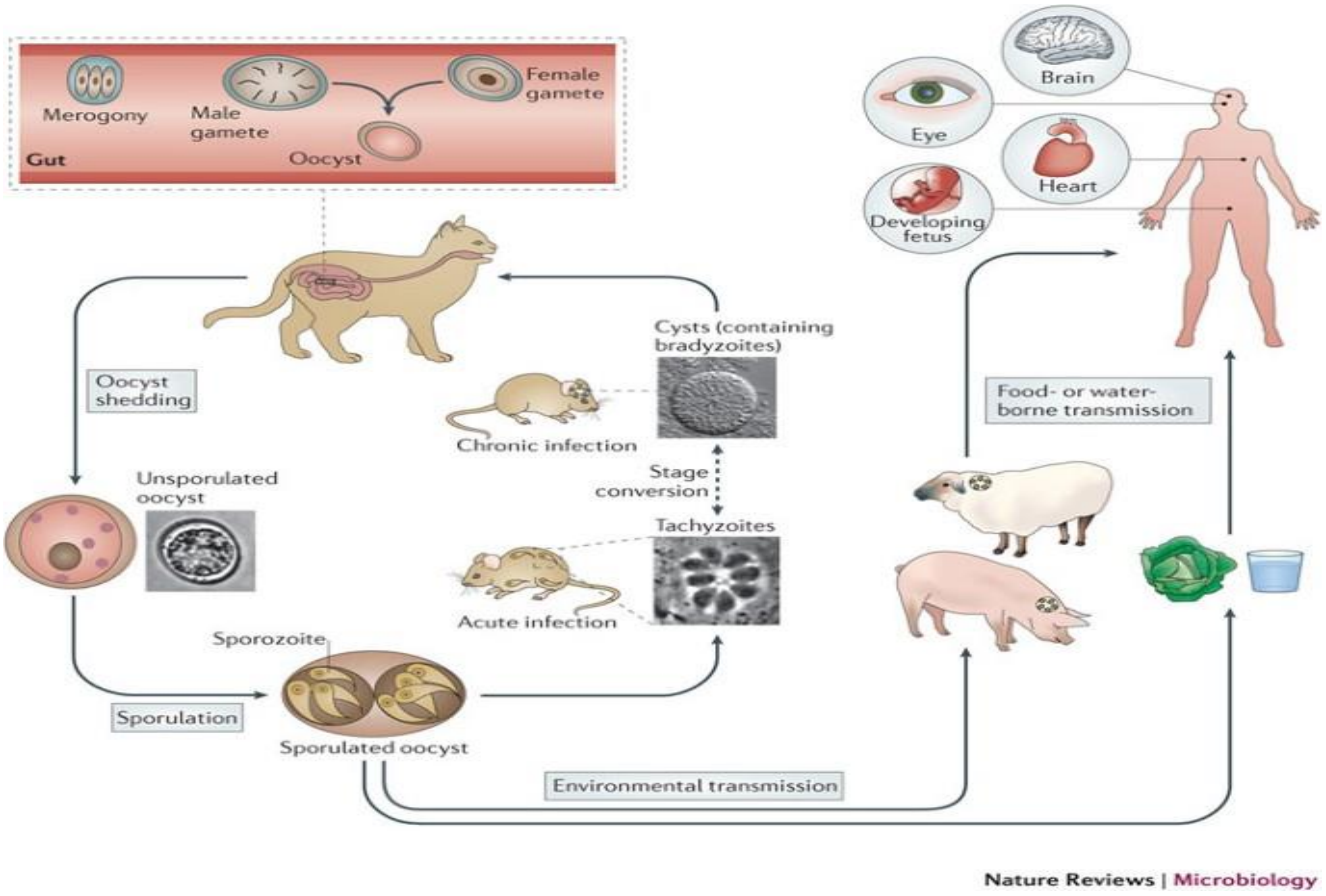
Life cycle

toxoplasma gondii completes its life cycle in 2 hosts:

Definitive host: Cats and other felines, in which sexual cycle takes place.

Intermediate hosts: Man, and other mammals, in which only the asexual cycle takes place

Causes: toxoplasmosis



Humans acquire infection through:

- Eating uncooked or undercooked infected meat, particularly lamb and pork containing tissue cysts
- Ingestion of mature oocysts through food, water, or fingers contaminated with cat feces directly or indirectly
- Intrauterine infection from mother to fetus (congenital toxoplasmosis)
- Blood transfusion or transplantation from infected donors.

Pathogenesis

- The disease picture : fever – headache – myalgia lymphadenitis – extreme fatigue.
- Abortion in the first trimester.
- Congenital defects : hydrocephalus – microcephalus – intracerebral calcification.
- In a very few cases retinochoroiditis occurs which may progress to produce blindness.

Diagnosis

- Direct microscopic examination.
- Latex agglutination test (LAT).
- Enzyme linked immune sorbent assay (ELISA).
- Polymerase chain reaction (PCR).

نشاط (1/1/6)

Q\ How does a person get infected with a parasite toxoplasma *gondii?*

الوحدة السادسة - المحاضرة الثانية - الزمن: 90 دقيقة

أهداف المحاضرة الثانية:

يتوقع في نهاية الجلسة أن يكون الطالب قادراً على:

1- Enumerate of plasmodium types
2- Mention of the disease of each type OF plasmodium
3- Explain the morphology of Plasmodium
4- Raw life cycle of malaria

موضوعات المحاضرة الثانية:

- Genus Plasmodium
- Terms used in malaria
- Classification
- Causative Agents of Human Malaria
- Life cycle

الأساليب والأنشطة والوسائل التعليمية

م	الأساليب والأنشطة التدريبية	الوسائل التدريبية
1	• محاضرة • مناقشة • سؤال وجواب • نشاط (1/2/6) نشاط	• جهاز حاسوب • جهاز عرض • سبورة • اوراق واقلام

خطة إجراءات تنفيذ المحاضرة الثانية

الوحدة	المحاضرة	الإجراءات	الزمن بالدقيقة
السادسة	الثانية	القاء المحاضرة مستخدما جهاز العرض والسبورة	90 دقيقة
		طرح بعض الاسئلة خلال القاء المحاضرة	
		نشاط (1/2/6) تمرين تعداد	

المادة العلمية:

Malaria

*Protozoan parasites characterized by the production of spore-like oocysts containing sporozoites were known as sporozoa.
*They live intracellularly, at least during part of their life cycle.
*At some stage in their life cycle, they possess a structure called the apical complex, by means of which they attach to and penetrate host cells.
*These protozoa are therefore grouped under the Phylum Apicomplexa.
*The medically important parasites in this group are the malaria parasites, Coccidia, and Babesia.
*The Phylum Apicomplexa includes 2 classes viz. haematozoa and coccidia and 3 orders: eimeriida, haemosporida, and piroplasmida.

Note:

Many minutes intracellular protozoa formerly grouped as sporozoa have been reclassifi ed because of some structural differences. These are now called microspora. They infect a large spectrum of hosts including vertebrates and invertebrates.
Infection is mostly asymptomatic, but clinical illness is often seen in the immunodeficient.

Classification

Malaria parasite belongs to
Phylum: Apicomplexa
Class: Sporozoa
Order: Haemosporida
Genus: Plasmodium.
*The genus Plamodium is divided into 2 sub genera; P. vivax, P. malariae and P. ovale belong to the subgenus Plasmodium while P. falciparum is allocated to subgenus Laverania because it differs in a number of aspects from the other 3 species.
*P. vivax, P. malariae, and P. ovale are closely related to other primate malaria parasites. P. falciparum on the other hand, is more related to bird malaria parasites and appears to be a recent parasite of humans, in evolutionary terms. Perhaps for this reason, falciparum infection causes the severest form of malaria and is responsible for nearly all fatal cases.
*P. knowelsi, a parasite of long–tailed Macaque monkeys may also affect man.

Causative Agents of Human Malaria

**Plamodium vivax:* Benign Tertian Malaria
**Plasmodium falciparum*: Malignant Tertian Malaria
**Plasmodium malariae*: Benign Quatrain Malaria
**Plasmodium ovale:* Benign Tertian Malaria.

Malaria Parasite

*Malaria affects mainly poor, underserved, and marginalized population in rural remote areas as well as in urban areas. An epidemic can develop when there are changes in environmental, economic, and social conditions such as migrations and heavy rains following draughts.

*The relative prevalence of the 4 species of malaria parasites varies in different geographical regions.

* P. vivax is the most widely distributed, being most common in Asia, North Africa, and Central and South America.

*P. falciparum, the predominant species in Africa,Papua New Guinea, and Haiti, is rapidly spreading in South-east Asia and India.

* P. malariae is present in most places but is rare, except in Africa.

* P. ovale is virtually confined to West Africa where it ranks second after P. falciparum.

Vectors

Human malaria is transmitted by over 60 species of female Anopheles mosquito.

*The male mosquito feeds exclusively on fruits and juices, but the female needs at least 2 blood meals, before the first batch of eggs can be laid.

*Out of 45 species of Anopheles mosquito in India, only few are regarded as the vectors of malaria. These are An *culicifacies, An fluvatilis, An stephansi, An minimus, An phillippenensis, An sundaicus, etc.*

Life Cycle

Malaria parasite passes its life cycle in 2 hosts.

Definitive host: Female Anopheles mosquito.

Intermediate host: Man.

* The life cycle of malaria parasite comprises of 2 stages: an asexual phase occurring in humans, who act as the intermediate host and a sexual phase occurring in mosquito, which serves as a definitive host for the parasite.

Asexual phase:

*In this stage, the malaria parasite multiplies by division or splitting a process designated to as Schizogony (from schizo: to split, and gone: generation).

Because this asexual phase occurs in man, it is also called the vertebrate, intrinsic, or endogenous phase.

In humans, schizogony occurs in 2 locations: in the red blood cell (erythrocytic schizogony) and in the liver cells (exoerythrocytic schizogony or the tissue phase). Because schizogony in the liver is an essential step before the parasites can invade

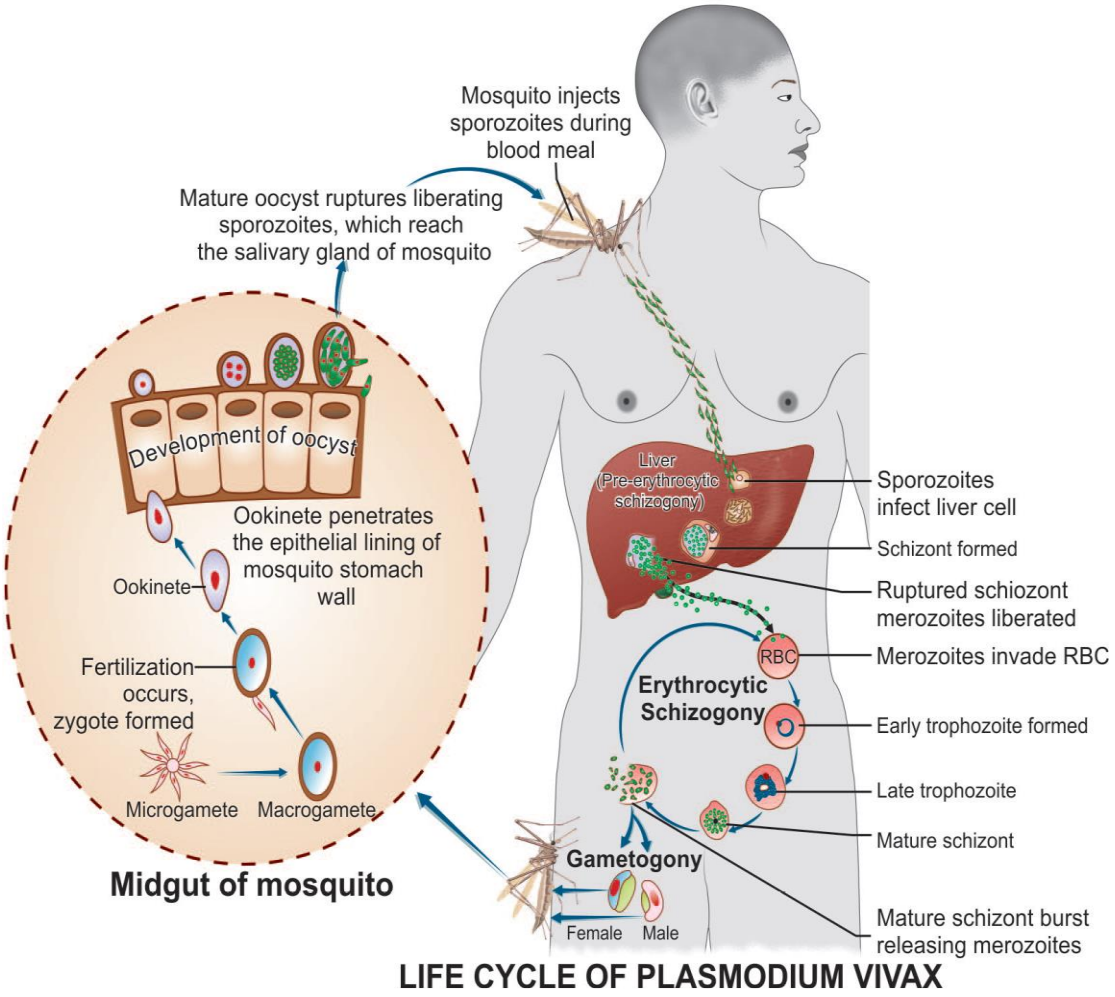
erythrocytes, it is called pre-erythrocytic schizogony. The products of schizogony, whether erythrocytic or exoerythrocytic, are called merozoites (meros: a part, zoon: animal).

Sexual phase:

*The sexual phase takes place in the female Anopheles mosquito, even though the sexual forms of the parasite (gametocytes) originate in human red blood cells.

*Maturation and fertilization take place in the mosquito, giving rise to a large number of sporozoites (from sporos: seed). Hence this phase of sexual multiplication is called sporogony. It is also called the invertebrate, extrinsic, or exogenous phase.

*There is, thus an alternation of generations in the life cycle of malaria parasites: asexual and sexual generations, alternatively.



Human Cycle (Schizogony)

Human infection comes through the bite of the infective female Anopheles mosquito.

*The sporozoites, which are infective forms of the parasite are present in the salivary gland of the mosquito.

*They are injected into blood capillaries when the mosquito feeds on blood after piercing the skin.

*Usually, 10–15 sporozoites are injected at a time, but occasionally, many hundreds may be introduced.

*The sporozoites pass into the blood stream, where many are destroyed by the phagocytes, but some reach the liver and enter the parenchymal cells (hepatocytes).

Pre-erythrocytic (Tissue) Stage or Exoerythrocytic Stage

Within an hour of being injected into the body by the mosquito, the sporozoites reach the liver and enter the hepatocytes to initiate the stage of pre-erythrocytic
schizogony or merogony.

*The sporozoites, which are elongated spindle-shaped bodies, become rounded inside the liver cells.

*They enlarge in size and undergo repeated nuclear division to form several daughter nuclei; each of which is surrounded by cytoplasm.

*This stage of the parasite is called the pre-erythrocytic or

exoerythrocytic schizont or meront.

*The hepatocyte is distended by the enlarging schizont and the liver cell nucleus is pushed to the periphery.

*Mature liver stage schizonts are spherical (45–60 μm), multinucleate, and contain 2000–50,000 uninucleate merozoites.

*Unlike erythrocytic schizogony, there is no pigment in liver schizonts. These normally rupture in 6–15 days and release thousands of merozoites into the blood stream.

*The merozoites infect the erythrocytes by a process of invagination.

*The interval between the entry of the sporozoites into the body and the first appearance of the parasites in blood is called the prepatent period.

*The duration of the pre-erythrocytic phase in the liver, the size of the mature schizont, and the number of merozoites produced vary with the species of the parasite.

*Latent stage: In P. vivax and P. ovale, two kinds of sporozoites are seen, some of which multiply inside hepatic cells to form schizonts and others persist and remain dormant (resting phase). The resting forms are called hypnozoites (hypnos: sleep). From time to

time, some are activated to become schizonts and release merozoites, which go on infecting RBCs producing clinical relapse.

* In P. falciparum and P. malariae, initial tissue phase disappears completely, and no hypnozoites are found. However, small number of erythrocytic parasites persist in the blood stream and in due course of time, they multiply to reach significant numbers resulting in clinical disease (short-term relapse or recrudescence).

Erythrocytic Stage

The merozoites released by pre-erythrocytic schizonts invade the red blood cells.

* The receptor for merozoites is glycophorin, which is a major glycoprotein on the red cells. The differences in the glycophorins of red cells of different species may account for the species specificity of malaria parasites.

* Merozoites are pear-shaped bodies, about 1.5 μm in length, possessing an apical complex (rhoptery). They attach to the erythrocytes by their apex and then the merozoites lie within an intra-erythrocytic parasitophorus vacuole formed by red cell membrane by a process of invagination.

*In the erythrocyte, the merozoite loses its internal organelles and appears as a rounded body having a vacuole in the center with the cytoplasm pushed to the periphery and the nucleus at one pole. These young parasites are, therefore called the ring forms or young trophozoites. The parasite feeds on the hemoglobin of the erythrocyte. It does not metabolize hemoglobin completely and therefore, leaves behind a hematin-globin pigment called the malaria pigment or haemozoin pigment, as residue.

The appearance of malaria pigments varies in different species as follows:

••P vivax: Numerous fine golden-brown dust-like particles
••P. falciparum: Few 1–3 solid blocks of black pigment
••P. malariae: Numerous coarse dark brown particles
••P ovale: Numerous blackish brown particles.

*The malaria pigment released when the parasitized cells rupture is taken up by reticuloendothelial cells. Such pigment-laden cells in the internal organs provide histological evidence of previous malaria infection.

*As the ring form develops, it enlarges in size becoming irregular in shape and shows amoeboid motility. This is called the amoeboid form or late trophozoite form.

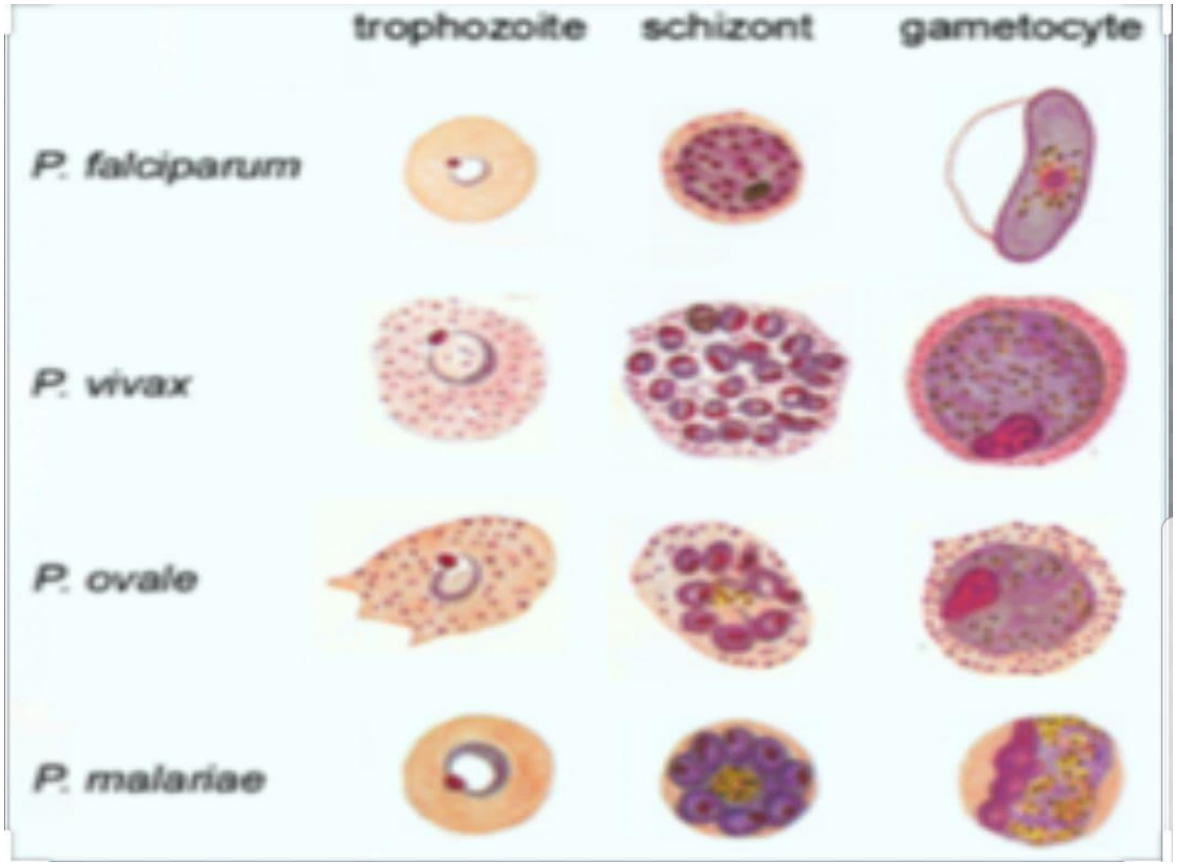
*When the amoeboid form reaches a certain stage of development, its nucleus starts dividing by mitosis followed by a division of cytoplasm to become mature schizonts or meronts.

*A mature schizont contains 8–32 merozoites and hemozoin. The mature schizont bursts releasing the merozoites into the circulation.

*The merozoites invade fresh erythrocytes within which they go through the same process of development.
This cycle of erythrocytic Schizogony or merogony is repeated sequentially, leading to progressive increase in the parasitemia, till it is arrested by the development of host immune response.
*The rupture of the mature schizont releases large quantities of pyrogens. This is responsible for the febrile paroxysms characterizing malaria.
*The interval between the entry of sporozoites into the host and the earliest manifestation of clinical illness is the incubation period, which is the time taken from entry of the sporozoites to the first appearance of malaria parasite in peripheral blood.
*In P. falciparum, erythrocytic schizogony always takes place inside the capillaries and vascular beds of internal organs. Therefore, in P. falciparum infections, schizonts, and merozoites are usually not seen in the peripheral blood.

Gametogony

After a few erythrocytic cycles, some of the merozoites that infect RBC's do not proceed to become trophozoites or schizonts but instead, develop into sexually differentiated forms, the gametocytes.
*They grow in size till they almost fill the RBC, but the nucleus remains undivided.
*Development of gametocytes generally takes place within the internal organs and only the mature forms appear in circulation.
*The mature gametocytes are round in shape, except in P. falciparum, in which they are crescent-shaped.
*In all species, the female gametocyte is larger (macrogametocyte) and has cytoplasm staining dark blue with a compact nucleus staining deep red. In the smaller male gametocyte (microgametocyte), the cytoplasm stains pale blue or pink and the nucleus is larger, pail stained and diffuses. Pigment granules are prominent.
*Female gametocytes are generally more numerous than the male.
*Gametocyte appear in circulation 4–5 days after the first appearance of asexual form in case of P. vivax and 10–12 days in P. falciparum.
*A person with gametocytes in blood is a carrier or reservoir.
*The gametocytes do not cause any clinical illness in the host, but are essential for transmission of the infection.
*A gametocyte concentration of 12 or more per cumm of blood in the human host is necessary for mosquitoes to become infected.



نشاط (1/2/6)

Q\Enumerate of plasmodium types with mention of the disease of each type

الوحدة السابعة - المحاضرة الأولى - الزمن: 90 دقيقة

أهداف المحاضرة الاولى:

يتوقع في نهاية الجلسة أن يكون الطالب قادراً على:

1-Enumerate of the classification of medical Helminthology

2- Enumerate of the General characters of Helminthes

3- Enumerate of the General characters of trematoda

موضوعات المحاضرة الاولى:

1- Introduction to Helminths
2- Classification of helminthes into
3- Phylum Platyhelminths which includes
4- Class trematoda

الأساليب والأنشطة والوسائل التعليمية

م	الأساليب والأنشطة التدريبية	الوسائل التدريبية
1	• محاضرة • مناقشة • سؤال وجواب • نشاط (1/1/7) نشاط	• جهاز حاسوب • جهاز عرض • سبورة • اوراق واقلام

خطة إجراءات تنفيذ المحاضرة الاولى

الوحدة	المحاضرة	الإجراءات	الزمن بالدقيقة
السابعة	الأولى	القاء المحاضرة مستخدما جهاز العرض والسبورة	90 دقيقة
		طرح بعض الاسئلة خلال القاء المحاضرة	
		نشاط (1/1/7) تمرين تعداد مع مثال	

المادة العلمية:

Helminthes

Introducion

Helminthes are trophoblastic metazoa (multi-cellular organisms). Helminthes are among the common parasitic causes of human suffering. They are the cause of high morbidity and mortality of people worldwide.

They cause different diseases in humans, but few helminthic infections cause life-threatening diseases. They cause anemia and malnutrition. In children they cause a reduction in academic performance. Helminthes also cause economic loss as a result of infections of domestic animals. The sources of the parasites are different. Exposure of humans to the parasites

may occur in one of the following ways:

1. Contaminated soil (Geo-helminthes), water (cercariae of blood flukes) and food (Taenia in raw meat).
2. Blood sucking insects or arthropods (as in filarial worms).
3. Domestic or wild animals harboring the parasite (as in echinococcus in dogs).
4. Person to person (as in Enterobius vermicularis, Hymenolopis nana).
5. Oneself (auto-infection) as in Enterobius vermicularis.

They enter the body through different routes including: mouth, skin and the respiratory tract by means of inhalation of airborne eggs.

Classification of Medical Helminthology

These are:

1. Trematodes (Flukes)
2. Nematodes (Round worms)
3. Cestodes (Tape worms) The Trematodes and Cestodes are groups of flat worms.

Helminthology (helminths):

1- Platyhelminths (flat worms)
- Class: Trematoda (Flat Worms or Flukes).
- Class: Cestoda (Tape worms).

2- Nemathelminths (round worms)
- Class: Nematoda (Round worms).

General characters of parasites

1. Trematodes: Un-segmented, leaf-shaped, and hermaphrodite (except schistosomes), e.g.

Fasciola.

2. Cestodes: Long, segmented, tape-like and hermaphrodite, e.g., Taenia saginata.

3. Nematodes: Elongated, cylindrical with pointed ends and unisexual, e.g., As Specific helminths can be identified through microscopic examination of their eggs (ova) found in faecal samples. The number of eggs is measured in units of eggs per gram (? egg/gm of feces).

However, it does not quantify mixed infections, and in practice, is inaccurate for quantifying the eggs of schistosomes and soil-transmitted helminths.
Sophisticated tests such as

- serological assays,
- antigen tests,
- molecular diagnosis

They are also available; however, they are time-consuming, expensive and not always reliable.

Platyhelminthes (flat worms)
General Character's
Class: Trematoda (Flukes)
General characters:
1) Adults are leaf like, pear shaped or elongated worms, flattened dorsoventrally.
2) Bilaterally symmetrical except schistosomes.
3) Size: varies, some are large fleshy (Fasciola) others are just visible by naked eye (Heterophyes).
4) Covered with protective cuticle that may be smooth, spiny or tuberculated.
5) No body cavity, all organs are embedded in loose connective tissue cells.
6) Suckers: for attachment, usually 2 in
number, in some there are 3(Heterophyes heterophyes).
7) Digestive system:
- Starts by the mouth opening, found at the bottom of the oral sucker.
- The mouth leads to a pharynx, then a short oesophagus which bifurcates into two long intestinal caeca.
- Caeca end blindly with no anus.

Digestive system

8) Excretory system
- Starts by a definite number of excretory cells called (flame cells).
- Waste products pass from the cell excretory tubules excretory duct excretory bladder, excretory pore at the posterior end of the fluke.

Flame cell

9) Nervous system: consists of a ring of nerve ganglion around the pharynx, from which nerve fibers arise.
10) Respiration and nutrition:

-Adult flukes are anaerobic.
-They feed on biliary secretion, intestinal contents, tissue juices or blood according to their habitat.
11) Genital (reproductive system):
-Nearly all trematodes are hermaphroditic with exception (schistosomes).
• The male reproductive organs consist of two or more testes.
• The female genital organs consist of a single ovary situated in front of the two testes.

Trematode parasites (flukes) include:
• Hepatic or liver flukes:
- Fasciola gigantica - Fasciola hepatica
- Opisthorchis viverrini
• Intestinal flukes:
- Heterophyes heterophyes
• Lung flukes:
-Paragonimus westermani
• Blood flukes:
-Schistosoma haematobium, mansoni, japonicumand intercalatum.

نشاط (1/1/7)

Q\ Enumerate of the classification of medical Helminthology with an example for each class

الوحدة السابعة - المحاضرة الثانية - الزمن: 90 دقيقة

أهداف المحاضرة الثانية:

يتوقع في نهاية الجلسة أن يكون الطالب قادراً على:

1- Enumerate the General Tapeworm Morphology
2- Enumerate the General characteristics of Nematodes
3- How is infection transmitted by worms?
4- Distinguish between types of worms by shape

موضوعات المحاضرة الثانية:

- Class: Cestoda (tapeworm)
- General cestoda Morphology-
- Class: Nematodes (round worms)
- -General characteristics of Nematodes

الأساليب والأنشطة والوسائل التعليمية

م	الأساليب والأنشطة التدريبية	الوسائل التدريبية
1	• محاضرة • مناقشة • سؤال وجواب • نشاط (1/2/7) نشاط	• جهاز حاسوب • جهاز عرض • سبورة • اوراق واقلام

خطة إجراءات تنفيذ المحاضرة الثانيه

الوحدة	المحاضرة	الإجراءات	الزمن بالدقيقة
السابعة	الثانية	القاء المحاضرة مستخدما جهاز العرض والسبورة	90 دقيقة
		طرح بعض الاسئلة خلال القاء المحاضرة	
		نشاط (1/2/7) تشخيص ديدان	

المادة العلمية:

Cestodes (tapeworms)

Taenia solium
Taenia saginata
Diphylloborthrium latum

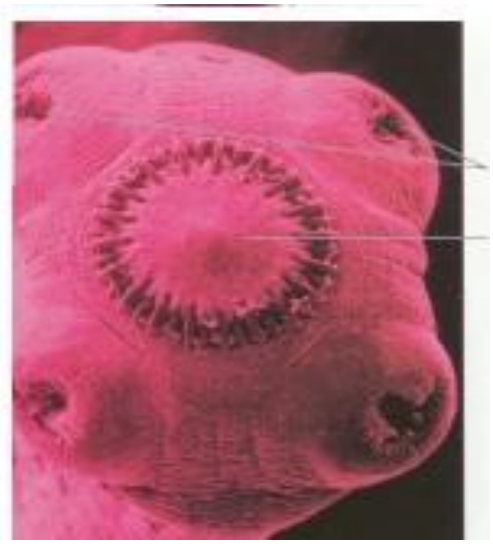
- Cestodes are flat and ribbonlike with heads armed with organs for attachment (scolex)
- All are hermaphroditic with male and female reproductive organs in each mature protoglottid (individual segments togather make strobili).
- No digestive system, food is absorbed from host intestine
- Eggs are nonoperculated (except D. latum)
- Infections with larval stages are always in tissues, or by ingestion of eggs passed in feces (cysticercus, hydatid cyst).
    - Tapeworms are entirely endoparasitic .
    - Adults inhabit the of vertebrate definitive hosts.
    - life cycles involve larval development in 1 or 2 intermediate hosts.

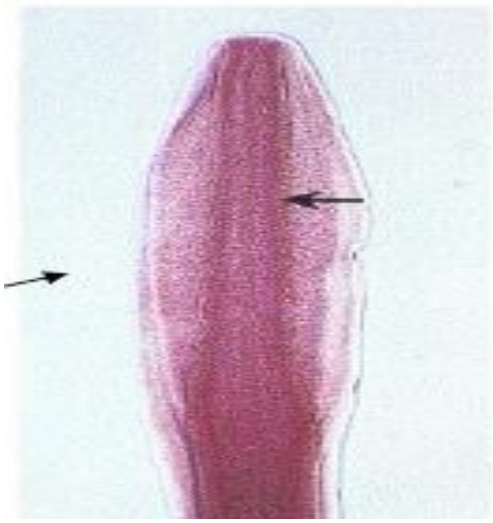
General Tapeworm Morphology

1- Scolex: anterior attachment structure

- According taxonomic order there are two types of scolex:
- Scolex contains slit-like suckers
- Scolex contains central domed area with or without hooks.
- Scolex is an important taxonomic structure.

2. Neck: undifferentiated region posterior scolex.



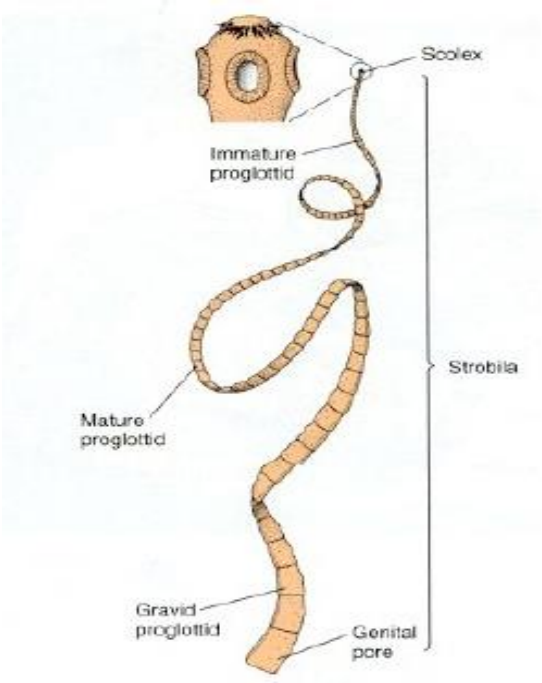


3. Strobilla:
Linear series of segments or proglottids.
Proglottids are continuously formed posterior to the neck region in a process called strobilization.
A new proglottids move posteriorly and beome sexually mature:

- Immature proglottids
- Mature proglottids
- Gravid proglottids

4. Gravid proglottids
Male and female organs in gravid proglottids degenerate as the uterus fills with eggs.
Gravid proglottids often detach from the strobili:

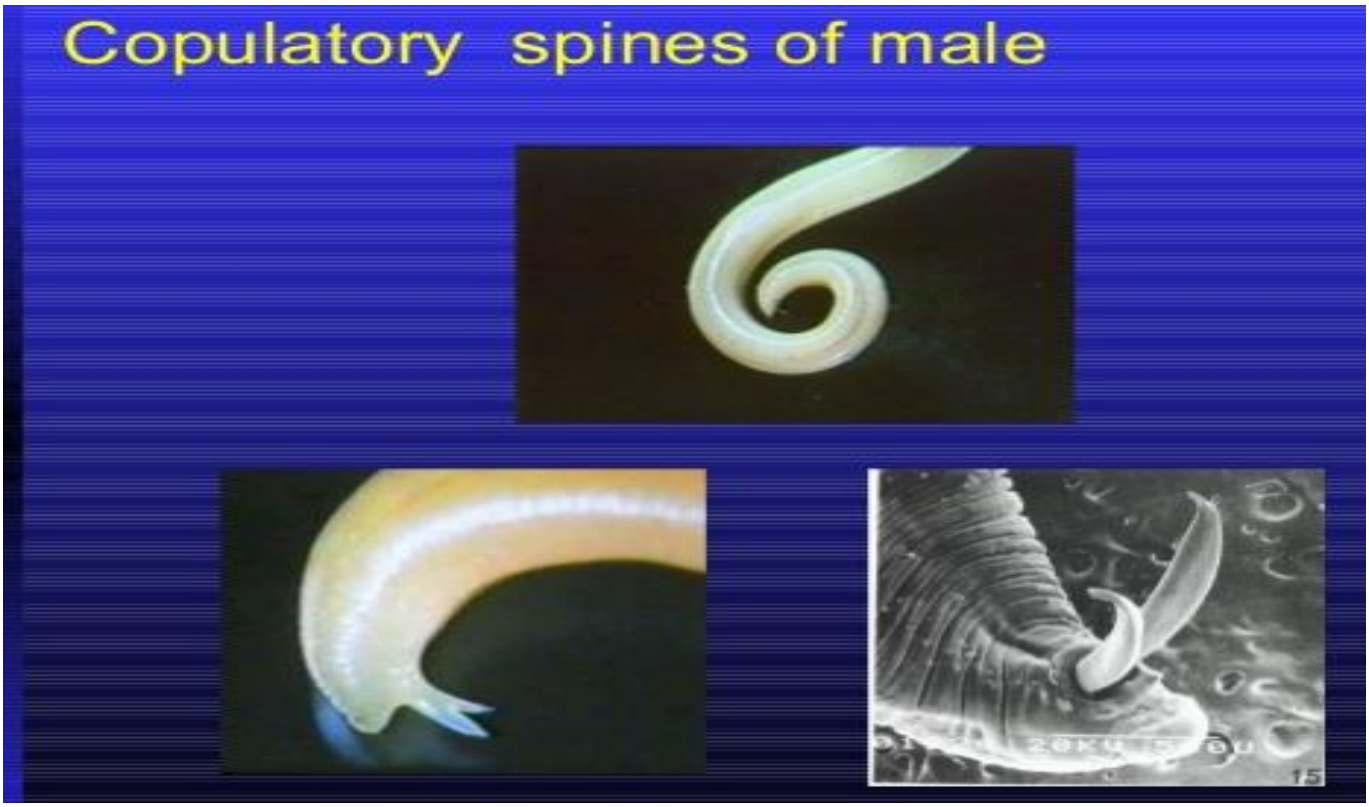
- Disintegrate during passage through the digestive tract releasing eggs in the feces
- Or are released intact in the feces.



Nematodes (round worms)
General characteristics of Nematodes

They assume three basic morphologic forms:

- Egg, Larvae and adult worms
- Reproduce sexually with male and female worms.
- Complete digestive tract and complete reproductive systems
- The adult cylindrical, bilaterally symmetrical and tapered at both end.
- Diocoious (male and female). At the curved posterior end of male is a copulatory organ.



Nematodes (round worms)

Most easily recognized from of intestinal parasite: large size and cylindrical, unsegmented body.

Live primarily as adults in intestine and confirmed by identification of eggs in feces

- Hookworms

Ascaris lumbricoides
Necatur americanus

- Whipworm

Trichuris trichiura

- Pinworm

Enterobius vermicularis
filarialworm

Ancylostoma doudenale
Ancylostoma ceylanicum

Infetions is transmitted by:

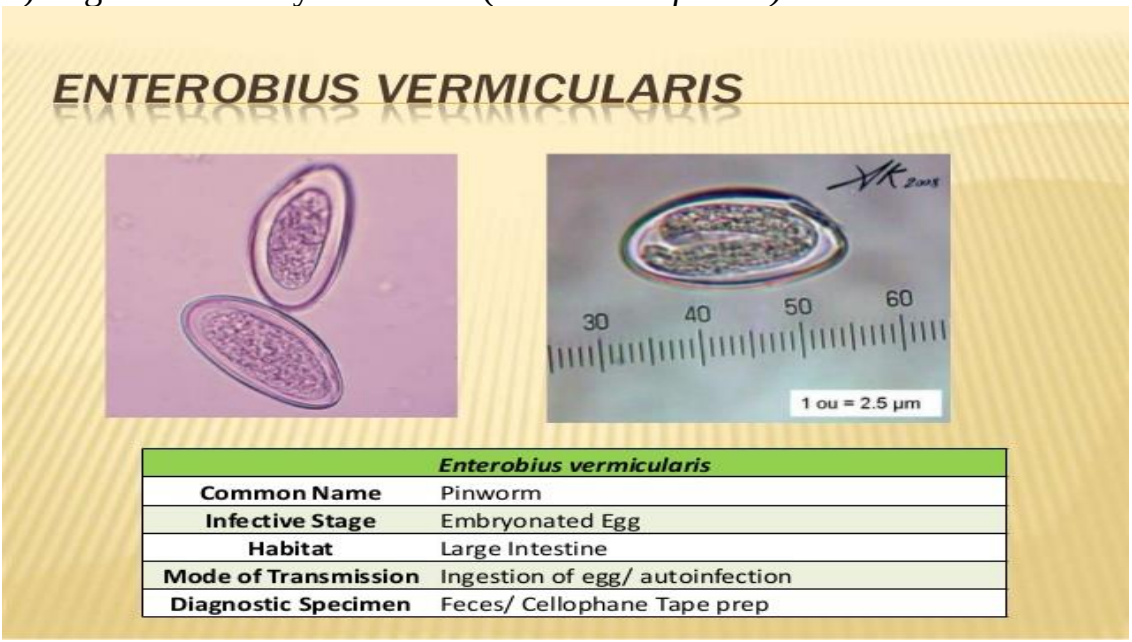
1) Ingestion of eggs

(*Ascaris lumbriccoides)*

1) Penetration of larvae through surfaces

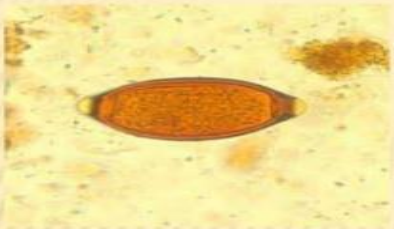
(Necatur americanus)

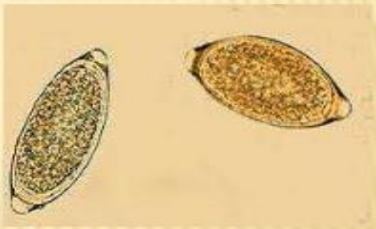
1) Insect bite (*Filaria)*
2) Ingestion of encysted larvae (*Trichnella spiralis)*



Enterobius vermicularis	
Common Name	Pinworm
Infective Stage	Embryonated Egg
Habitat	Large Intestine
Mode of Transmission	Ingestion of egg/ autoinfection
Diagnostic Specimen	Feces/ Cellophane Tape prep

TRICHURIS TRICHURA

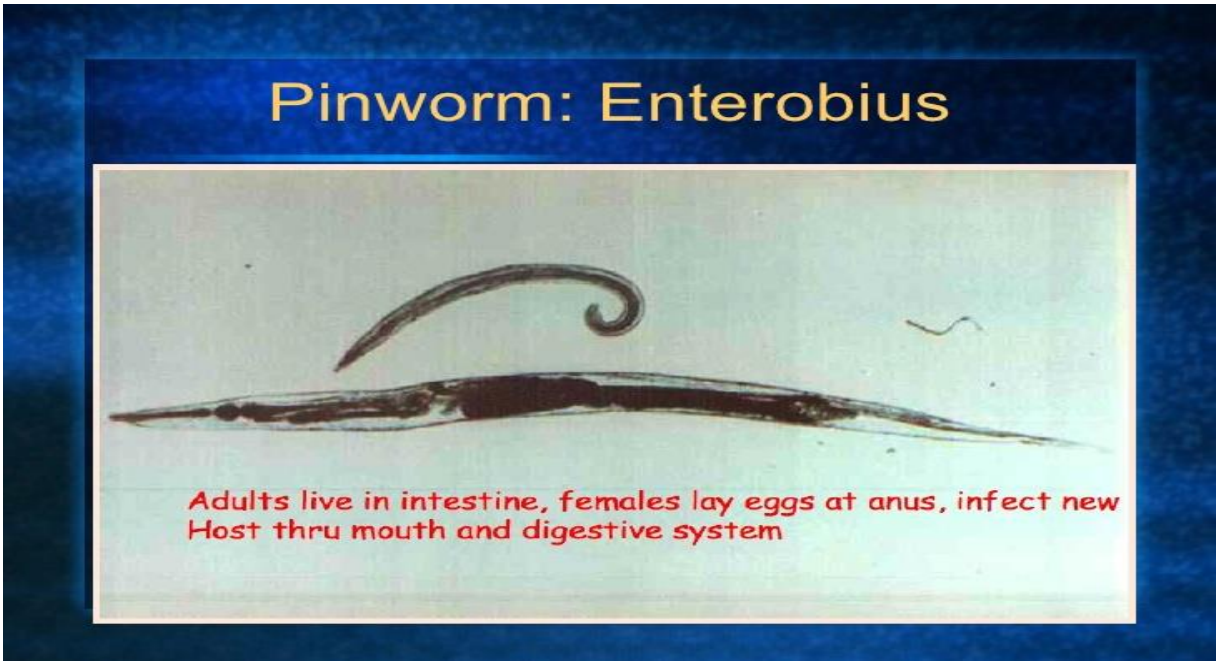


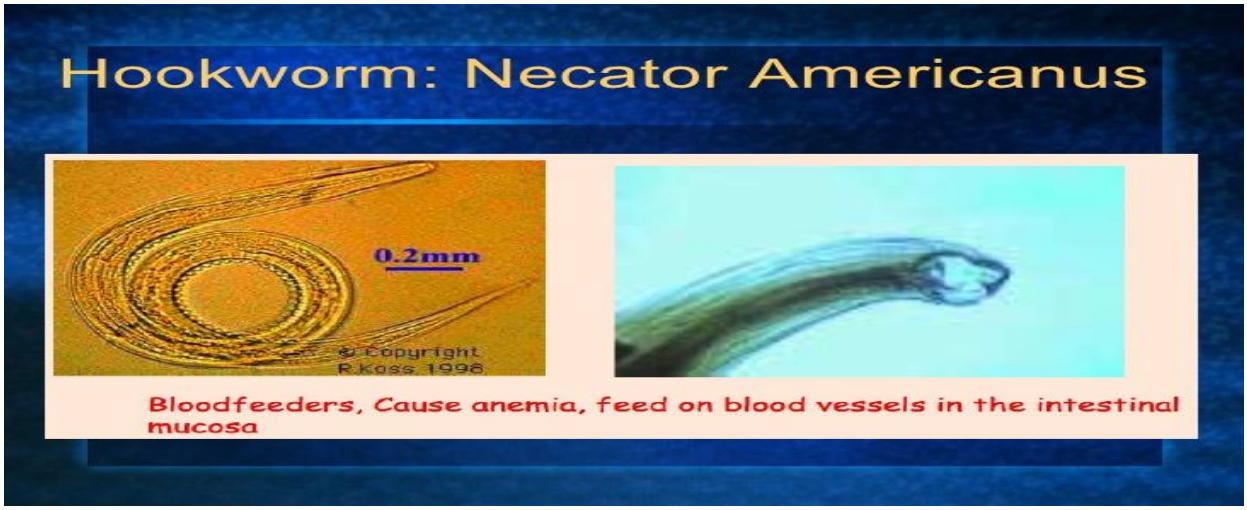


Trichuris trichura	
Common Name	Whipworm
Infective Stage	Embryonated Egg
Habitat	Large Intestine
Mode of Transmission	Ingestion of egg via contaminated food/ water
Diagnostic Specimen	Feces

TRICHURIS TRICHURA 2


pinworm
roundworm


Pinworm: Enterobius
Adults live in intestine, females lay eggs at anus, infect new
Host thru mouth and digestive system


Hookworm: Necator Americanus
0.2mm
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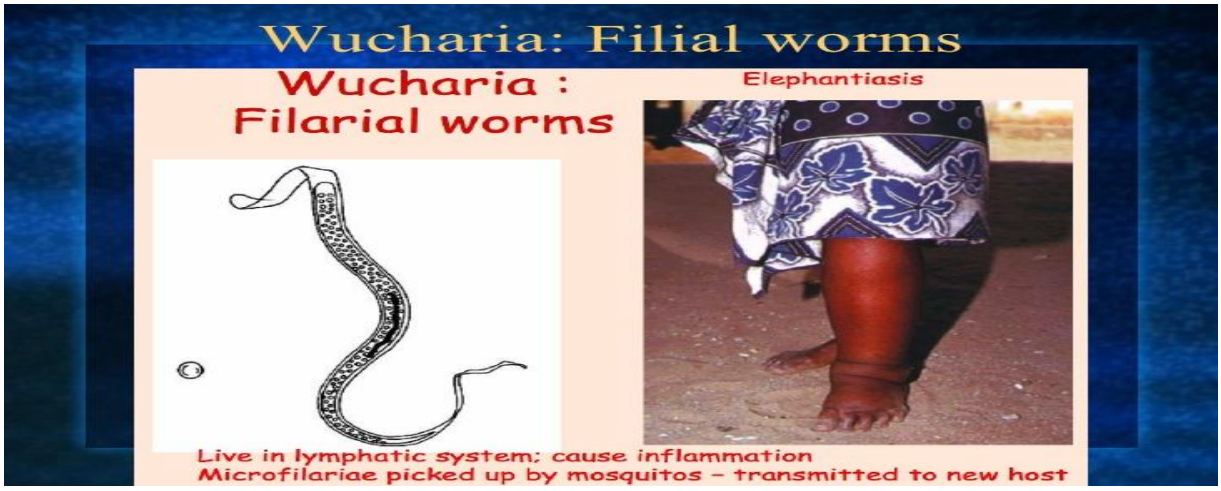
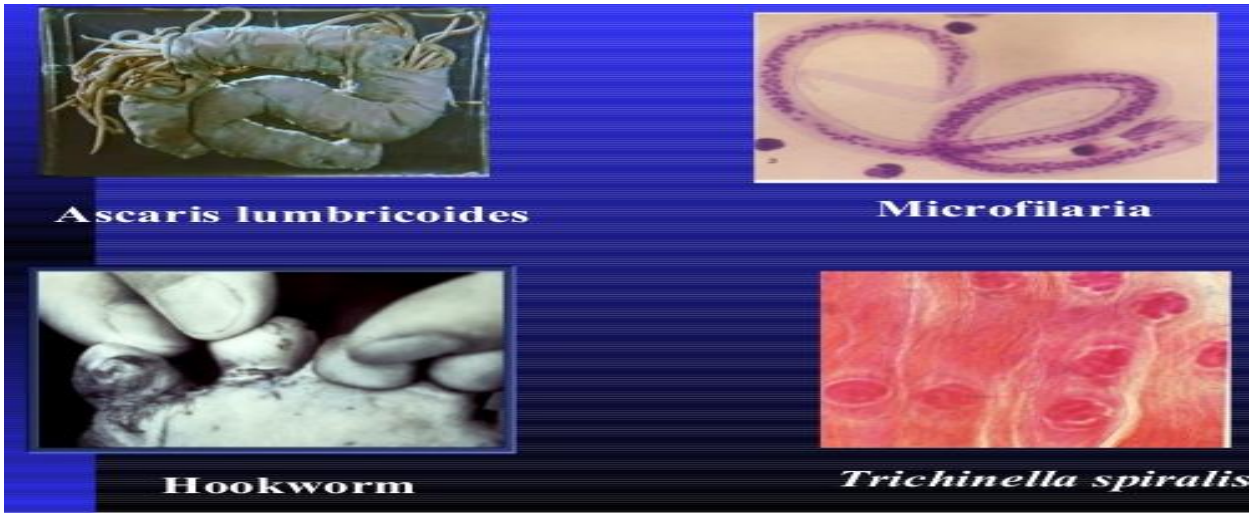
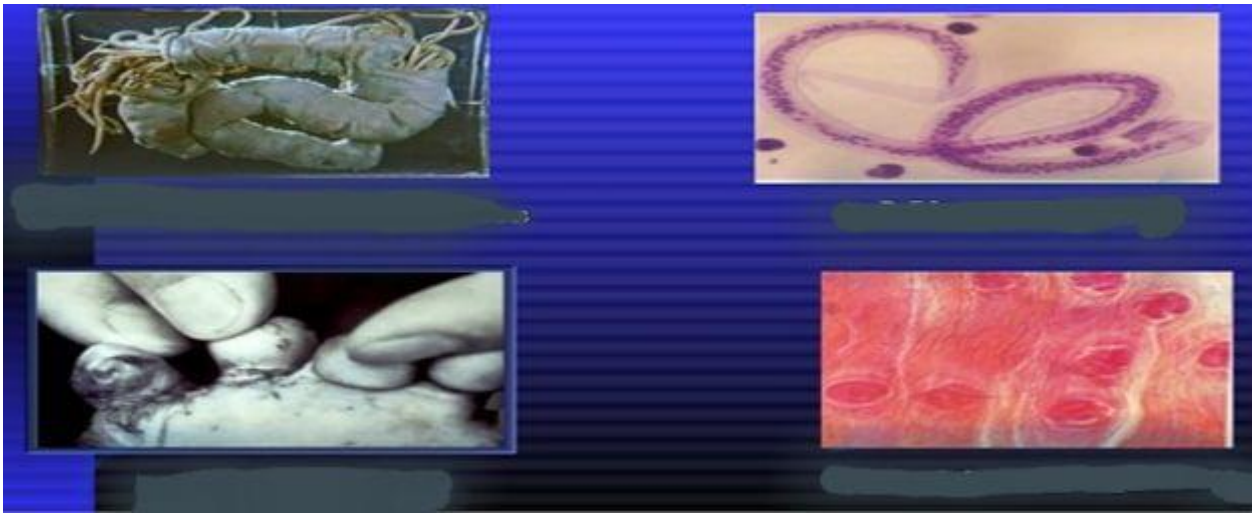
R.Koss 1998
Bloodfeeders, Cause anemia, feed on blood vessels in the intestinal mucosa


Wucharia: Filial worms
Wucharia :
Filarial worms
Elephantiasis
Live in lymphatic system; cause inflammation
Microfilariae picked up by mosquitos – transmitted to new host


Ascaris lumbricoides
Microfilaria
Hookworm
Trichinella spiralis

نشاط (1/2/7)

Q\Diagnosis of the following worms



الوحدة الثامنة - المحاضرة الأولى - الزمن: 90 دقيقة

أهداف المحاضرة الاولى:

يتوقع في نهاية الجلسة أن يكون الطالب قادراً على:

1- Difference between *Taenia saginata* &*T. soluim* through the scolex
2- Enumerate the morphology of *Taenia saginata* &*T. soluim*
3- Draw the Life cycle of *Taenia Saginata* &*T. soluim*
4- Enumerate the Disease symptoms of *Taenia Saginata* &*T. soluim*
5- Diagnosis of egg, Proglottids and scolex of *Taenia Saginata* &*T. soluim*

موضوعات المحاضرة الاولى:

- Genus Taenia including *Taenia saginata* &*T. soluim*
- History and Distribution
- habitat -
- Morphology, mode of infection, infective stage,-
- Life cycle of *Taenia Saginata*-
- Life Cycle of *Taenia Solium*
- Pathogenicity and Clinical Features
- laboratory diagnosis

الأساليب والأنشطة والوسائل التعليمية

م	الأساليب والأنشطة التدريبية	الوسائل التدريبية
1	• محاضرة • مناقشة • سؤال وجواب • نشاط (1/1/8) نشاط	• جهاز حاسوب • جهاز عرض • سبورة • اوراق واقلام

خطة إجراءات تنفيذ المحاضرة الاولى

الوحدة	المحاضرة	الإجراءات	الزمن بالدقيقة
الثامنة	الأولى	القاء المحاضرة مستخدما جهاز العرض والسبورة	90 دقيقة
		طرح بعض الاسئلة خلال القاء المحاضرة	
		نشاط (1/1/8) تمرين ارسم مخطط	

المادة العلمية:

Taenia Saginata and *Taenia Solium*

Common name

Taenia saginata - Beef tapeworm
Taenia solium - Pork tapeworm

History and Distribution

T. saginata has been known as an intestinal parasite of man from very ancient times. But it was only in 1782 when Goeze differentiated
it from the pork tapeworm, T. solium. Its life cycle was elucidated when Leuckart, in 1861, first experimentally demonstrated that cattle serve as the intermediate host for the worm.
*The name Taenia is derived from the Greek word meaning tape or band. It was originally used to refer to most tapeworms, but is now restricted to the members of the Genus Taenia.
*T. saginata is worldwide in distribution, but the infection is not found in vegetarians and those who do not eat beef. T. solium is also worldwide in distribution except in the countries and communities, which proscribe pork as taboo.

Habitat

The adult worms of both T. saginata and T. solium live in the human small intestine, commonly in the jejunum.
Cestode living in small intestine
••*Diphyllobothrium latum*
••*Taenia solium*
••*Taenia saginata saginata*
••*Taenia saginata asiatica*
••*Hymenolepis nana*

Morphology

Adult Worm of T. saginata The adult T. saginata worm is opalescent white in color, ribbon-like, dorsoventrally flattended, and segmented, measuring 5–10 m in length.
*The adult worm consists of head (scolex), neck, and strobila (body). The general features of adult worm are similar to any cyclophyllid an cestodes.
*Scolex: The scolex (head) of T. saginata is about 1–2 mm in diameter, quadrate in cross-section, bearing 4 hemispherical suckers situated at its four angles. They may be pigmented. The scolex has no rostellum or hook lets (which are present in T. solium) T.

saginata is, therefore called the unarmed tape worm. The suckers serve as the sole organ for attachment.
*The neck is long and narrow. The strobila (trunk) consists of 1000 to 2000 proglottides or segments—immature, mmature and gravid.
*The gravid segments are nearly four times as they are broad, about 20 mm long and 5 mm broad. The segment contains male and female reproductive structures. The testes are numerous, 300 to 400 (twice as many as in T. solium. The gravid segment has 15 to 30 lateral branches (as against 7 to 13 in T. solium). It differs from T. solium also in having a prominent vaginal sphincter and in lacking the accessory ovarian lobe. The common genital pore opens on the lateral wall of the segments.
*The gravid segments break away and are expelled singly, actively forcing their way out through the anal sphincter. As there is no uterine opening, the eggs escape from the uterus through its ruptured wall.

Adult Worm of T. solium
*The adult worm is usually 2-3 meters long.
*The scolex of T. solium is small and globular about 1 mm in diameter, with 4 large cup-like suckers (0.5 mm in diameter), and a conspicuous rounded rostellum, armed with a double row of alternating round and small dagger-shaped hooks, 20–50 in number.
*The neck is short and half as thick as the head.
*The proglottides number less than a thousand. They resemble those of T. saginata in general. The gravid segments are twice as long as broad, 12 mm by 6 mm. The testes are composed of 150 to 200 follicles. There is an accessory lobe for the ovary. The vaginal sphincter is absent. The uterus has only 5 to 10 (under 13) thick lateral branches. A lateral thick-lipped genital pore is present, alternating between the right and left sides of adjacent segments.
*The gravid segments are not expelled singly, but pass passively out as short chains. The eggs escape from the ruptured wall of the uterus.
The other differentiating features of *T. saginata* and *T. solium.*

Eggs
Eggs of both species are indistinguishable.
*The egg is spherical, measuring 30–40 μm in diameter.
*It has a thin hyaline embryonic membrane around it, which soon disappears after release.
*The inner embryophore is radially striated and is yellow-brown due to bile staining.
*In the center is a fully-developed embryo (oncosphere) with 3 pairs of hooklets (hexacanth embryo).
*The eggs do not float in saturated salt solution.

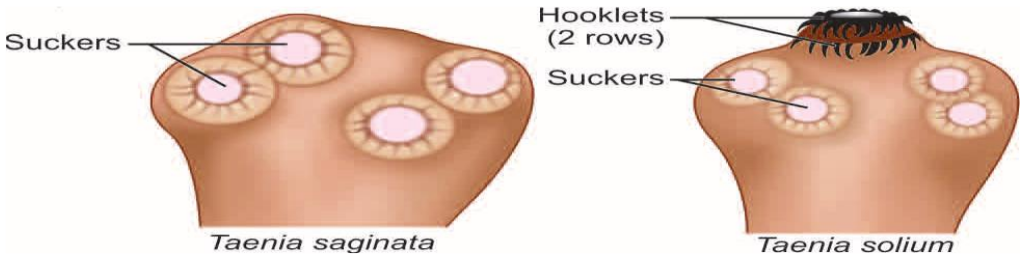
*The eggs of T. saginata are infective only to cattle and not to humans, whereas the eggs of T. solium are infective to pigs and humans too.

Larva

The larval stage of Taenia is called as cysticercus.

*Cysticercus bovis is the larva of T. saginata.

*Cysticercus cellulosae is the larva of T. solium



Scolex of *Taenia saginata* and *Taenia solium*
Difference between *Taenia saginata* and *Taenia solium*

	Taenia saginata	*Taenia solium*
Length	5–10 m	2–3 m
Scolex	Large quadrate	Small and globular
	Rostellum and hooks are absent	Rostellum and hooks are present
	Suckers may be pigmented	Suckers not pigmented
Neck	Long	Short
Proglottids	1,000–2,000	Below 1,000
Measurement (gravid segment)	20 mm × 5 mm	12 mm × 6 mm
Expulsion	Expelled singly	Expelled passively in chains of 5 or 6
Uterus	Lateral branches 15–30 on each side; thin and dichotomous	Lateral branches 5–10 on each side; thick and dendritic
Vagina	Present	Absent
Accessary lobe of ovary	Absent	Present
Testes	300–400 follicles	150–200 follicles
Larva	Cysticercus bovis; present in cow not in man	Cysticercus cellulosae; present in pig and also in man
Egg	Not infective to man	Infective to man
Definitive host	Man	Man
Intermediate host	Cow	Pig, occasionally man
Disease	Causes intestinal taeniasis	Causes intestinal taeniasis and cysticercosis

Larva of T. saginata Cysticercus bovis

* It is the larval form of T. saginata.
* The name cysticercus in derived from the Greek, kystis—bladder and kerkos—tail.
*The larva (cysticercus bovis) is infective stage for humans.
* The cysticercus is an ovoid, milky-white opalescent fluid-filled vesicle measuring about 5 mm × 10 mm in diameter, and contains a single invaginated scolex (bladder worm).
*The cysticerci are found in the muscles of mastication, cardiac muscles, diaphragm and tongue of infected cattle.
They can be seen on visual inspection as shiny white dots in the infected beef (measly beef).
* Cysticercus bovis is unknown in humans.

larva of T. solium Cysticercus cellulosae

*It is the larval form of T. solium and also the infective form of the parasite.
* It can develop in various organs of pig as well as in man.

* The cysticercus cellulosae or 'bladder worm' is ovoid opalescent milky-white, measuring 8–10 mm in breadth and 5 mm in length.
*The scolex of the larva, with its suckers, lies invaginated within the bladder and can be seen as a thick white spot. It remains viable for several months.

Life Cycle of Taenia Saginata

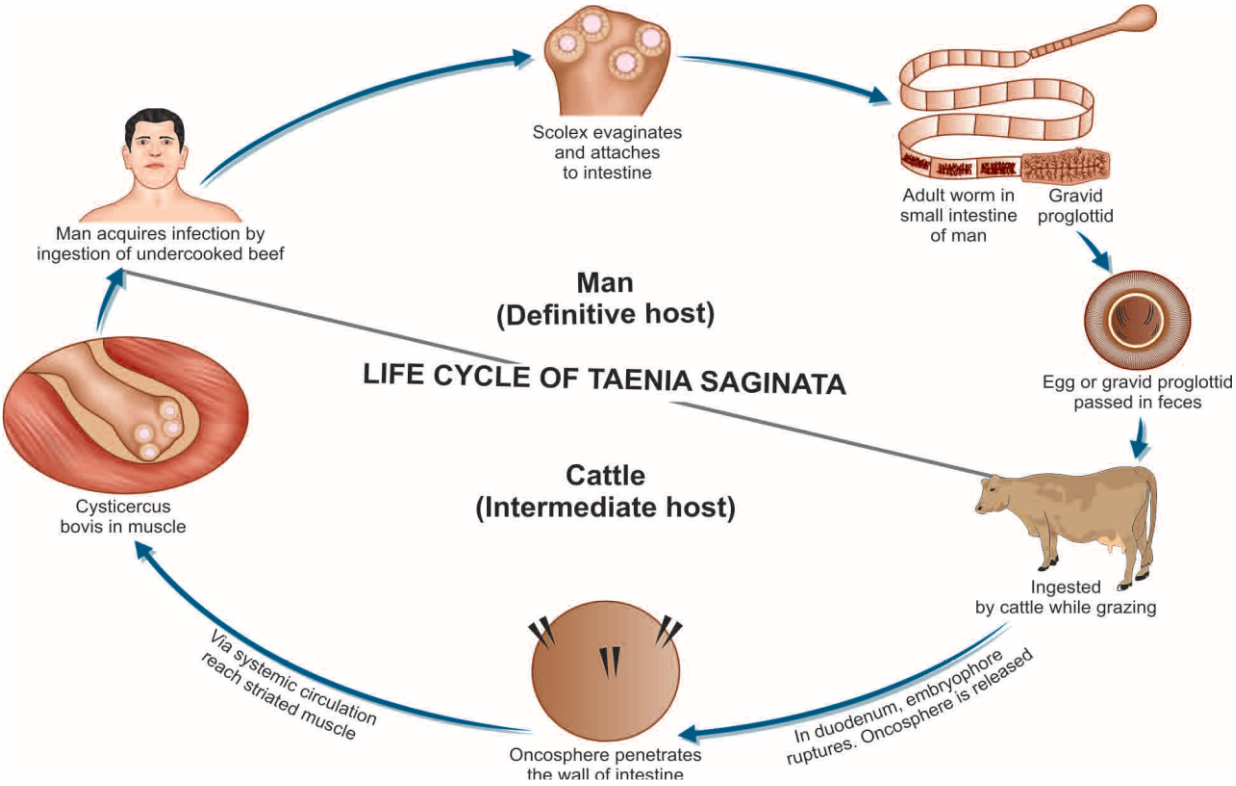
T. saginata passes its life cycle in 2 hosts.
Definitive host: Humans are the definitive hosts and harbor the adult worm.
Intermediate host: Cattle (cow or buffalo) are the intermediate host and harbor the larval stage of the worm.
Infective stage: Cysticercus bovis (larval stage) is the infective stage to man, while eggs are infective to cattle.
*The adult worm lives in the small intestine of man. The gravid segments from the adult worm breakaway and are expelled singly. They actively force their way-out through the anal sphincter.
*The eggs or gravid segments are passed out with feces on the ground.
*The eggs deposited in soil remain viable for several weeks.
*They are infective to cattle, which ingest the eggs while grazing.

Development in Cattle

When ingested by cattle (cow or buffalo), the eggshell ruptures releasing onchosphere in the duodenum.
*The onchospheres, with their hooklets penetrate the intestinal wall, reach the mesenteric venules or lymphatics and enter the systemic circulation.
*They get filtered out in the striated muscles, particularly in muscles of the tongue, neck, shoulder, ham, and in the myocardium. In these sites, the onchospheres lose their hooks and in about 60–70 days develop in the mature larva, cysticercus bovis.
*The cysticerci can live in flesh of cattle for about 8 months, but can develop further only when ingested by man, its definitive host.

Development in Man

Man acquires infection by ingesting raw or undercooked beef containing cysticerci.
*The cysticerci are digested out of the meat in the stomach.
*In the upper part of the small intestine, the head (scolex) evaginates out of the cysticercus, becomes attached to the mucosa, and by gradual strobilization develops into the adult worm in about 2–3 months. The adult worm has a life span of 10 years or more.
Infection in usually with a single worm, but sometimes multiple infection is seen and 25 or more worms have been reported in patients.



Life Cycle of Taenia Solium

When T. solium causes intestinal taeniasis, its life cycle is similar to that of T. saginata except.

Definitive host: Man

Intermediate host: Pig

Infective stage: Cysticercus cellulosae (larva)

*Humans are infected by consuming inadequately cooked pork containing cysticercus cellulosae (measly pork).

*Man harboring adult worms may autoinfect oneself either by unhygenic personal habits or by reverse peristaltic movements of the intestine.

When Taenia leads to cysticercosis, the life cycle is as follows:

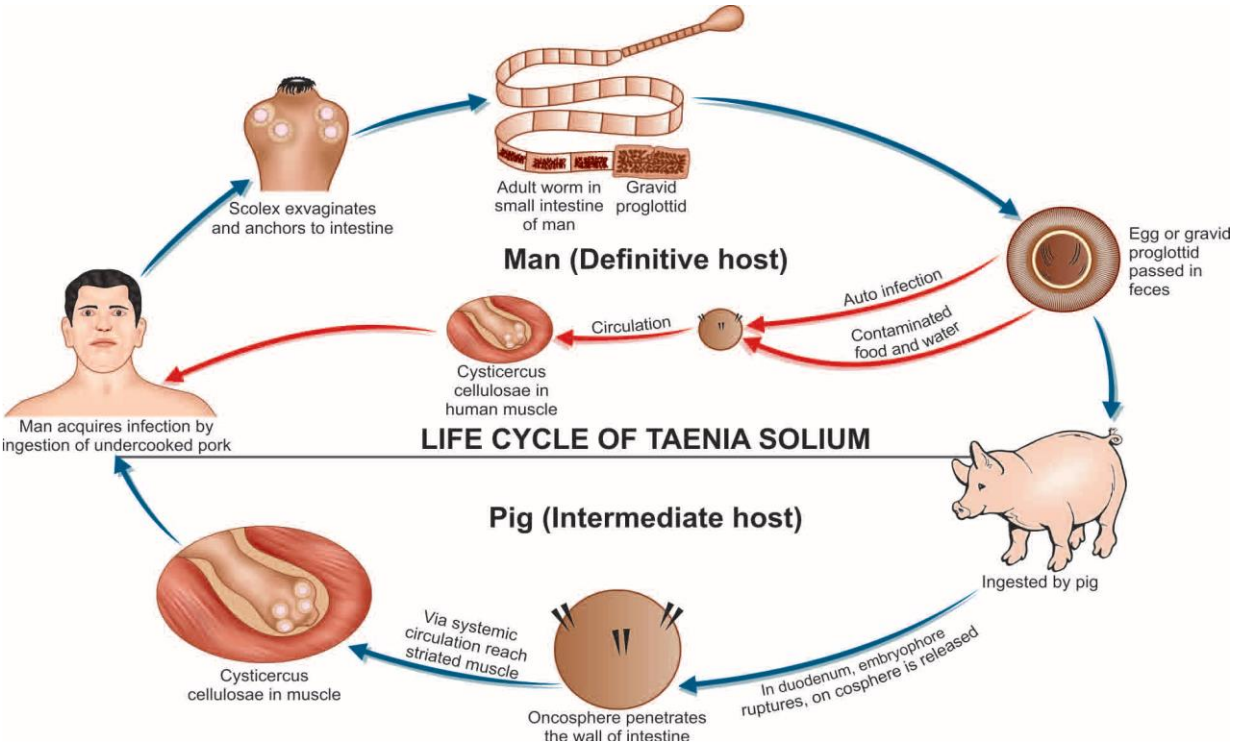
Definitive host and Intermediate host: Both man

Infective stage: Eggs of T. solium (not larva)

*Mode of infection: Man acquires infection by ingesting eggs with contaminated food and water.

*Autoinfection: A man harboring adult worm may auto infect oneself, either by unhygenic personal habits or by reverse peristalsis of the intestine.unhygenic personal habits or by reverse peristalsis of the intestine.

*The further development of the eggs is similar in man and pigs.
*The oncospheres are released in the duodenum or jejunum and penetrate the intestinal wall.
*They enter the mesenteric venules or lymphatics and are carried in systemic circulation to the different parts of the body.
*They are filtered out principally in the muscles, where they develop into the larval stage, cysticercus cellulosae in about 60–70 days.
*In humans, it is a dead end and the larvae die without further development.
••Intestinal infection with T. solium occurs only in persons eating undercooked pork and usually in persons of low socio-economic condition with poor sanitation. It is uncommon in Jews and Mohammedans, who are not generally pork eaters. But cysticercosis may occur in any person residing in endemic areas, even in vegetarians because the mode of infection is contamination of food or drink with egg deposited in soil.
••Eggs of T. solium are infective to pigs as well as to man.



Pathogenicity and Clinical Features

Intestinal Taeniasis

It can be caused by both T. saginata and T. solium.

*The adult worm, inspite of its large size, causes surprisingly little inconvenience to the patient.

*When the infection is symptomatic, vague abdominal discomformt, indigestion, nausea, diarrhea, and weight loss may be present. Occasional cases of acute intestinal obstruction, acute appendicitis, and pancreatitis have also been reported.

Cysticercosis

It is caused by larval stage (cysticecus cellulosae) of T. solium.

*Cysticercus cellulosae may be solitary or more often multiple.

*Any organ or tissue may be involved, the most common being subcutaneous tissues and muscles. It may also affect the eyes, brain, and less often the heart, liver, lungs, abdominal cavity, and spinal cord.

*The cysticercus is surrounded by a fibrous capsule except in the eye and ventricles of the brain.

*The larvae evoke a cellular reaction starting with infiltration of neutrophils, eosinophils, lymphocytes, plasma cells, and at times, giant cells. This is followed by fibrosis and death of the larva with eventual calcification.

*The clinical features depend on the site affected Subcutaneous nodules are mostly asymptomatic Muscular cysticerosis may cause acute myositis Neurocysticerosis (cysticercosis of brain) is the most common and most serious form of cysticercosis.

About 70% of adult-onset epilepsy is due to neurocysticercosis. Other clinical features of neuro-cysticercosis are increased intracranial tension, hydrocephalus, psychiatric disturbances, meningoencephalitis, transient paresis, behavioral disorders aphasia, and visual disturbances. It is considered as the second most common cause of intracranial space occupying lesion (ICSOL) after Tuberculosis in India.

*In ocular cysticercosis, cysts are found in vitreous humor, subretinal space and conjunctiva. The condition may present as blurred vision or loss of vision, iritis, uveitis, and palpebral conjunctivitis.

Laboratory Diagnosis

Stool Examination

* Eggs

€ Microscopic examination of feces shows characteristic eggs of Taenia in 20–80% of patients.

€ Formol ether sedimentation method of stool concentration is useful.

€ Eggs can also be detected by cellophane swab method (NIH Swab) in 85–95% patients.

€ Species identification cannot be made from the eggs, since the eggs of T. saginata and T. Solium are Similar.

* Proglottids

€ Species identification can be done by examining with a hand lens, the gravid proglotlid pressed between
2 slides, when branching can be made out. (15–20 lateral branches in T saginata; under 13 in T. Solium).

* Scolex

€ Definitive diagnosis can also be established by demonstration of unarmed scolex in case of T. saginata after antihelmenthic treatment.

*Detection of Taenia Antigen in feces: Antigen capture enzyme-linked immunosorbent assay (ELISA) using polyclonal anti sera against Taenia is employed to detect coproantigen in feces since 1990 and is more sensitive than microscopy (specificity 100% and sensitivity 98%). The drawback of the test is that it cannot differentiate between T. saginata and T. solium.

Serodiagnosis

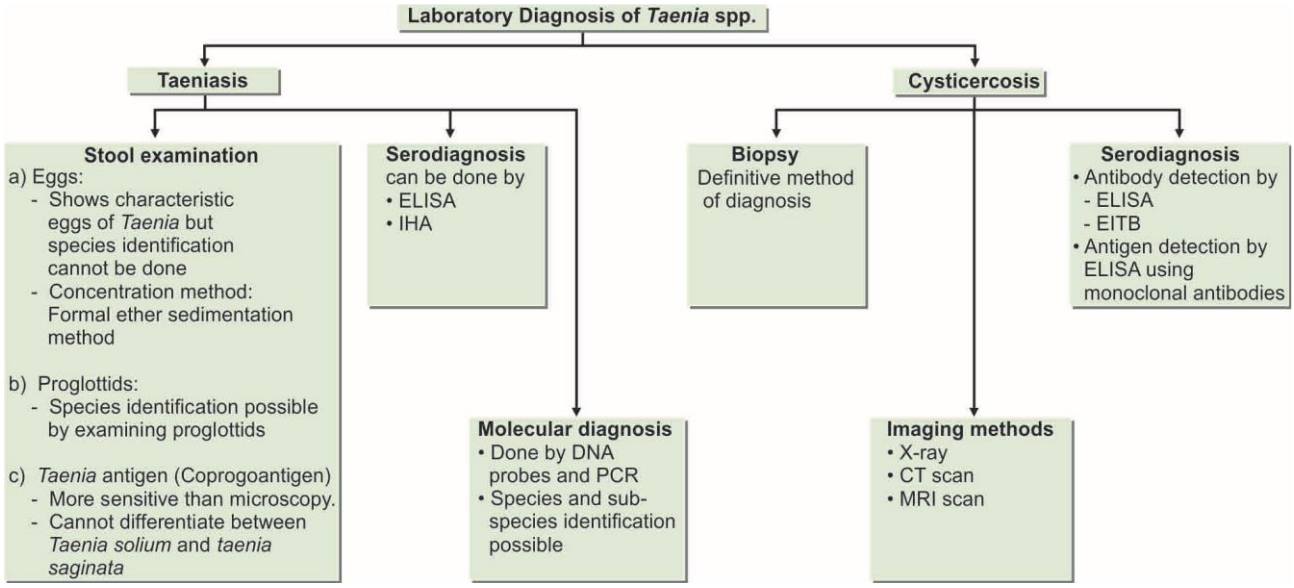
Specific antibodies in serum can be demonstrated by ELISA, indirect immunofluroscence test and indirect hemagglutination (IHA) test.

Molecular Diagnosis

Both DNA probes and polymerase chain reaction (PCR) technique are used to detect and differentiate between eggs and proglottids of T. saginata and T. solium.
It can also differentiate between the 2 subspecies of
T. saginata, viz T. saginata saginata and T. saginata asiatica.

Laboratory Diagnosis of Cysticercosis

Diagnosis of cysticercosis is based on the following:



Laboratory diagnosis of Taenia spp.

Biopsy

Definitive diagnosis of cysticercosis is by biopsy of the lesion and its microscopic examination to show the invaginated scolex with suckers and hooks.

Imaging Methods

*__X-ray__: Calcified cysticerci can be detected by radiography of subcutaneous tissue and muscles particularly in the buttocks and thigh. X-ray of the skull many demonstrate cerebral calcified cyst.

*__Computed tomography (CT) scan__ of brain is the best method for detecting dead calcified cysts. The cysticercal lesions appear as small hypodensities (ring or disc-like) with a bright central spot.

*__Magnetic resonance imaging (MRI) scan__ of the brain is more helpful in detection of non-calcified cysts and ventricular cysts. It also demonstrates spinal cysticerci.

Serology

Antibody detection

Anticysticercus antibodies in serum or cerebrospinal fluid (CSF) can be detected by ELISA and enzyme-linked immunoelectrotrasfer blot (EITB) tests.

Antigen detection

Antigen can be detected in serum and CSF by ELISA, using monoclonal antibodies and indicate recent infection.

Others

*Ocular cysticercosis can be made out by opthalmoscopy

*Eosinophilia: Usually occurs in early stage of cysticercosis, but is not constant.

Cysticercosis

*For cysticercosis, excision is the best method, wherever possible.
*Asymptomatic neurocysticercocis requires no treatment.
*For symptomatic cerebral cysticercosis, praziquantel in a dose of 50 mg/kg in 3 divided doses for 20–30 days and albendazole in a dose of 400 mg twice daily for 30 days may be administered.
*Corticosteroids may be given along with praziquantel or albendazole to reduce the inflammatory reactions caused by the dead cysticerci.
*In addition, antiepileptic drugs should be given until the reaction of the brain has subsided.
*Operative intervention is indicated for hydrocephalus.

Note

*Beef and pork to be eaten by man should be subjected to effective inspection for in slaughter house.
*Avoidance of eating raw or undercooked beef and pork. The critical thermal point of cysticercus is 56°C for 5 minutes.
*Maintainence of clean personal habits and general sanitary measures.

Note

*Commonest, large ribbon-like tapeworm.
*Rostellum and hooks absent (unarmed tapeworm).
*1,000–2,000 proglottids with 15–30 dichotomously branched uterus.
*Definitive host: Man
*Intermediate host: Cow
*Mode of infection: Undercooked (measly) beef containing cysticercus bovis
*Eggs are not infective to human.
*Asymptomatic, clinical features occur occasionally— abdominal discomfort, indigestion.
*Diagnosis: Eggs or proglottids in stool, serodiagnosis, molecular diagnosis
excision in case of cysticercosis.

نشاط (1/1/8)

Q\ Trace the life cycle *Taenia Saginata* from the egg to the adult worm.

الوحدة الثامنة - المحاضرة الثانية - الزمن: 90 دقيقة

أهداف المحاضرة الثانية:

يتوقع في نهاية الجلسة أن يكون الطالب قادراً على:

1- Enumerate the morphology of *Echinococcus granulosus*
2- Knowing How a person gets infected with a helminths *Echinococcus granulosus*
3- Enumerate the diagnosis of *Echinococcus granulosus*
4- What are the hosts of *Echinococcus granulosus*

موضوعات المحاضرة الثانية:

- Genus *Echinococcus granulosus*
- History and Distribution-
- Habitat
- Morphology
- Life Cycle
- Pathogenesis
- Clinical Features
- Laboratory Diagnosis

الأساليب والأنشطة والوسائل التعليمية

م	الأساليب والأنشطة التدريبية	الوسائل التدريبية
1	• محاضرة • مناقشة • سؤال وجواب • نشاط (1/2/8) نشاط	• جهاز حاسوب • جهاز عرض • سبورة • اوراق واقلام

خطة إجراءات تنفيذ المحاضرة الثانية

الوحدة	المحاضرة	الإجراءات	الزمن بالدقيقة
الثامنة	الثانية	القاء المحاضرة مستخدما جهاز العرض والسبورة	90 دقيقة
		طرح بعض الاسئلة خلال القاء المحاضرة	
		نشاط (1/2/8) تمرين تعداد	

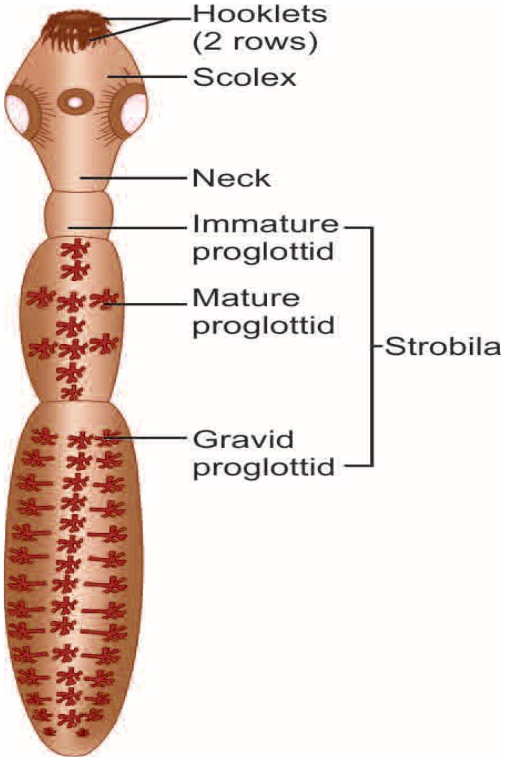
المادة العلمية:

Echinococcus Granulosus
Common name: Dog tape worm

History and Distribution
Hydatid cysts had been described by Hippocrates and other ancient physicians.
*Adult E. granulosus was described by Hartmann in the small intestine of dog in 1695 and the larval form (hydatid cysts) was recognized in 1782 by Goeze.
*The disease is prevalent in most parts of the world, though it is most extensive in the sheep and cattle raising areas of Australia, Africa, and South America. It is also common in Europe, China, and the Middle East.
*It is a significant health problem in India. It is seen more often in temperate than in tropical regions.

Habitat
*The adult worm lives in the jejunum and duodenum of dogs and other canine carnivore (wolf and fox).
*The larval stage (hydatid cyst) is found in humans and herbivorus animals (sheep, goat, cattle and horse).

Morphology
Adult Worm
It is a small tapeworm, measuring only 3–6 mm in length.
*It consists of a scolex, a short neck, and strobila.
*The scolex is pyriform, with 4 suckers and a prominent rostellum bearing 2 circular rows of hooklets (25–30).



*The neck is short than the rest of the worm (3 mm × m6 mm).
*The strobila is composed of only 3 proglottids, the anterior immature, the middle mature, and the posterior gravid segment.
*The terminal proglottid is longer and wider than the rest of the worm and contains a branched uterus filled with eggs.
*The adult worm lives for 6–30 months.

Egg

*The eggs of Echinococcus are indistinguishable from those of Taenia species.
*It is ovoid in shape and brown in color.
*It contains an embryo with 3 pairs of hooklets.

Larval Form

The larval form is found within the hydatid cyst developing inside various organs of the intermediate host.
*It represents the structure of the scolex of adult worm and remains invaginated within a vesicular body.
*After entering the definitive host, the scolex with suckers and rostellar hooklets, becomes exvaginated and develops into adult worm.

Life Cycle

The worm completes its life cycle in 2 hosts:

Definitive hosts: Dog (optimal host), wolf, jackal, and fox

Intermediate host: Sheep and Cattle. Sheep is the ideal intermediate host.

* Man acts as an accidental intermediate host (dead end).

*The larval stage of the parasite is passed in intermediate hosts, including man, giving rise to hydatid cyst.

*The adult worm lives in the small intestine of dogs and other canine animals. These animals discharge numerous eggs in the feces.

*Intermediate hosts (sheep and cattle) ingest them while grazing.

*Human infection follows ingestion of the eggs due to intimate handling of infected dogs or by eating raw vegetables or other food items contaminated with dog feces.

*The ova ingested by man or by sheep and cattle are liberated from the chitinous wall by gastric juice liberating the hexacanth embryos which penetrate the intestinal wall and enter the portal venules, to be carried to the liver along the portal circulation.

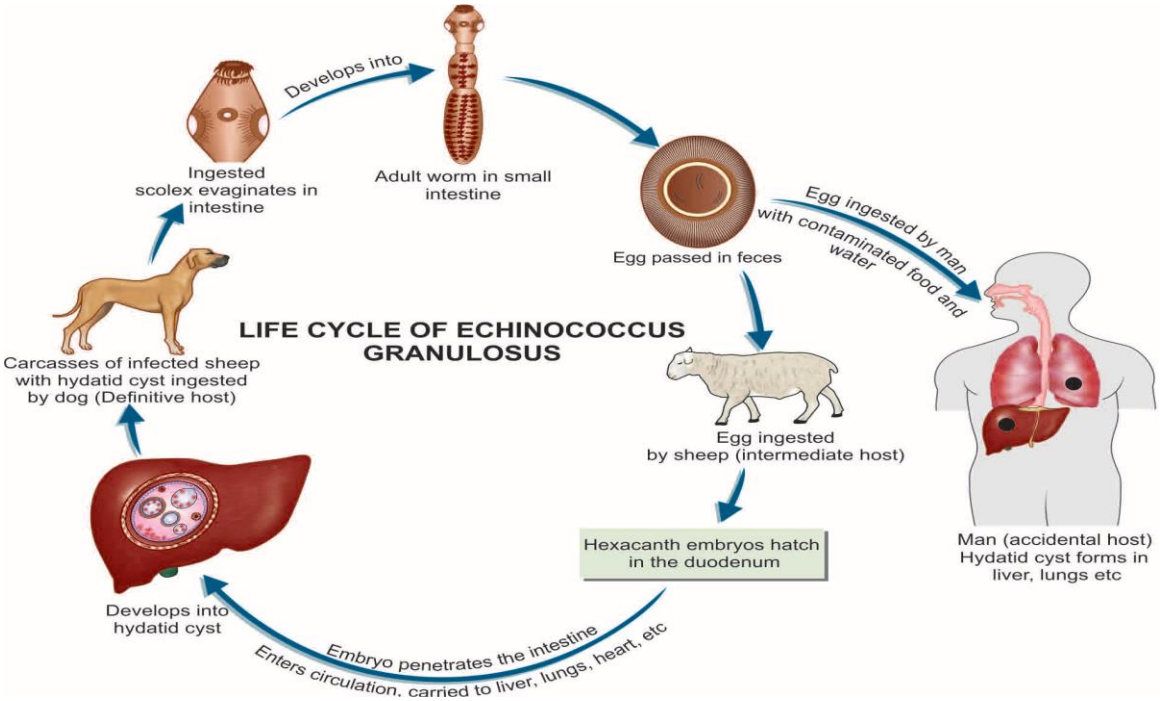
*These are trapped in hepatic sinusoids, where they eventually develop into hydatid cyst. About 75% of hydatid cyst develop in liver, which acts as the first filter for embryo.

*However, some embryo which pass through the liver, enter the right side of heart and are caught in pulmonary capillaries (forming pulmonary hydatid cysts), so that the lung acts as the second filter.

*A few enter the systemic circulation and get lodged in various other organs and tissues such as the spleen, kidneys, eyes, brain, or bones.

*When sheep or cattle harboring hydatid cysts die or are slaughtered, dogs may feed on the carcass or offal. Inside the intestine of dogs, the scolices develop into the adult worms that mature in about 6–7 weeks and produce eggs to repeat the life cycle.

*When infection occurs in humans accidentaly, the cycle comes to a dead end because the human hydatid cysts are unlikely to be eaten by dogs.



Pathogenesis

Evolution of Hydatid Cyst

At the site of deposition, the embryo slowly develops into a hollow bladder or cyst filled with fluid.

This becomes the hydatid cyst (Greek hydatis: a drop of water).

*It enlarges slowly and reaches a diameter of 0.5–1 cm in about 6 months. The growing cyst evokes host tissue reaction leading to the deposition of fibrous capsule around it.

*The cyst wall secreted by the embryo consists of indistinguishable layers.

*Pericyst is the outer host inflammatory reaction consisting of fibroblastic proliferation, mononuclear cells, eosinophils, and giants cells, eventual developing into dense fibrous capsule which may even calcify.

*Ectocyst is the intermediate layer composed of characteristic a cellular, chitinous, laminated hyaline material. It has the appearance of the white of a hardboiled egg.

* Endocyst is the inner germinal layer which is cellular and consists of number of nuclei embedded in a protoplasmic mass and is extremely thin (22–25 mm).

The germinal layer is the vital layer of the cyst and is the site of asexual reproduction giving rise to brood capsules with scolices. It also secretes hydatid fluid, which fills the cyst.

*Hydatid fluid: The interior of the cyst is filled with a clear colorless or pale-yellow fluid called as hydatid fluid.

*pH of the fluid is 6.7 (acidic).

Composition: It contains salts (sodium chloride 0.5%, sodium sulphate, sodium phosphate, and salts of succinic acid) and proteins.
* It is antigenic and highly toxic so that its liberation into circulation gives rise to pronounced eosinophilia or may even cause anaphylaxis.
* The fluid was used as the antigen for Casoni's intradermal test.
*A granular deposit or hydatid sand is found at the bottom of the cyst, consisting of free brood capsules and protoscolices and loose hooklets.

Brood capsules
From the germinal layer, small knob-like excrescences or gem mules protrude into the lumen of the cyst. These enlarge, become vacuolated, and are filled with fluid. These are called as brood capsules.
*They are initially attached to the germinal layer by a stalk, but later escape free into the fluid-filled cyst cavity.
*From the inner wall of the brood capsules, protoscolices (new larvae) develop, which represent the head of the potential worm, complete with invaginated scolex, bearing suckers and hooklets.
*Several thousands of protoscolices develop into a mature hydatid cyst, so that this represents an asexual reproduction of great magnitude.
*Inside mature hydatid cysts, further generation of cyst, daughter cysts and grand-daughter cysts may develop. The cyst grows slowly often taking 20 years or more to become big enough to cause clinical illness and is therefore, particularly seen in man.
Acephalocysts
Some cysts are sterile and may never produce brood capsules, while some brood capsule may not produce scolices. These are called acephalocysts.
Fate of hydatid cysts
The cyst may get calcified or spontaneously evacuated following inflammatory reaction. Hydatid cyst of liver may rupture into lung or other body cavity producing disseminated hydatid lesions.

Clinical Features
*Most of the times infection is asymptomatic and accidentally discovered.
*Clinical disease develops only when the hydatid cyst has grown big enough to cause obstructive symptoms.
Disease results mainly from pressure effects caused by the enlarging cysts.
*In about half the cases, the primary hydatid cyst occurs in liver (63%) in the right lobe. Hepatomegaly, pain, and obstructive jaundice are the usual main festations.

*The next common site is the lung (25%) (most common being the lower lobe of the right lung). Cough, hemoptysis, chest pain, pneumothorax, and dyspnea constitute the clinical picture.
*In the kidney (2%), hydatid cyst causes pain and hematuria.
*Other sites affected include spleen (1%), brain (1%), pelvic organs, orbit, and bones (3%). Cerebral hydatid cysts may present as focal epilepsy.
€ * When hydatid cyst is formed inside the bones, the laminated layer is not well developed because of confinement by dense osseous tissues. The parasite migrates along the bony canals as naked excrescences that erode the bone tissue. This is called osseous hydatid cyst. Erosion of bone may lead to pathological fractures.
*Apart from pressure effects, another pathogenic mechanism in hydatid disease is hypersensitivity to the echinococcal antigen. The host is sensitized to the antigen by minute amounts of hydatid fluid seeping through the capsule. Hypersensitivity may cause urticaria. But if a hydatid cyst ruptures spontaneously or during surgical interference, massive release of hydatid fluid may cause severe, even fatal anaphylaxis.

Laboratory Diagnosis

Imaging

Radiological examinations and other imaging techniques such as ultrasonography (USG), CT scan, and MRI reveal the diagnosis in most cases of cystic echinococcosis
*USG is the diagnostic procedure of choice. Cyst wall typically shows double echogenic lines separated by a hypoechoic layer (double contour). Pathogenic findings include daughter cysts and the ‘water-lily’ sign due to detached endocyst floating within the cavity.
*CT scan is superior for the detection of extrahepatic disease.
*MRI appears to add diagnostic benefit for cysts, especially at difficult sites such as spinal vertebrae and cardiac cysts.
*Plain X-rays permit the detection of hydatid cyst in lung and bones. In cases where long bones are involved, a mottled appearance is seen in the skiagram.
*IV pyelogram is often helpful for detection of renal hydatid cyst.

Examination of Cyst Fluid

Examination of aspirated cyst fluid under microscope after trichome staining reveals scolices, brood capsules, and hooklets. Exploratory puncture of the cyst to obtain cystic fluid should be avoided as it may cause escape of hydatid fluid and consequent anaphylaxis. Therefore, fluid aspirated from surgically removed cyst should only be examined

Casoni's Intradermal Test

It is an immediate hypersensitivity (Type 1) skin test introduced by Casoni in 1911, using fresh sterile hydatid fluid. The antigen in hydatid fluid is collected from animal or human cysts and is sterilized by Seitz or membrane filtration. The fluid is injected (0.2 mL) intradermally in one arm and an equal volume of saline as control is injected in the other arm. In a positive reaction, a large wheal of about 5 cm in diameter with multiple pseudopodia like projections appears within half an hour at the test side and fades in about an hour. A secondary reaction consisting of edema and induration appears after 8 hours. The test is almost abandoned now due to non specificity and has been supplemented by serological tests.

Serology

Antibody detection

*Detection of serum antibodies using specific antigens (8and 16 KDA) from hydatid fluid are frequently used to support the clinical diagnosis of cystic echinococcosis.

The tests include IHA, indirect immnofluroscence, and ELISA. In hepatic cysts, the sensitivity of test is relatively superior (85–98%) than pulmonary cyst (50–60%).

*The slide latex agglutination test and immune electrophoresis using hydatid fluid fraction 5 antigen are also widely used. Precipitin test and complement fixation test (CFT) with hydatid antigen have also been ound to be positive. CFT is not very sensitive and false positive reaction is seen in those receiving neural

antirabic vaccine. CFT is useful after surgical removal of cysts, when a negative test has a better prognostic value.

Antigen detection

Specific echinococcal antigen in sera and in CSF can be detected by double diffusion and counter immunoelectrophoresis technique (CIEP).

Blood Examination

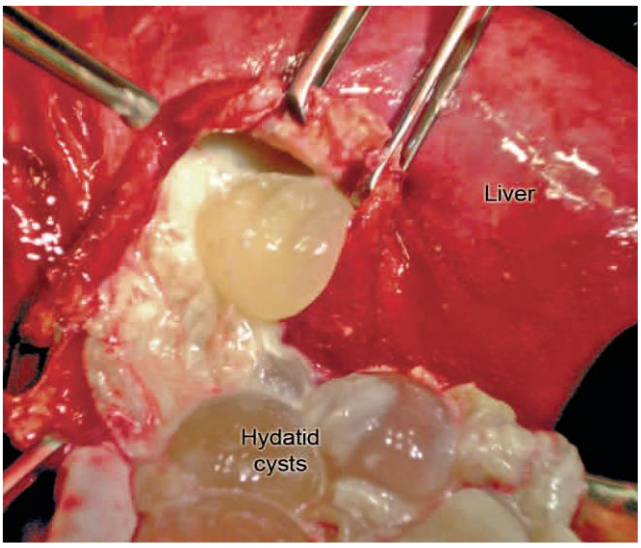
It may reveal a generalized eosinophilia of 20–25%.

Excretion of the Scolices

Excretion of scolices into the sputum or urine may be observed in pulmonary or renal cyst, respectively and can be demonstrated by acid fast staining or lactophenol cotton blue **(LPCB) staining.**

Specific Molecular Diagnostic

Specific molecular diagnostic methods have been developed involving DNA probes and PCR, but their application is limited by their technical complexity.



Laboratory Diagnosis of *Echinococcus granulosus*

Imaging techniques	Examination of cyst fluid	Casoni's test	Serodiagnosis	Others
• **USG:** Diagnostic procedure of choice • **CT scan:** For extra-hepatic disease • **MRI:** For cysts in spinal vertebrae and cardiac cysts • **X-ray:** For cysts of bones and lungs • **IV pyelogram:** For renal cysts	• Reveals- Scolices, brood capsules and hooklets • Diagnostic puncture of cyst is not recommended	• Immediate hypersensitivity skin test • Abandoned due to non-specificity	1) **Antibody detection** Tests detecting antibody against antigen B (8 and 16 KDA) • IHA • Indirect immunofluorescence • ELISA Tests detecting antibody against hydatid fluid fraction 5 antigen • CFT • Precipitation test 2) **Antigen detection** • Double diffusion • CIED	• Blood- shows eosinophilia • Molecular diagnosis by DNA probes and PCR

Treatment

Traditionally surgical removal was considered as the best mode of treatment of cysts. Currently, ultrasound staging is recommended and management depends on the stage.

In early stages, the treatment of choice is punture, aspiration, injection, and reaspiration (PAIR).

Puncture, Aspiration, Injection, and re aspiration (PAIR)

PAIR, considered as a controversial procedure earlier, is now widely used in early stages of the disease.

Induction of PAIR

••Cysts with internal echoes on ultrasound (snowflake sign) multiple cysts, cysts with detached laminar membrane.

••Contraindications of PAIR for superficially located cysts, cysts with multiple thick internal septal divisions (honey combing pattern), cysts communicating with biliary tree.

The basic steps involved in PAIR include-

* Ultrasound or CT- guided puncture of the cyst

€ * Aspiration of cyst fluid

€ * Infusion of scolicidal agent (usually 95% ethanol; alternatively, hypertonic saline)

*Reaspiration of the fluid after 5 minutes.

*Great care is taken to avoid spillage and cavities are sterilized with 0.5% silver nitrate or 2.7% sodium chloride for prophylaxis of secondary peritoneal echinococcosis due to inadvertent spillage of fluid during PAIR.

*Albendazole (15 mg/kg in 2 divided doses) is initiated 4 days before the procedure and continued for 4 weeks.

Scolicidal agent and there complications

••Cetrimide—can cause acidosis

••Alcohol 95%—can cause cholangitis

••Hypertonic saline—hypernatraemia

••Sodium hypochlorite—hypernataraemia

••Hydrogen peroxide

Note: In cases with biliary communication only hypertonic saline (15–20%) is used

Surgery

It is the treatment of choice for complicated E. granulosus cysts like those communicating with the biliary tract and in those cysts where PAIR is not possible.

*The preferred surgical approach is pericystectomy. For pulmonary cyst, treatment consists of wedge resection or lobectomy.

*Recurrence after surgery is common.

*Pre- and postostoperative chemotherapy with albendazole for 2 years after curative surgery is recommended.

*Positron emission tomography (PET) scanning can be used to follow disease activity.

*Other new treatment modalities include laparoscopic hydatid liver surgery and percutaneous thermal ablation (PTA) of the germinal layer of the cyst using radiofrequency ablation device.

Chemotherapy

Chemotherapy with benzimidazole agents are restricted to residual, post-surgical, and inoperable cysts. Albendazole and praziquantel have proved beneficial.

Notes:

E. granulosus infection can be prevented by:

* Ensuring pet dogs do not eat animal carcass or offal. Periodical deworming of pet dogs.

*Destruction of stray and infected dogs.

*Mantaining personal hygiene such as washing of hands after touching dogs and avoidance of kissing pet dogs.

نشاط (1/2/8)

Q\Enumerate of hosts that *Echinococcus granulosus* undergoes during its life cycle

الوحدة الثامنة - المحاضرة الثالثة - الزمن: 90 دقيقة

أهداف المحاضرة الثالثة:

يتوقع في نهاية الجلسة أن يكون الطالب قادراً على:

1- Difference between *Schistosoma Mansoni* & *Schistosoma haematobium* through the egg
2- Enumerate the morphology of *Schistosoma Mansoni* & *Schistosoma haematobium*
3- Draw the Life cycle of *Schistosoma Mansoni* & *Schistosoma haematobium*
4- Enumerate the Disease symptoms of *Schistosoma Mansoni* & *Schistosoma haematobium*
5- diagnosis of egg of *Schistosoma Mansoni* & *Schistosoma haematobium*

موضوعات المحاضرة الثالثة:

- Genus *Schistosoma*
- *Schistosoma Mansoni*: History and Distribution
- Habita-
- Morphology
- Life cycle
- Pathogenicity and Clinical Features
- Laboratory Diagnosis
- **Schistosoma haematobium*
- Habitat
- Morphology
- Life cycle
- Pathogenicity and Clinical Features
- Laboratory Diagnosis

الأساليب والأنشطة والوسائل التعليمية

م	الأساليب والأنشطة التدريبية	الوسائل التدريبية
1	• محاضرة • مناقشة • سؤال وجواب • نشاط (1/3/8) نشاط	• جهاز حاسوب • جهاز عرض • سبورة • اوراق واقلام

خطة إجراءات تنفيذ المحاضرة الثالثة

الوحدة	المحاضرة	الإجراءات	الزمن بالدقيقة
الثامنة	الثالثة	القاء المحاضرة مستخدما جهاز العرض والسبورة	90 دقيقة
		طرح بعض الاسئلة خلال القاء المحاضرة	
		نشاط (1/3/8) تمرين مقارنة مع الرسم	

المادة العلمية:

Schistosoma Mansoni

History and Distribution

In 1902, Manson discovered eggs with lateral spines in the feces of a West Indian patient that led to the recognition of this second species of human schistosomes. It was, therefore named S. mansoni.

*It is widely distributed in Africa, South America, and the Caribbean islands.

Habitat

Adult worm lives in the inferior mesenteric vein.

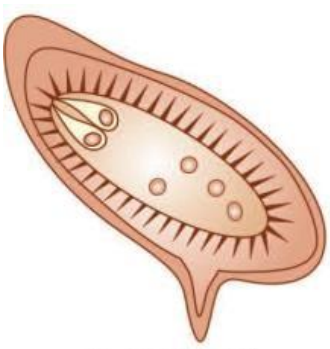
Morphology

S. mansoni resembles S. haematobium in morphology and life cycle, except The adult worms are smaller and their integuments studded with prominent coarse tubercles.

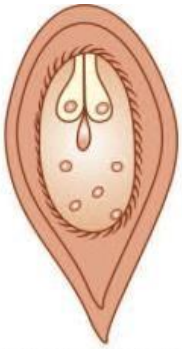
*In the gravid female, the uterus contains very few eggs, usually 1–3 only.

*The prepatent period (the interval between cercarial penetration and beginning of egg laying) is 4–5 weeks.

*The egg has a characteristic lateral spine more near to the rounded posterior end. The eggs are non-operculated and yellowish brown.



S. mansoni
Ova with a lateral spine
(obtained from stool)

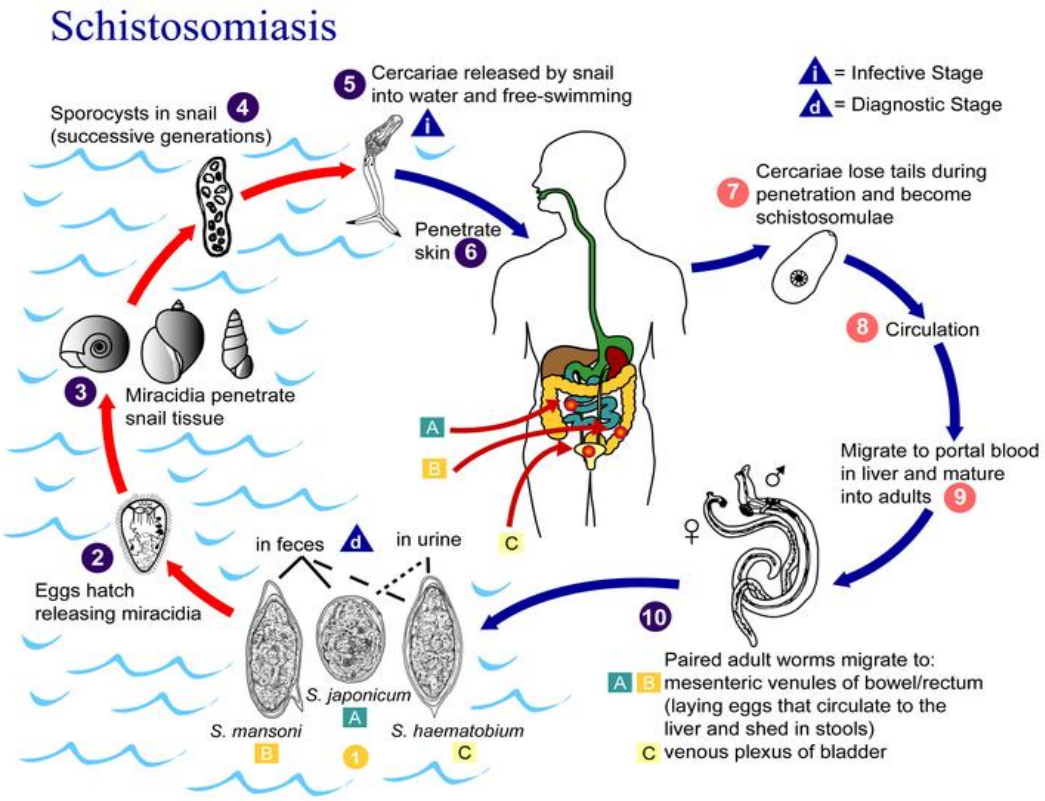


S. haematobium
Ova with a terminal spine
(obtained from urine)

S. japonicum
Ova with a lateral knob
(obtained from stool)
Note: The characteristic surround of tissue particles

Life cycle

Definitive host: Humans are the only natural definitive hosts, though in endemic areas monkeys and baboons have also been found infected. Intermediate host: Planorbid fresh-water snails of the genus Biomphalaria.



Schematic diagram to show distinguishing features of eggs of S. mansoni, S. haematobium, and S. japonicum Infective form: Fork-tailed cercaria. In humans, the schistosomulae mature in the liver and the adult worms move against the blood stream into the venules of the inferior mesenteric group in the sigmoidorectal area. Eggs penetrate the gut wall, reach the colonic lumen, and are shed in feces.

Pathogenicity and Clinical Features

*Following skin penetration by cercariae: A pruritic rash called as cercarial dermatitis or swimmers itch may develop locally. Cercarial dermatitis is a self-limiting clinical entity.
*During maturation and at the beginning of oviposition (i.e. 4–8 weeks after skin invasion): Acute schistosomiasis or Katayama fever (a serum sickness-like syndrome) with fever, rash, myalgia, arthralgia, cough, generalized lymph adenopathy, and hepatosplenomegaly may develop.
*Individual with acute schistosomiasis show high peripheral blood eosinophilia.
*Parasite-specific antibodies may be detected at this stage before schistosoma eggs are identified in feces.
*During the stage of egg deposition: The symptomatology is mainly intestinal as the eggs are deposited in the small intestine. This condition is, therefore known as intestinal bilharziasis or schistosomal dysentery. Patients develop colicky abdominal pain and bloody diarrhea, which may go on intermittently for many years. €*The eggs deposited in the gut wall cause inflammatory reactions leading to micro- abscesses, granulomas, hyperplasia, and eventual fibrosis. Egg granulomas are found in the distal part of the colon and rectum. Ectopic lesions include hepatosplenomegaly and periportal fibrosis, portal hypertension, as some of the eggs are carried through portal circulation into liver.
* Portal hypertension may cause gastrointestinal hemorrhage.

Laboratory Diagnosis

Stool Microscopy

Eggs with lateral spines may be demonstrated microscopically in stools. Kato-katz thick smear or other concentration methods may be required when infection is light. Kato-katz thick smear provides quantitative data on the intensity of infection, which is of value in assessing the degree of tissue damage and monitoring the effect of chemotherapy.

Rectal Biopsy

Proctoscopic biopsy of rectal mucosa may reveal eggs when examined as fresh squash preparation between 2 slides.

Serological Diagnosis

Serological diagnosis by detecting schistomal antigen and antibody is similar to that of S.haematobium.

Imaging

Ultrasonography (USG) is useful to detect hepatosplenomegaly and periportal fibrosis.

Blood Examination

Blood examination may reveal eosionophilia, and increased levels of alkaline phosphatase.

Schistosoma Japonicum

Common name: Oriental blood fluke

Distribution S. japonicum is found in the far east, Japan, China, Taiwan, Philippines, and Sulawesi.

Habitat

The adult worms are seen typically in the venules of the superior mesenteric vein draining the ileocecal region. They are also seen in the intrahepatic portal venules and hemorrhoidal plexus of veins.

Morphology

Morphologically, they are similar to the schistosomes described above except:

*The adult male is comparatively slender (0.5 mm thick) and does not have cuticular tuberculations.

*In the gravid female, the uterus contains as many as 100 eggs at one time and up to 3,500 eggs may be passed daily by a single worm.

*The prepatent period is 4–5 weeks.

*The eggs are smaller and more spherical than those of S. haematobium and S. mansoni. The egg has no spine, but shows a lateral small rudimentary knob.

Life Cycle

Life cycle of S. japonicum is similar to S. haematobium with the following exceptions. Definitive host: Man is the definitive host but in endemic areas, natural infection occurs widely in several domestic animals and rodents, which act as reservoirs of infection. Intermediate host: Amphibian snails of the genus Oncomelania. Infective form for humans: Fork tailed cercaria.

*Eggs deposited in the superior mesenteric venules penetrate the gut wall and are passed in feces.

*They hatch in water and the miracidia infect the intermediate hosts, amphibian snails of the genus Oncomelania. *The fork-tailed cercaria, which escapes from the snails, is the infective form for men and other definitive hosts.

Pathogenicity and Clinical Features

Disease caused by S. japonicum is also known as oriental schistosomiasis or Katayama disease.

*Its pathogenesis is similar to that in other schistosomiasis, but probably because of the higher egg output, the clinical manifestations are more severe.

Differentiating Features of S. haematobium, S. mansoni and S. japonicum

	Schistosoma haematobium	*Schistosoma mansoni*	*Schistosoma japonicum*
Habitat	Veins of the vesical and pelvic plexuses, less commonly in portal vein and its mesenteric branches	Inferior mesenteric vein and its branches	Superior mesenteric vein and its branches
Morphology Size: *Male* *Female*	 1.5 cm × 1 mm 2 cm × 0.22 mm	 1 cm × 1 mm 1.4 cm × 0.25 mm	 1.2–2 cm × 0.5 mm 2.6 cm × 0.3 mm
Integument	Finely tuberculated	Grossly tuberculated	Non-tubercular
Number of testes	4–5 in groups	8–9 in a zigzag row	6–7 in a single file
Ovary	In the posterior one-third of the body	In the anterior half of the body	In the middle of the body
Uterus	Contains 20-30 eggs	1–3 eggs	50 or more eggs
Egg	Elongated with terminal spine	Elongated with lateral spine	Round with small lateral knob
Cephalic glands in Cercariae	2 pairs oxyphilic and 3 pairs basophilic	2 pairs oxyphilic and 4 pairs basophilic	5 pairs oxyphilic, no basophilic
Distribution	Africa, Near East, Middle East, India	Africa and south America	China, Japan, far east (oriental)
Definitive host	Man	Man	Man (mainly) domestic animals & rodents (which act as reservoir of infection
Intermediate host	Snail of Genus *Bulinus*	Snail of Genus *Biomphalaria*	Amphibian snail of Genus *Oncomelania*

During the acute phase of the disease, Katyama fever is similar to that seen in S. mansoni.
*In the chronic illness intestial, hepatosplenic as well as several manifestations associated with portal hypertension are seen. Intestinal disease manifests as colicky abdominal pain, bloody diarrhoea and anemia. However, the liver is the site maximally affected. There is initial hepatomegaly followed by periportal fibrosis (clay pipe stem fibrosis). Portal Compare with drawing betweenhypertension leads to esophageal varices and gastrointestinal bleeding. The spleen is secondarily enlarged.
*Cerebral and pulmonary involvement (cor pulmonale) may occur in some cases.

Laboratory Diagnosis
Similar to that of S. mansoni

نشاط (1/3/8)

Q\ Compare with drawing between egg of *Schistosoma Mansoni* and egg of *Schistosoma haematobium*