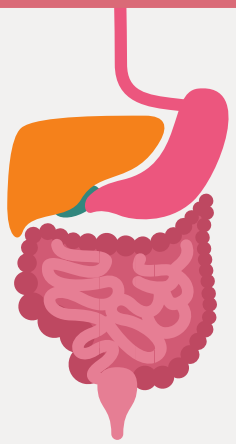
Treatment of dysentery and amebiasis



Objectives:

- To understand different causes of dysentery.
- To describe different classes of drugs used in treatment of both bacillary dysentery and amebic dysentery.
- To be able to describe actions, side effects of drugs for treating bacillary dysentery.
- To understand the pharmacokinetics, actions, clinical applications and side effects of antiamebic drugs.
- To be able to differentiate between types of antiamebic drugs; luminal amebicides, and tissue amebicide.

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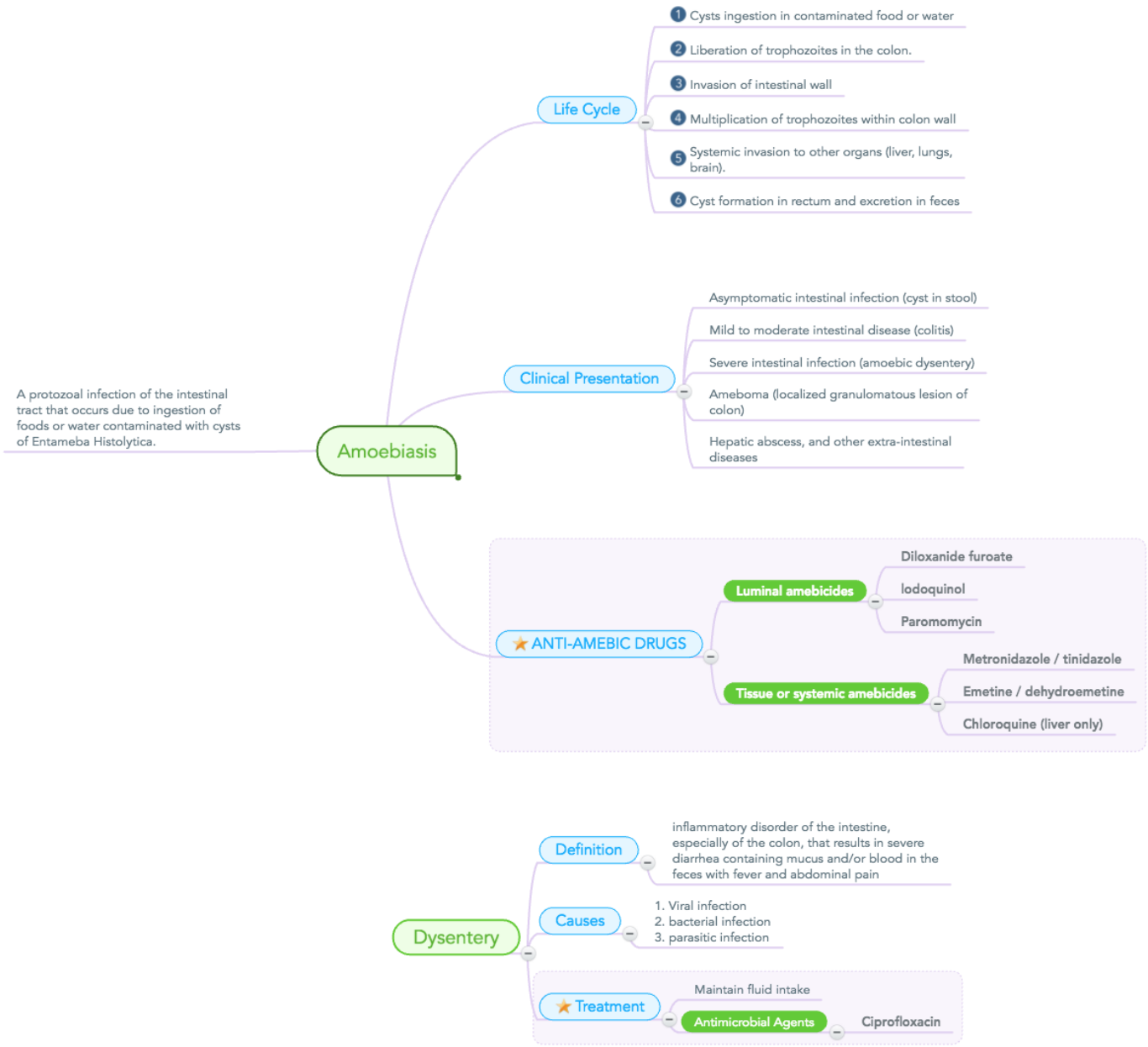
Drugs names | Doctors notes | Important | Extra

« **باذلًا وسعي** في استنقاذها من الهلاك والمرض، والألم والقلق »

Mind map



Click here to see it clearly

Study microbiology lecture first to make it easy!

Then when you study it & don't have enough time, studying slides (5-13) is enough ☺

Dysentery

Definition

It is an inflammatory disorder of the intestine, especially the colon, that results in severe diarrhea containing mucus and/or blood in the feces with fever and abdominal pain caused by any kind of infection.

It results from:

1. Viral infection. 2. Bacterial infection 3. Parasitic infection

Treatment

- Maintain fluid intake using **oral rehydration** therapy or intravenous fluid therapy.
- **Antimicrobial agents** should not be given until stool analysis is done to specify the etiological agent.
- Anti diarrheal drugs are **contraindicated** because they delay fecal excretion that can prolong fever (diphenoxylate or loperamide).

  → Why? Bc When you decreasing GI motility → retaining the organism (not excreted from the body)

The two most common causes:

Amebic dysentery

Protozoal infection
Mainly by ***Entamoeba Histolytica***

Bacillary dysentery

Bacterial infection
Mainly by ***Shigella***

Entamoeba Histolytica exists in two forms:

Cyst (Infective Stage)	Trophozoites (Non-infective, Invasive Stage)
- Can survive outside human body. - When ingested, liberate trophozoites in the lumen of the intestine.	- Multiply and feed on intestinal bacterial flora. - They may invade and ulcerate the wall of large intestine or may migrate to liver or other tissue - In rectum, trophozoites transform to cysts and are excreted in the feces

Amebiasis

Definition

8:48 min

Amebiasis is a protozoal infection of the intestinal tract that occurs due to ingestion of food or water contaminated with cysts of *Entameba Histolytica*. Patients show varying degree of illness from no symptoms to mild diarrhea to severe dysentery.

Clinical presentations

1. Asymptomatic intestinal infection (carriers, passing cysts in stool)
2. Mild to moderate intestinal disease (Colitis)
3. Severe intestinal infection (amoebic dysentery)
4. Ameboma (localized granulomatous lesion of colon)
5. Hepatic abscess, and other extra-intestinal diseases.

Life Cycle:

1. **Cysts** ingestion in contaminated food or water.
2. Liberation of trophozoites in the colon & Invasion of intestinal wall.
3. Multiplication of trophozoites within colon wall.
4. Systemic invasion to other organs (liver, lungs & brain)
5. Cyst formation in rectum & excretion in feces.

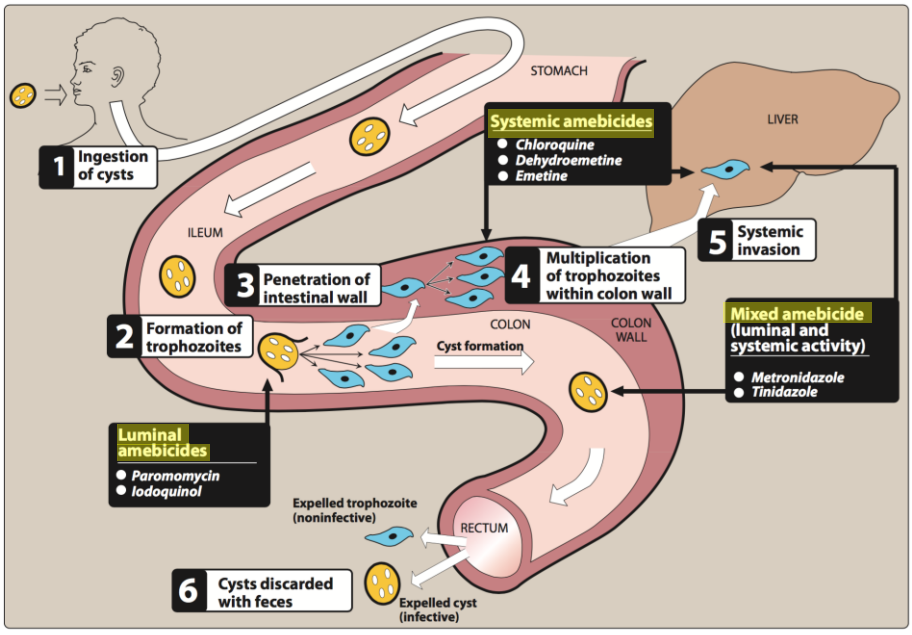


Figure 36.2
Life cycle of Entamoeba histolytica, showing the sites of action of amebicidal drugs.

Antiamebic Drugs

Divided into two types:

Luminal amebicides

Act on the parasites in the **lumen** of the bowel. → They are **poorly absorbed** from the intestine → make their action in the intestine.

Used for the treatment of **Asymptomatic amebiasis** (**carriers**). → didn't invade the wall → the organism in the **cycst** form.

- **Diloxanide furoate**
- **Iodoquinol**
- **Antibiotic:**
  - **Paromomycin**
  - **Tetracycline**

We add antibiotics to the treatment regimen, because it results in a reduction in **intestinal flora**, the ameba's major food source.

Tissue or systemic amebicides

Act on the ameba in **tissues** (trophozoites form) e.g. The **intestinal wall** and/or other **extra-intestinal** tissues as **liver**, brain and lung. → they are **absorbed easily** from the intestine.

Used for treatment of systemic form of the disease (invasive amebiasis) e.g. intestinal wall infection or liver abscesses.

- **Metronidazole**/ **Tinidazole**
- **Emetine** / **dehydroemetine**
- **Chloroquine** (**liver** only)

We should start treatment with **Luminal amebicides** after finishing the course of tissue amebicides → bc the pt in this case will be **carrier** → we have to eradicate the organism completely!

* قبل ما نبدأ، افهموا هذي المايند ماب زين، بتختصر لكم الموضوع.. وتذكروا إنه في أغلب هذي الأدوية **الأطفال والحوامل** غير محبذ نستخدمها معهم.

Tissue or Systemic Amebicides

Drug: Metronidazole

Type: Tissue amoebicide. | **Organism:** Acts on trophozoites.

MOA

- **Inhibits DNA replication**. → It has a **nitro group** that receives electrons from ferredoxin (present in anaerobic parasites) in a redox reaction. The resultant compound bind both to proteins & DNA and is cytoctoxic. (**free radicals** that damage the trophozoite's DNA)
- **Does not eradicate cysts from intestine.** لأنه أول ما يوصل للأمعاء يصير له امتصاص بسرعة وما يمديه يشتغل على ال lumine

P.K

1) Given orally (PO) or IV. → Absorption is rapid and complete by PO (give a systemic effect)
2) Wide distribution to all tissues and body fluids (CSF, saliva, milk, vaginal fluids).
3) Plasma half life is (8 h)
4) Metabolized in liver (by CYP-450) by mixed function **oxidase** followed by **glucuronidation** (consider drug interactions).
5) Clearance is decreased in liver impairment.
6) Excreted in urine. → should be used with precaution w\ kidney & liver diseases.

Indications

1) **Drug of choice for treating invasive (tissue) amebic infections** (intestinal & extraintestinal amebiasis).

   → N.B. should be followed by **luminal amebicides** (mainly **diloxanide**).

Effective in all other protozoal infections, such as:

2) Giardiasis. (by *Giardia lamblia*) → given in low dose.
3) Trichomoniasis (*by Trichomonas vaginalis*) → metronidazole is the treatment of choice.
4) Broad spectrum of **anaerobic bacterial** infections. e.g.:
   - **Peptic ulcer** (*Helicobacter pylori*)
   - **Pseudo-membranous colitis** (*Clostridium difficile*). (the drug of choice)

ADRs

- GIT:
- Dry mouth → Oral Thrush (infection may result from the dryness of mouth) (Moniliasis, yeast infection).
- **Metallic taste.**
- Nausea, vomiting, diarrhea (NVD)
- Dysuria, **dark** urine.
- Neutropenia (low neutrophils) → Reversible
- **Disulfiram-like effect if taken with alcohol.**

- **CNS: Neurotoxicological effects:**
- Insomnia, dizziness
- **Peripheral neuropathy**, **paresthesia** → should **stop the treatment** w\ Metr.
- Encephalopathy, convulsion (IV infusion, rare)

C.I

1. Pregnancy and breast feeding women. (especially in the 1st trimester) 2. Alcohol intake
2. 3.**CNS diseases** 4. Severe renal disease 5. **Severe hepatic disease**.

Tissue or Systemic Amebicides

Drug	Metronidazole (cont.)
Drug – Alcohol Interaction	Disulfiram like-effect of metronidazole
	Combining metronidazole and alcohol causes nausea, vomiting, abdominal distress, flushing, headache, tachycardia, hyperventilation. Ethanol → (Alcohol dehydrogenase) → Acetaldehyde → (Aldehyde dehydrogenase) → Acetate
	1. CYP-450 Enzyme inhibitors (cimetidine, ketoconazole -all azole **anti-fungal** are inhibitors-) → **increase** duration of action of metronidazole. 2. CYP-450 Inducers (phenytoin and phenobarbitone -most of the antiepileptic drugs- & rifampicin)→ **decrease** duration of action of metronidazole. 3. Metronidazole **inhibits** CYP-450 (2C9 & 3A4) so→ **increases anticoagulant effect of** warfarin & Increases lithium toxicity. → → ليش ما أثر على الدرقز اللي قبل زي الفينيتوين؟ لأن تأثير الانهيشن حقه على سب كلاس غير الدرقز المذكورة سابقًا.. عشان كذا ماله تأثير عليهم، إنما تأثيره يكون على الدرقز اللي تنتمي لهذا السب كلاس..

Drug	Tinidazole
MOA	○ Tinidazole has similar activity to metronidazole but better potency.
Advantages	○ Has **longer duration of action** (12-14h) → يستخدم بمرات أقل في اليوم ○ A simpler dosing regimen → more potent. ○ A **better toxicity** profile than metronidazole.

Drug	Chloroquine
MOA	○ Anti-malarial drug.
Indications	○ Used in combination with metronidazole or dehydroemetinie for **amebic liver diseases** → why only liver? Bc they are concentrated in the liver.
ADRs	○ **Pruritus** is common. ○ Nausea, vomiting, abdominal pain, anorexia. ○ **Blurring of vision** → Remember from the neuropsychiatry block, they were depositing in the eye. ○ **Hemolysis** in G6PD deficient patients. → if I give it to pt w\ G6PD deficiency → It will cause hemolytic anemia!

Tissue or systemic amebicides

Drug	Emetine & Dehydroemetine
MOA	متى أستخدمهم؟ لما المريض ما ينفع معه metranidazole or Tinidazole ○ Both are effective against tissue **trophozites** of *E.histolytica* causing **irreversible block of protein synthesis**.
P.K	○ Emitine is an alkaloids derived from ipeca. While Dehydroemetine is a synthetic analog. ○ Have erratic oral absorption. ○ Given preferably **subcutaneously** but could be given by IM, **NEVER IV** → (bc of CVS toxicity) ○ Has long plasma half life about 5 days. ○ Metabolized & excreted slowly via kidney so they have a **cumulative effect**. ○ Should **not** be used for more than 10 days (usually 3-5 days) → bc their T1\2 is long & excreted slowly, therefore they will be accumulated if used fro a long time.
Indications	○ Amoebic liver abscess. ○ Intestinal wall infections. ○ **Severe forms of amebiasis** → Acute amoebic dysentery Dehydroemitine is **preferable** due to less toxicity (3-5 days). ○ Their use is limited to unusual circumstances in which severe amebiasis requires effective therapy and metronidazole cannot be used. This is because of their major toxicity effects.
ADRs	○ Dehydroemitine is less toxic than Emetine. ○ GIT: nausea, vomiting, diarrhea. ○ **Serious toxicities**: **cardiotoxicity**; hypotension, cardiac arrhythmias, heart failure. ○ → Because of major toxicity concerns they have been almost completely replaced by metronidazole.
C.I	○ The drug should not be used in: • Patients with cardiac or renal diseases • Young children. • In **pregnancy**.

Luminal amoebicides = w\ Asymptomatic

Drug	Diloxanide furoate	Iodoquinol
Mech. of action	○ Mechanism of action is unknown. ○ Direct amoebicidal action against luminal forms → Not active against **trophozoites** in intestinal wall or extra-intestinal tissue.	Remember Iodine when you see Iodoquinol! ○ Mechanism of action is unknown. ○ It is effective against organisms in the bowel lumen but **not** against trophozoites in the intestinal wall or extraintestinal tissues.
P.K	○ Ester of **Diloxanide** (active antiamebic substance) + furoic acid. ○ Given orally. ○ It splits in the intestine liberating diloxanide. ○ The **unabsorbed diloxanide** is the **amoebicidal** agent. ○ The absorbed portion is excreted in urine.	○ Given orally. ○ Poorly absorbed (remain in intestine to do its luminal amoebicide action) ○ Excreted in **feces**. → طبيعي لأنه ما راح يصير له امتصاص خارج الأمعاء
Indications	○ Drug of choice for **Asymptomatic** intestinal infection (**cyst passers**). ○ To **eradicate cysts** of *E. histolytica* **after** treatment of invasive disease with systemic amebicides.	○ Luminal amoebicide for **asymptomatic amebiasis**.
ADRs	○ Flatulence. ○ Nausea, vomiting, abdominal cramps. بما إنهم ما يصير لهم absorption برا GI، فأكيد الأعراض الجانبية بتكون خاصة بالGI	○ GIT: Nausea, vomiting, diarrhea. ○ Peripheral neuropathy: including **optic neuritis**. (w\ high dose) ○ Enlargement of the thyroid gland. ○ **Iodine sensitivity**. ○ Interference with thyroid function tests (increase protein-bound serum iodine, decrease in measured (^{131}I uptake) → When the pt use Iodoquinol, It will do similar effect of ^{131}I in the test → will give false measurements.
C.I	○ Pregnancy ○ Children (less than 2 years)	○ Patients with optic neuropathy, or thyroid disease. ○ **Discontinued** if it produces **persistent diarrhea** or signs of **iodine toxicity** → (dermatitis, urticarial, pruritus, fever)

Luminal amoebicides

Drug	Paromomycin Sulphate
MOA	○ Aminoglycoside antibiotic. 1. **Direct amebicidal** action (causes leakage by its action on cell membrane of parasite). 2. **Indirect killing of bacterial flora** essential for proliferation of pathogenic amoebae.
P.K	○ Given orally ○ Not significantly absorbed from GIT → Effective only against **luminal forms** of ameba. ○ Small amount absorbed is excreted unchanged in urine *(may accumulate with renal insufficiency)*. → Remember that aminoglycosides may cause nephrotoxicity!
Uses	○ Used in chronic amebiasis to eliminate **cysts** (in cysts passers).
ADRs	○ **GI** distress and diarrhea.
Precautionn	○ Severe renal disease & patients with GIT ulceration

Summary for treatment of amebiasis

Asymptomatic dysentery (cyst carriers)	Amebic colitis and dysentery, ameboma, and extra-intestinal disease	Hepatic abscess
Luminal amebicides: Diloxanide or iodoquinol or Paromomycin	Metronidazole or tinidazole followed by luminal amebicides.	Metronidazole or tinidazole or chloroquine or dehydroemetine

Bacillary dysentery

0:45 min

Treatment of Bacillary dysentery:

1- **Fluoroquinolones** such as **ciprofloxacin** or **ofloxacin.**

2- **Beta-lactams**: Ampicillin, amoxicillin,
Third generation cephalosporins (cefixime, ceftriaxone)

3- **Macrolides**: **Azithromycin**

4- **Cotrimoxazole** (trimethoprim-sulfamethoxazole) commonly used in **traveler's diarrhea**.

Antimicrobial therapy is typically administered for 5 days.

- **Resistance** to **ampicillin**, **amoxicillin** and **sulfonamides**, has been reported worldwide, and these agents are **not recommended as empirical therapy**.
- **Fluoroquinolones** are **first-line** treatment for ***shigellosis***.
- **Second line** therapy include **third generation cephalosporins**.

→ In case of pregnant women or children or breast feeding women → use cephalosporins! Not Fluoroquinolones!

Until the culture results come in dysentery caused by infection, give empiric therapy in → combination of anti-protozoal + antibiotic.

Bacillary dysentery treatment (cont.)

Fluoroquinolones →

FluoroquinoLONES attaches to your **BONES**

Drug	**Ciprofloxacin** or **ofloxacin**
MOA	○ Block bacterial DNA synthesis and growth (DNA gyrase & topoisomerases). ○ Active against a variety of gram-positive and gram-negative bacteria.
Uses	○ Bacterial diarrhea (caused by *shigella* (1st choice), *salmonella* and *E.coli*) ○ Urinary tract infections ○ Respiratory tract infections ○ Soft tissues, bones, and joint infections
ADRs	○ **Arthropathy** (damage of growing cartilage), GIT disorders (nausea, vomiting, diarrhea). ○ CNS disorders (headache, dizziness), CVS disorder (prolonged QT interval). ○ Phototoxicity, Liver toxicity.
C.I	○ Children, pregnancy & nursing mother. (absolute C.I) ○ Epilepsy & Arrhythmias. (not absolute C.I, used w\ precaution) ○ Should **not** be combined with **antacids**, divalent cations. → antacids containing aluminum or magnesium, or dietary supplements containing iron or zinc can interfere with the absorption.

3rd generation Cephalosporins

Cefixime (PO) or **Ceftriaxone** (IV)

MOA	○ Act by interfering with synthesis of **peptidoglycan**, a major structural component of bacterial cell wall.
Uses	○ In case of **children** or patient **allergic** to **sulfonamides** → **cephalosporins** or **azithromycin** may be used. (note that **sulfonamides** are also C.I w\ preg. & child) ○ They are safe and effective.

Quick summary

Maintain *fluid* intake (oral rehydration therapy or Intravenous fluid therapy).

Asymptomatic luminal amebiasis is treated by luminal amebicides (diloxanide, or iodoquinol or paromomycin).

Metronidazole is the mainstay of therapy for invasive amebiasis (followed by luminal amebicides to prevent relapse).

Chloroquine has also been used for patients with hepatic amebiasis.

Dehydroemetine is useful but not preferable due to CVS toxicity.

Ciprofloxacin is the drug of choice in bacillary dysentery.

In children and pregnancy, ceftriaxone or cefixime is the choice.

Summary for treatment of amebiasis

1- Systemic Amebicides

Metronidazole

MOA

Inhibits DNA replication

P.K

- Given orally or IV.
- Rapid absorption
- Wide distribution Plasma 1/2 life is 8h
- Metabolized in liver by mixed function oxidase followed by glucuronidation
- Excreted in urine.
- Clearance is decreased in liver impairment

ADRs

- GIT: Dry mouth, metallic taste ,nausea, vomiting, diarrhea
- Oral Thrush (Moniliasis, yeast infection).
- CNS: Neurotoxicological effect, insomnia, dizziness, peripheral neuropathy, paresthesia, encephalopathy, convulsion
- Dysuria, dark urine.
- Neutropenia
- Disulfiram-like effect if taken with alcohol

Indications

- Drug of choice for treating invasive amebic infections (should be followed by luminal amebicides)
- Giardiasis
- Trichomoniasis
- Broad spectrum of anaerobic bacterial infections (e.g. peptic ulcer)

#

- Pregnancy and breast feeding women.
- Alcohol intake
- CNS diseases
- Severe renal disease
- Severe hepatic disease

Drug Interaction

- Enzyme inhibitors (cimetidine, ketoconazole) → increase duration of action of metronidazole
- Inducers (phenytoin and phenobarbitone) → decrease duration of action of metronidazole
- Inhibits CYP-450 (2C9 & 3A4) so:
  - Increases anticoagulant effect of warfarin.
  - Increases lithium toxicity.

Tinidazole

Similar activity to metronidazole

Advantages:

- has longer duration of action (12-14h)
- a simpler dosing regimen
- a better toxicity profile than Metronidazole

2- Systemic Amebicides (cont.)

	Emetine/ dehydroemetine	Chloroquine
MOA	irreversible block of protein synthesis	Anti-malarial drug
P.K	• Have erratic oral absorption. • S.C /IM, NEVER I.V. • Has long plasma half life about 5 days. • Metabolized & excreted slowly via kidney • Should not be used for more than 10 days (3-5 days).	
ADRs	• Dehydroemetine is less toxic than emetine • GIT: nausea, vomiting, diarrhea. • Serious toxicities: **cardiotoxicity** • Hypotension, cardiac arrhythmias, heart failure	• Pruritus is common • Nausea, vomiting, abdominal pain, anorexia. • Blurring of vision. • Hemolysis in G6PD deficient patients
Indications	• Amoebic liver abscess. • Intestinal wall infections. • **Acute** amoebic dysentery (dehydroemetine is preferable due to less toxicity (3-5 days)).	Used in combination with metronidazole or dehydroemetine for amebic liver diseases
#	• Cardiac or renal disease • Young children • Pregnancy	

3- Luminal Amebicides

	Diloxanide furoate	Iodoquinol	Paromomycin Sulphate
MOA	Unknown	Unknown	Aminoglycoside antibiotic
P.K	• Given orally. • It splits in the intestine liberating diloxanide • The unabsorbed diloxanide is the amoebicidal agent • The absorbed portion is excreted in urine	• Is given orally • Poorly absorbed • Excreted in feces.	• Given orally • Not significantly absorbed from GIT • Has direct amebicidal action (causes leakage by its action on cell membrane of parasite). • Indirect killing of bacterial flora (food source for amoeba) • Small amount absorbed is excreted unchanged in urine
ADRs	• Flatulence • Nausea, vomiting, abdominal cramps	• GIT: Nausea, vomiting, diarrhea. • Peripheral neuropathy including optic neuritis • Enlargement of the thyroid gland. • Iodine sensitivity. • Interference with thyroid function tests (increase protein-bound serum iodine, decrease in measured **(131I uptake).**	Gastrointestinal distress and diarrhea
Indications	• Drug of choice for asymptomatic intestinal infection (cysts passers). • To eradicate cysts of E histolytica after treatment of invasive disease with systemic amebicides	Luminal amoebicide for asymptomatic amebiasis	Effective only against luminal forms of ameba Used in chronic amebiasis to eliminate cysts (in cysts passers).
#	• Pregnancy • Children (less than 2 years)	Precautions: • Optic neuropathy • Thyroid disease	Precautions: • Severe renal disease • Patients with GIT ulceration

Extra summary

TABLE 52–5 Treatment of amebiasis. Not all preparations are available in the USA.[1]

Clinical Setting	Drugs of Choice and Adult Dosage	Alternative Drugs and Adult Dosage
Asymptomatic intestinal infection	Luminal agent: Diloxanide furoate,[2] 500 mg 3 times daily for 10 days *or* Iodoquinol, 650 mg 3 times daily for 21 days *or* Paromomycin, 10 mg/kg 3 times daily for 7 days	
Mild to moderate intestinal infection	Metronidazole, 750 mg 3 times daily (or 500 mg IV every 6 hours) for 10 days *or* Tinidazole, 2 g daily for 3 days *plus* Luminal agent (see above)	Luminal agent (see above) *plus either* Tetracycline, 250 mg 3 times daily for 10 days *or* Erythromycin, 500 mg 4 times daily for 10 days
Severe intestinal infection	Metronidazole, 750 mg 3 times daily (or 500 mg IV every 6 hours) for 10 days *or* Tinidazole, 2 g daily for 3 days *plus* Luminal agent (see above)	Luminal agent (see above) *plus either* Tetracycline, 250 mg 3 times daily for 10 days *or* Dehydroemetine[2] or emetine,[2] 1 mg/kg SC or IM for 3–5 days
Hepatic abscess, ameboma, and other extraintestinal disease	Metronidazole, 750 mg 3 times daily (or 500 mg IV every 6 hours) for 10 days *or* Tinidazole, 2 g daily for 5 days *plus* Luminal agent (see above)	Dehydroemetine[2] or emetine,[2] 1 mg/kg SC or IM for 8–10 days, followed by (liver abscess only) chloroquine, 500 mg twice daily for 2 days, then 500 mg daily for 21 days *plus* Luminal agent (see above)

[1]Route is oral unless otherwise indicated. See text for additional details and cautions.

[2]Not available in the USA.

Drugs used in amoebiasis

Amoebiasis is caused by infection with *E. histolytica*, which causes dysentery and liver abscesses. The organism may be present in motile invasive form or as a cyst. The main drugs are:

- **Metronidazole** given orally (half-life 7 h). Active against the invasive form in gut and liver but not the cysts. Unwanted effects (rare); gastrointestinal disturbances and central nervous system symptoms. **Tinidazole** is similar.
- **Diloxanide** is given orally with no serious unwanted effects. It is active, while unabsorbed, against the non-invasive form in the gastrointestinal tract.

MCQs

1- Which of the following is the drug of choice for treating tissue amebiasis?
A- Iodoquinol
B- Ciprofloxacin
C- Metronidazole
D- Cotrimoxazole

2- Choose the most effective drug for mild intestinal amoebiasis and asymptomatic cyst passers:
A- Metronidazole
B- Emetine
C- Quiniodochlor
D- Diloxanide furoate

3- Metronidazole should be taken carefully in a patient taking which of the following medication?
A- Proton pump inhibitors
B- Antiepileptic (phenytoin)
C- Anticoagulant (warfarrin)
D- B & C

4- The anti amoebic agent implicated in causing subacute myelo-optic neuropathy (SMON) is
A- Diloxanide furoate
B- Iodochlorohydroxyquin
C- Emetine
D- Metronidazole

5- Metronidazole is least likely to be effective in the treatment of:
A- Amebiasis
B- Psudomembranous colitis
C- Pneumocystosis
D- Trichomoniasis

6- Which one is associated with Ciprofloxacin side affects?
A- Optic nuertitis
B- Enlargment of thyroid gland
C- Arthropathy
D- Iodine sensitivity

Answers: 1-C 2-D 3-D 4-B 5-C 6-C

MCQs

7- Which stage is considered the invasive stage?
A- Trophozoites
B- Cysts

8- Patients treated with the following drug should be cautioned not to consume alcoholic beverages:
A- Mebendazole
B- Metronidazole
C- Methimazole
D- Metamizol

9- Choose the correct statement about metronidazole:
A- It is a first line drug for amoebic dysenteryas well as amoebic liver abscess.
B- It affords the most rapid symptom relief in amoebic dysentery.
C- It is the most effective drug in eradicating amoebic cysts from the colon.
D- All of the above

10- In addition to amoebiasis, metronidazole is used for:
A- Roundworminfestation
B- Hookworminfestation
C- Kala-azar
D- Giardiasis

11- Metronidazole is selectively active against anaerobic organisms because:
A- Aerobes have an active transport mecha- nism to pump it out of their cell.
B- Only anaerobes reduce it to generate the reactive nitro radical.
C- It is rapidly inactivated in the presence of oxygen.
D- It binds to DNA of anaerobes with high affinity.

12- Emetine is now used only as a reserve drug for amoebiasis because:
A- It is less effective than metronidazole
B- It produces a slower response than metroniDazole
C- It has cardiotoxic potential.
D- It is not effective in extraintestinal amoebiasis.

Answers: 7-A 8-B 9-A 10-D 11-B 12-C

Thank you for checking our team!

Pharmacology 435

@ pharmacology435

Sources:

1. 435's slides.
2. Pharmacology (Lippincotts Illustrated Reviews Series), chapter 36, 5th edition.
3. Basic & Clinical Pharmacology by Katzung, chapter 52,12th edition.
4. Rang & Dale's pharmacology, chapter 53, 7th edition.
5. Pharmacology recall, by Ramachandran. Chapter 48, 2nd edition.