**CYSTICERCOSIS**

Life cycle of *Taenia solium* (pork tapeworm):

- **Egg** in feces

**INTERMEDIATE HOST** (pigs, humans): Egg in mouth → embryos hatch in duodenum → lymphatics or systemic circulation → embryos mature into larvae in tissues

**DEFINITIVE HOST** (only humans): Larvae in mouth → larvae mature into adult in small bowel → egg in feces

*T. solium* can infect man in two different ways:

A. **Adult worm** in human as definitive host: eating undercooked infested (measly) pork → encysted larvae are released in small bowel → larvae mature within intestine into adult over 2 months → taeniasis (intestinal tapeworm); scolex (head) of segmented adult worm attaches by means of four suckers and two rows of hooklets to wall of small intestine where worm absorbs food directly through its cuticle:
- proglottids (mature segments, each containing reproductive organs) produce eggs which are liberally excreted along with gravid proglottids in feces.
- nervous system is not affected!

B. **Larva** in human as intermediate host: ingesting eggs*

- **a)** food (usually vegetables) or water contaminated with human feces containing eggs or gravid proglottids (this is means whereby pigs acquire disease)
  - b) fecal-oral autoinoculation in individual harboring adult tapeworm
  - c) autoinfection by reverse peristalsis of gravid proglottids from intestine into stomach during vomiting (unproven theoretical possibility) → swallowing regurgitated eggs.
- in duodenum shelf of ova dissolves → hatched embryos (oncospheres) burrow through small bowel wall → lymphatics or systemic circulation → in tissues embryos develop cyst wall in 2 months (immature cyst) which matures in 4 months to larva → cysticercosis:
  - a) neurocysticercosis (see below >>)
  - b) eye (immunologically privileged, like brain)
  - c) skeletal muscle
  - d) subcutaneous tissue (palpable nodules)
  - e) heart
- larval cysts are usually rapidly eliminated by immune system; many larvae die naturally within 5-7 yrs or with cysticidal therapy → inflammatory reaction → collapse of cyst (granular nodular stage), sometimes calcify (nodular calcified stage); in pigs, larva lie dormant in muscle, "waiting" to be eaten.

Scolex (head) of *Taenia solium*:

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**Sclolex** (head) of *Taenia solium*

**NEUROCYSTICERCOSIS** (NCC)

**PATHOPHYSIOLOGY, CLINICAL FEATURES**

**ESCIARAE STAGES**

**DIAGNOSIS**

**TREATMENT**

Anthelmintic drugs

Follow-up

**ECHINOCOCCOSIS**

E. granulosus (EG) → cystic echinococcosis

E. multilocularis / alveolaris (EM/EA) → alveolar echinococcosis

**CONTACTS**

Follow up

**TAGES**

- Amebic abscess
- Primary amebic meningoencephalitis (PAM)
- E. multilocularis / alveolaris (EM/EA)
- E. granulosus (EG)

**SIGNS/ SYMPTOMS**

- T. solium: fasciolopsiasis (intestinal fasciolopsiasis)

**TREATED:**

- Surgery
- Antiepileptics
- Steroids
- Antihelmintic drugs

**COMPPLICATIONS**

- focal inflammatory reaction
- collapse of cyst
- inflammatory reaction
- collapse of cyst (granular nodular stage), sometimes calcify (nodular calcified stage); in pigs, larva lie dormant in muscle, "waiting" to be eaten.

**INFECTIOUSNESS**

- prepatent period: 6-8 weeks

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**DEFINITION:**

Cysticercosis: Cystic larval stage of *T. solium* in the central nervous system (CNS) of humans.

- Adult *T. solium* worms live in the human intestine, where they release thousands of eggs that pass into the feces and are ingested by pigs (intermediate host).
- Once in the pig's small intestines, the eggs hatch and release tiny larvae called scolexes that attach to the wall of the gut. The scolex then releases four suckers and two rows of hooklets.
- The scolex then migrates to other organs or tissues, such as muscles or the nervous system, where it develops into a cysticercus (larval cyst).

**CLINICAL FEATURES:**

- Neurocysticercosis (NCC): Cysticerci in the CNS can cause a range of symptoms, including seizures, headaches, and focal neurological deficits.
- Echinococcosis: Cystic echinococcosis (CE) is a parasitic infection caused by the larval form of the tapeworm *Echinococcus granulosus*.
  - Alveolar echinococcosis (AE) is a less common form of the disease caused by *Echinococcus multilocularis*. It is characterized by the development of multiple, often large, cysts in the liver or other organs.

**DIAGNOSIS:**

Cysticercosis is diagnosed through imaging studies such as CT or MRI scans, which can detect the characteristic cystic lesions in the brain or other organs.

**TREATMENT:**

Treatment options for cysticercosis include:

- Surgery: For large cystic lesions, surgery may be necessary to remove the cysts.
- Antiparasitic medications: These medications can help kill the parasites and reduce symptoms.
- Antiepileptic medications: For neurocysticercosis, antiepileptic medications may be necessary to control seizures.

**COMPLICATIONS:**

- Focal neurological deficits
- Severe headache
- Cerebral edema
- Intracranial hemorrhage
- Seizures

**INFECTIOUSNESS:**

- Cysticercosis is not contagious from person to person.

**REFERENCES:**

NEUROCYSTICERCOSIS (NCC)

Cysticercosis → see p. 289 (3-4)

- Intracranial encystment of larva of Taenia solium
- Most common parasitic CNS infection.
- Epidemic in South and Central America – seroprevalence up to 11% (esp. in rural areas); 18% in Madagascar.
- In USA: government inspection identifies 10 cases of cysticercosis in 80 million hogs that are slaughtered each year; important sources: 1) immigrant Hispanics 2) contact with infected immigrant food preparers (e.g. neucysticercosis has also been reported in Orthodox Jewish people with no travel outside the USA or exposure to pork products).

PATHOPHYSIOLOGY, CLINICAL FEATURES

- Larva has marked predilection for neural tissue - brain is involved in 60-90% of cases of cysticercosis; spinal cord and peripheral nerve involvement is rare.
- Extraneural cysticercosis may occur in skeletal musculature, conjunctiva or retina, but is rarely present in persons with NCC.

Pathologic stages

Host tolerates worm as long as embryo is alive (vesicular stage)
↓ 2-6 years after infection
Worm dies
Disintegration of parasite triggers vigorous inflammatory tissue reaction → severe symptoms; cyst wall and fluid become infiltrated by mononuclear cells (collodion stage).
↓ Fibrosis with collapse of cyst cavity (granular-nodular stage)
Parasite decays into eosinophilic desiccated material.
↓ Dyssympathic calcified nodule (calcific stage)

N.B: Intense from ingestion of eggs to symptomatic* neucysticercosis: 2-6 years (83% cases show symptoms within 7 years of exposure)

*Release of T. solium antigens from dying parasite, mass effects → acute, chronic or resolving clinical picture

Two types of cysts in brain:

1. Cysticercus celluloseus: regular thin-walled cyst (3-20 mm) in parenchyma or narrow subarachnoid spaces; contains larva with scolex, usually static - produces only mild inflammation during active phase
2. Cysticercus racemous: large (4-12 cm) cysts without larva, grows actively producing grape-like clusters in basal subarachnoid spaces and produces intense inflammation; usually degenerate in 2-5 years → capsule thickens and clear cyst contents are replace by whitish gel → calcium deposition with concomitant cyst shrinkage.

Location of cysts:

1. Parenchymal (30-63%) → seizures (most common cause of acquired epilepsy in developing countries), other focal deficits (paresis, visual loss); cysticercotic encephalitis (most often seen in young girls and is characterized by numerous intraparenchymal cysts and diffuse cerebral edema) produced by active phase
2. Meningeal (27-56%) a) Adherent or free-floating C. celluloseus cysts in dorsolateral subarachnoid spaces causing minimal symptoms
b) Expanding C. racemous cysts in basal subarachnoid space → chronic basal meningitis (kaytomycystercerosis) with meningeal signs; fibrosis can cause cranial neuropathies and strokes, obstruct foramina of Luschka and Magendie (→ obstructive hydrocephalus), extremely high mortality.
3. Ventricular (12-18%) via choroid plexus; 50% in 4th ventricle; pedunculated or free floating cysts can cause obstructive hydrocephalus with intermittent intracranial hypertension (brain syndrome); MRI shows adjacent ependymal enhancement (ependymitis).
4. Spinal cord (most often – cervical/thoracic) → radiculopathy or myelopathy.

N.B: Increased ICP may be due to hydrocephalus or mass effect of giant cysts.
**ESCOBAR STAGES**

<table>
<thead>
<tr>
<th>Escobar stage</th>
<th>Pathology</th>
<th>Imaging findings</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>VESICULAR</strong></td>
<td>Larva alive, invaginated, cyst fluid translucent</td>
<td>Smooth, thin-walled cyst that is isodense/isointense to CSF; may see small nodule. Edema: 0. Enhancement: 0</td>
<td>Asymptomatic, treatment with anthelmintic: effective</td>
</tr>
<tr>
<td><strong>COLLOIDAL VESICULAR</strong></td>
<td>Parasite dying; degenerative changes present; cyst fluid jelly-like and white; fibrous capsule develops with perilesional edema</td>
<td>Cyst with thick capsule; fluid signal hyperdense/hyperintense (on T1) to CSF. No DWI restriction. MRS shows succinate, acetate, and lactate peaks. Edema: 3+. Enhancement: 3+ ring-enhancing capsule.</td>
<td>Usually asymptomatic; treatment with anthelmintic: effective</td>
</tr>
<tr>
<td><strong>GRANULAR NODULAR</strong></td>
<td>Cyst smaller; cyst fluid with coarse granules and pericystic gliosis</td>
<td>Thickened, retracting cyst wall.</td>
<td>Symptomatic; treatment with anthelmintic: probably not necessary; treat seizures with anticonvulsant</td>
</tr>
<tr>
<td><strong>NODULAR CALCIFIED</strong></td>
<td>Complete mineralization of cyst fluid; no host immune response.</td>
<td>Small calcified nodule (bright dots on CT, black dots on MR). HEME sequence may show “blooming”</td>
<td>Symptomatic; treatment with anthelmintic: not indicated; treat seizures with anticonvulsant</td>
</tr>
</tbody>
</table>

**DIAGNOSIS**

CT, MRI

1) **non-enhancing areas of edema** → **homogeneous contrast-enhancing lesions** → **SYMPTOMATIC STAGE**: low density nonenhancing cyst(s) with eccentric punctate high density (scolex = tapeworm head); little inflammatory response (edema) occurs as long as larva is alive → low density ring-enhancing cysts (intermediate stage) with inflammatory edema → complete resolution or oval calcifications without edema (dead parasites) →.

2) **Hydrocephalus** is common; intraventricular cysts may be isointense with CSF (H. CBSS MRI or contrast CT ventriculography)

- MRI has higher sensitivity.
- If eccentric scolex is seen within cyst, NCC may be diagnosed confidently!
- Presence of multifocal cigar or rice-like calcifications in skeletal muscles is suggestive of cysticercosis, particularly in patients from endemic areas.

Nonenhanced (left) and enhanced (right) CT—peripheral meningeal enhancement around cysts in suprasellar and right sylvian cisterns.
Enhanced CT - live cyst with minimally enhancing wall and eccentric hyperattenuating scolex.

Nonenhanced CT - multiple calcified lesions of inactive parenchymal neurocysticercosis.

Nonenhanced (left) and enhanced (right) CT - multiple ring-enhancing lesions with perifocal edema.
Nonenhanced (left) and enhanced (right) CT - enhancing disk lesion with perifocal edema (granulomatous stage - arrow), plus, single ring lesions (colloid stage):

CT - numerous parenchymal lesions:

Intraventricular neurocysticercosis: CT - marked dilatation of right lateral ventricle. Contrast-enhanced ventriculogram - fourth ventricular cyst as a filling defect:
Parasitic Nervous System Infections

MRI - multiple, large, cysts in basal cisternal spaces:

T1, T2, and FLAIR MRIs show cyst with scolex (arrow):

Degenerating cyst with hypointense wall and surrounding edema:

Multiple cysticerci:
Cystic mass in frontal horn of the lateral ventricle extending into foramen of Monro producing enlarged ventricles; enhancing component within cyst which is calcified on CT is scolex.

Ocular involvement:
Enhanced CT scan of orbit shows:
calcified lesion in subretinal space of right globe.
granuloma in right lateral rectus muscle.

Serology on serum or CSF: enzyme-linked immunoelectrotransfer blot (ELIBT)* against glycoprotein antigens (100% specific and 70-98% sensitive in asymptomatic stages; but negative in 80% cases with solitary lesion or in calcified stages)
*effectively superseded ELISA (titer is significant at 1:64 in serum, and 1:8 in CSF)
• test of choice is serum ELIBT
Albendazole

Praziquantel

sometimes “sand” can be seen in dependent portion of cyst. “daughter” cysts); Two species of Echinococcus tapeworms:

- Hydatid disease
- Second most common parasitic CNS infection. Echinococcus granulosus (EG) or cysticercosis (medical history and serology); if suggestive of cysticercosis → CT/MRI

NCC has lesions that are in different stages - the most active stage of infection should determine if treatment is necessary!

Some cases of NCC have lesions that are in different stages - the most active stage of infection should determine if treatment is necessary!

- CT
- MRI
- Biopsy
determine if treatment is necessary

N.B. any cysticercidal drug may cause irreversible damage when used to treat ocular or spinal cysts, even with corticosteroid use

N.B. any cysticercidal drug may cause irreversible damage when used to treat ocular or spinal cysts, even with corticosteroid use

- if cyst is calcified or ring-enhancing, treatment with anthelmintics is probably not necessary.
- start after control of intracranial hypertension (usually after 3 doses of steroids).

Praziquantel - also cysticidal

1. ALBENDAZOLE – cysticidal agent of choice (more parasiticidal and fewer side effects than praziquantel), 400 mg q 24h or 15 mg/kg/day divided in 2-3 doses, taken with fatty meal to enhance absorption (same dose for pediatrics) for 3 months (can be stopped sooner if imaging shows resolution)

2. PRAZIQUANTEL – also cysticidal
   a) 50 mg/kg/d divided in 3 doses (same dose for pediatrics) for 15 days (doses of 100 mg/kg/d have been recommended because steroids reduce serum concentration by 50%).
   b) 10-100 mg/kg/d x 3-21 days
   c) higher dose single day regimen: 25-30 mg/kg q 2 hrs x 3 doses
   d) for intestinal infestation: single oral dose of 5-10 mg/kg

3. NICLOSAMIDE (Niclozid® and others) given orally for adult tapeworms in GI tract: 1 gm (2 tabs) chewed PO, repeated in 1 hour (total 2 gm).

- CT/MRI every 6 months until lesions disappear
- MRI every 6 months until lesions disappear
- if suggestive of cysticercosis → CT/MRI

- if cyst is calcified or ring-enhancing, treatment with anthelmintics is probably not necessary.
- start after control of intracranial hypertension (usually after 3 doses of steroids).

PERIODIC SYNCHRONOUS

Praziquantel for 3 days have been recommended because steroids reduce serum concentration by 50%.

- CT/MRI every 6 months until lesions disappear
- MRI every 6 months until lesions disappear
- if suggestive of cysticercosis → CT/MRI

- if cyst is calcified or ring-enhancing, treatment with anthelmintics is probably not necessary.
- start after control of intracranial hypertension (usually after 3 doses of steroids).

Praziquantel for 3 days have been recommended because steroids reduce serum concentration by 50%.

- CT/MRI every 6 months until lesions disappear
- MRI every 6 months until lesions disappear
- if suggestive of cysticercosis → CT/MRI

Hydatid disease or hydatid cyst (HC) – single large thin-walled cyst (but maybe multilocular with “daughter” cysts); no calcification, no edema, no enhancement, fluid isodense / isointense to CSF (sometimes “sund” can be seen in dependent portion of cyst).
**E. MULTILECULARIS / ALVEOLARIS (EM/EA)**

- Alveolar echinococcosis – multiple irregular cysts that are not sharply demarcated and enhance in ring-like/ nodular/ cauliflower patterns.

**AMEBIASIS**

**PRIMARY AMEBIC MENINGOENCEPHALITIS (PAM)**

- Acute, rapidly progressing, necrotizing, hemorrhagic meningoencephalitis and angiitis with focal lesions – numerous trophozoites are present, but no cysts are seen because of disease acuity.
  - *N. fowleri* in immunocompetent young adults swimming in warm fresh water during the summer.
  - *N. fowleri* invades the olfactory mucosa and enters the brain along the olfactory nerves.
  - Fatal within 48-72 hours.
GRANULOMATOUS AMERIC ENCEPHALITIS (GAE)
- granulomatous inflammation with multinucleated giant cells, trophozoites, and cysts.
- subacute to chronic condition.
- caused by one of six Acanthamoeba species or Balamuthia mandrillaris.
- no seasonal predilection.
- associated with immunodeficiency (and chronic debilitating conditions.)
AMEBIC ABSCESS
- pus with trophozoites at the edge of the lesion.
- caused by E. histolytica.
- most patients have intestinal or liver infection.
- not related to immunodeficiency.

BIBLIOGRAPHY for ch. “Infections of Nervous System” -> follow this LINK >>