

Leptospirosis :- CA: leptospira interrogans

aka **Bell's Disease** :-

Pathogenesis :-

- 1) Mode of transmission :- Indirect contact with water, swell soil & wet surfaces contaminated with animal urine or
- 2) Direct contact :- urine & products of excretion of placenta of infected animals.
- 3) Source :- Rats, dogs, cattle & pigs.
- 4) Seasonality :- Rainy
- 5) Risk factors :- Lower socioeconomic status, urban & rural slum areas, Rainfall & floods

PATHOGENESIS :-

1. Septhemic phase :- After entering through wound or abraded skin → intravascular spill hemolysis & dissemination into brain, liver, lung, heart & kidney.
2. Vascular damage :- peritum & invasion of tissue is due to active metastasis & release of hydrolytic enzymes.

2. Immune phase :- As Abs develop, spirochetes disappear from blood.
- Rural Copenhagen :- Bacilli + provisional rural humans brachioradial & erected in urine

CLF - IP: 10 (1-30 days)

- 1) Mild anicteric febrile illness :- 90-1% ; biphasic, flu like illness
- 2) Mild's ds :- (Hepato renal hemorrhagic syndrome) Severe form of icteric illness ↓ 10%

Lab Diagnosis :-

- CSF & blood (in first 10 days)
- Urine (10-30 days of infection)

Microscopy :-

Wet-film :- highly motile, spirally & helical movements
Staining :- Silver impregnation

Culture & Isolation :-

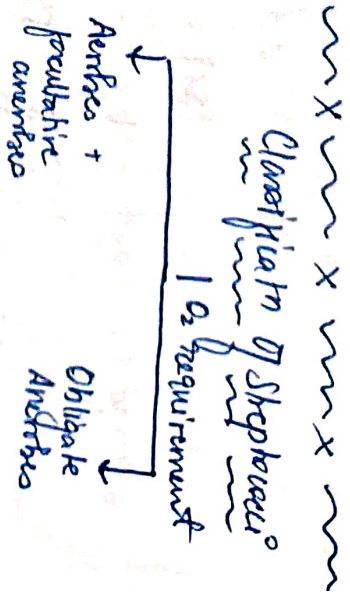
Temp, 4-6 weeks
Media :- EMJH liquid medium, Korthoff's, Flethel's sensitized

Serology :-

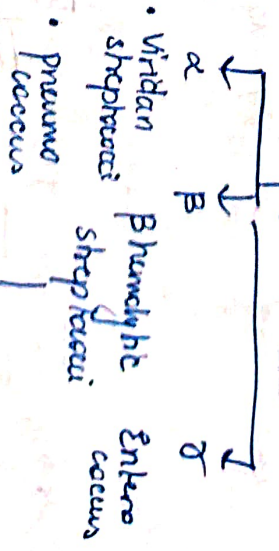
1. ELISA
2. Lepto dipstick assay
3. Immunofluorescent agglutination test (ICAT)

SEROVAR SPECIFIC :-
Microscopic Agglutination Test (MAT)
 ↳ gold standard

Rt's Oral Polyvalent (Group B)
 - 1 week



Hemolytic



20 Serogroups

Group A :- Shep. Pyrogenes

> 150m serotypes
 > 200 emm genoty,

Helicobacter pylori :-

curved, gram negative rod

Pathogenesis :-

Methyl - highly methylated
 Acid fastness - urease enzyme catalyze
 urea → ammonia → buffers the
 gastric acid.

adhesins & vasculins

Adhesins: bind group of binding cells.

Resistance to oxidative stress :-

Molecular mimicry :- LPS of H. pylori

is identical to Lewis blood group

Ag expressed on gastric parietal
 cell → immune tolerance

Involvement of auto Ab -
 contributing to the development
 of chronic gastritis

Alteration in gastric mucus :- LPS inhibits
 glycosylam & sulpham of gastric mucus

Lab Diagnosis

Invasive

Endoscopy guided
 multiple biopsies
 can be taken
 from gastric
 mucosa

HP: MARTIN

STARRY

SILVER

staining

Gram staining -
 curved, ovals
 spiral shaped
 morphology

Culture :-

SKIRROWS
 MEDIA &

CHODLA TE
 ANAR

Biopsy urease
 test

Non-invasive tests

1) Urea breath test
 (best, consistent,
 accurate, sensitive
 guide & simple)

2) Fecal Antigen
 Assay

3) Ab detectm
 by ELISA

Rx: DBTM quadruple
 therapy

Dmproazole

Bismuth subnitrate
 metronidazole
 Tetracycline

OCM or eCA :-
 Omproazole

Clarithromycin
 Metronidazole

Diff. b/w Amoebic & Bacillary Dysentery

	Amoebic	Bacillary
<u>Patho:</u>		
<u>Ulcer</u>	Deep	Shallow
<u>Margin</u>	Ragged & undermined	uniform
<u>Intensifying mucus</u>	normal	Inflamed
<u>cellular response</u>	mononuclear	polymorphonuclear
<u>Macro:</u>		
<u>No. of m/m:</u>	6-10/day	> 10/day
<u>Amount</u>	copious	small
<u>Color</u>	dark red	bright red
<u>Odor</u>	offensive	odorless
<u>Renn</u>	→ Acidic	Alkaline
<u>Consist</u>	Not adherent to container	Adherent to container
<u>Mix:</u>		
<u>RBC</u>	clumps	discrete
<u>pus</u>	few	numerous
<u>mucus</u>	few	numerous
<u>leucine</u>	⊕	⊖
<u>Cholesterol crystals</u>	⊕	⊖
<u>Pyrenitic body</u>	⊕	⊖
<u>Org.</u>	<i>E. histolytica</i>	Shigella

Lab Diagnosis of E. histolytica:

Specimen: Stool - 3 stool ^{alternate days} over 10 days because amoebae are shed intermittently

Stool microscopy:

- 1) Stool concentration - formalin ether sedimentation method
- 2) Staining - trichrome stain or hematoxylin stain

3) Culture methods:
Polyvalent culture methods

↓
Bacterial supplements such as NIH media & Bauch & Arbuzhar eg serum media

Axenic culture media:
(lacks bacterial supplement) eg - Diamond's medium

4.) Stool Antigen Detecto (Coproantigen)
⇒ ELISA detecting 170kDa lectin Ag

→ ICT: Triage parasite panel:
Giardia lamblia
E. histolytica
Cryptosporidium parvum

5.) Molecular Diagnosis:

- Nested multiplex PCR
- Real-time PCR
- Pab fix film assay

Diarrhoeagenic E. coli: 6

1. Enteropathogenic E. coli → infectious diarrhoea
 Poxen - to person spread is seen
 Non-toxicogenic & non-invasive

Mech: Adhesion to intestinal mucosa mediated by plasmid coded fimbriae forming Pili, which form cuplike projections called pedicels.

A/E lesion :- disruption of epithelium
 ↑ secretion & watery diarrhoea

2. Enterotoxigenic E. coli → traveler's diarrhoea
 ↳ toxicogenic but not invasive ⇒ Ne watery diarrhoea
 Pathogenesis of ETEC:-

- Attachment to intestinal mucosa :- CFA
 ↓
 colonisation factor Ag
- Toxin production - heat labile toxin (↑ cAMP)
 heat stable (↑ cAMP)
- 3. Enteroinvasive E. coli (EIEC)
 ↳ not toxicogenic but invasive.

Invasion mediated by :- Virulence marker antigen (VMA)
 Dysentery
 ⇒ Non-toxic & non-lactose fermenters

4) Enterohemorrhagic E. coli (EHEC)
 Transmission:- contaminated food
 Low infectious dose < 10² bacteria

Pathogenesis:- Verocytotoxin or Shiga toxin → inhibiting the protein synthesis by inhibiting 60S ribosome

Manifestations:-
 1) hemorrhagic colitis = gross bloody diarrhea, abdominal pain, endotoxaemia but no fever.

2) hemorrhagic uremic syndrome :- injury to small vessels of kidney & toxin ↓ bloody diarrhea, thrombocytopenia renal failure & encephalopathy but no fever

Diagnosis:- Sorbitol MacConkey Agar & Rasthson agar
 Toxin detection :- demonstration of cytotoxicity in Vero cell lines (gold standard method)

5-7 Enterococci aggregative pathogens :- Enterococci mediated by aggregative adhesion fimbriae + EAST toxin

6) Differently adherent E. coli Ability to adhere to HEp2 cells (diffuser)

Halophilic Vibrios

- Cannot grow in the absence of salt.
- tolerate and grow in higher salt concentration upto 7-10%.
- Examples :- V. parahaemolyticus
V. alginolyticus
V. vulnificus

ROTAVIRUS DIARRHEA :-

Virus causing gastroenteritis :-
RACANS

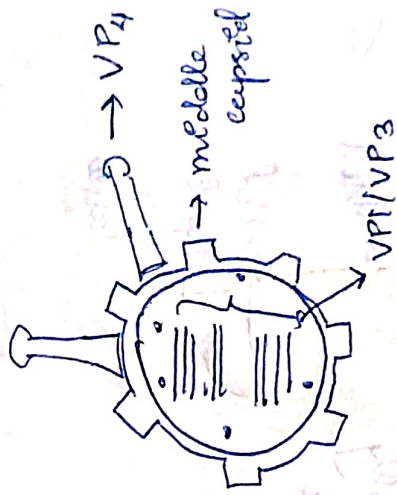
2- ROTAVIRUS - live attenuated
 (IP(2) strain) ; OR3, OR4, OR9
 2 doses + 1st at 6 weeks
 and - 4 weeks later

★ Lab Diagnosis

- 1) Direct detection of viruses - feces collected early in the illness - ideal specimen

• Immunoelectron microscopy :-

Rotaviruses have a sharp triple shelled capsid: look like spots grouped around the hub of a wheel



• Detect viral Ag :-
 ELISA & LAM.

• RT-PCR

★ VACCINES :-

- 1) ROTAVAC :-

⇒ live attenuated Rotavirus 116E (G9P[11] strain)
 Manufactured by - Bharat Biotech

- 3 doses (Schedule) :- orally
- 6, 10, 14 weeks follow with DPT 2PN

Schedule - varying availability

Morphology

Size : 60-80nm, icosahedral symmetry
triple layered wheel shaped capsid
 c/s RNA



• Protein :- 8m structural viral proteins

(V1 - V7 except V5) & 8m non-structural proteins (NSP1-6)

VP6 - group specific

Pathogenesis :-

Transmission :- feco-oral route

• Destroy enterocytes of small intestine & multiply in the cytoplasm of enterocytes & damage their transport mechanism

• NSP-4 (enterotoxin) induces secretion by altering epithelial cell function & permeability

Enterobius vermicularis | Pin worm :-

large intestinal parasite (nematode)

Pathogenesis & CLF :-

PERIANAL

Most common :- PERIANAL PRURITIS - gets worse at night due to nocturnal migration of female worms.

Reported scratching \rightarrow contaminated fingers

autoinfection

abdominal pain & weight loss in young infants.

LAB DIAGNOSIS :-

Microscopy of perianal swabs : detects characteristic eggs

1) Cellophane tape is applied to anal tape is mounted with a drop of saline.

2) NIH swabs :-

glass rod attached to cellophane tape by a rubber band. Cellophane part of glass rod is rolled over the perineal & perianal skin to collect sample.

No. of specimens :- 4-6 consecutive tapes on female worms migrate intermittently.

Timing :- chance of egg depositing

late evening : early morning

Eggs :-

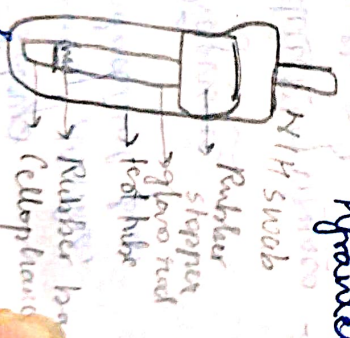
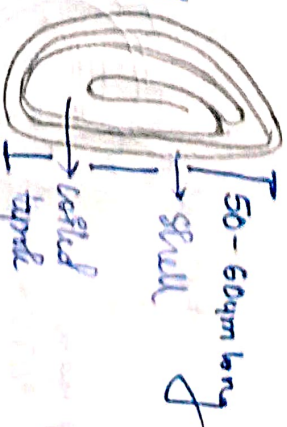
Parasitogram, 50-60 um long embryonated egg when freshly passed contains a tadpole shaped larva inside.

non-bite stained

colorless in saline mount

floats in saturated salt solution

Re :- Mubarekzade Alhendajde Riyadh R. Imwate



Complications : Rarely, pinworms migrate to FAT, vulva vaginitis, pelvic or perianal granulomas

HYATID DISEASE aka ECHINOCOCCOSIS CYSTIC

Course of Agent: Echinococcus granulosus (dog tapeworm)

Pathogenic forms: larvae - HYATID CYST - pseudocystic lesion in liver & other viscera of man.

Infective form: Eggs - embryo with 6 hooklets disarmed by an embryophore.

Host: definitive - dogs
intermediate - sheep
Man - accidental intermediate host

Pathogenesis:

In the duodenum, embryo is released penetrates foetal wall → PERITRIAD liver

⇒ hyatid cyst - fluid filled bladder like cyst.

HYATID CYSTS - 5-8cm

Cyst wall → outer pericyst (host-derived)

middle ectocyst
inner endocyst

Broad capsule: inner side of endocyst gives rise to broad capsule which contains a no. of protoscolices.

Hyatid fluid - clear, pale, yellow fluid, antigenic, toxic & anaphylactic

CF: Hyem occurs in childhood but manifests in adult life.

Cyst 5-10 cm
5-10 cm
Kidney, muscle, spleen, soft tissue, brain, bone

Symptoms: 1) Perium cyst - palpable abdominal mass, hepatomegaly, abdominal tenderness, post-HITN & ascites.

2) Obstruction - Choleliths, dyspnea

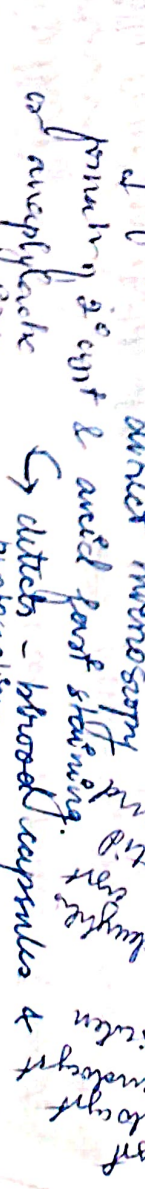
3) Pyogenic abscess

4) Anaphylactic rxn.

laboratory Diagnosis: 1) Hyatid fluid microscopy: fluid aspirated from a surgically removed cyst & subjected to direct microscopy

2) Rupture of cyst

primary cyst & anaphylactic reaction
direct parasitology
detailed - broad capsules & protoscolices



CASONI TEST

Imaging method: USG, CT, MRI

2) DNA (dot)

3) Antibody detection - ELISA

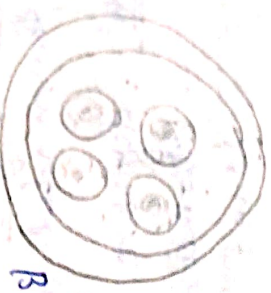
immunological filtration assay

neuron test

Entamoeba histolytica cysts:-

Uninucleated cysts

- Internal structure of the cyst is appreciated by H&E stain.
- Cyst appears round, 12-15um in size containing (1-4 nuclei)
- Both mature cysts (contain 4 nuclei) & immature cysts (contains 1 or 2 nuclei) are found in stool.
- Cysts form of uninucleated cyst contains few characteristic bodies and a large glycogen mass.



2) In size: 0.1mg LL

Factors regulating toxin production:-

- 1) Those coded: DT is coded by bacteriophage called B conjugophages - toxin gene

Diphtheria

Highly infectious diphtheroids

CA: Corynebacterium diphtheriae

Gram positive, non-spore forming, non capsulated, non-motile bacilli \Rightarrow club shaped.

1.) Chain like or cuneiform arrangement \Rightarrow snapping division.

2.) Metachromatic granules.

\downarrow
Pseudomonas phages (strain without, Neisser, Borden)

Virulence factors Diphtheria toxin



A \downarrow ADP ribosylase

B \downarrow EF-2 inhibitor

EF-2 \downarrow Inhibiting heart function

binds to host cells in entry of A

Pathogenesis & CLF:-

Diphtheria is toxin mediated

CLF. Diphtheria is focal

Diphtheria - diphtheria toxin

Exfoliation response

Warts of epithelium & erudate formation \Rightarrow Pseudo membrane coat

Surround by neutrophils, RBCs & bacteria

RBCs & bacteria

Bull's neck appearance:-

Wartlike heartless swelling and neck edema.

Patients with bull neck, thick speech & stridor

Enterovirus Diphtheria - peripheral out wheal-like lesion.

Systemic Complications

1) Pericardial involvement

Pericardial neuropathy
Cellulose paralysis

2) Myocarditis, arrhythmias