cocci to rods with short coccobacillary forms dominating

↑ → -ve, aerobic, non-motile

* obligate parasite, intracellular. ← **Brucella** → Trypticase soy agar or blood culture media
* grow in air except for B. abortus which requires 5-10% CO_2.

↳ causes brucellosis aka undulant fever or Malta fever
- acute bacteremic phase
- followed by a chronic stage

* Oxidase (+) catalase (+).
* H_2S produced by many strains.
* sensitive to heat and acidity → killed in milk by pasteurization.
* Hazardous in labs so classification tests should be performed in reference public health labs.
  ↳ additional note:- BSL-2 or BSL-

* pathogenesis:-
  - intestinal tract by ingestion infected milk → unpasteurized goats' milk is a common vehicle
  - mucous membrane by droplets
  - skin by contact with infected tissues of animals.

- lymphatic channels and regional lymph nodes → thoracic duct and bloodstream → parenchymatous organs
- Granulomatous nodules that may develop into abscesses in ① lymphatic tissue ② liver ③ spleen ④ bone marrow and other parts of the reticuloendothelial system
- can cause OM, meningitis or cholecystitis.
- Erythritol is a growth factor for brucella.
  ↓ not present in human placentas

B. abortus	B. canis	B. suis	B. melitensis
* mild disease w/o suppurative complications. * non-caseating granulomas.	* mild disease	* Chronic w/ suppurative lesions. * Caseating granulomas	* more acute and severe

* Clinical findings:-
  - incubation period is 1-4 wks.
  - insidious onset, malaise, fever, weakness, sweats and aches.
  - fever ↑ in the afternoon ↓ during the night and is accompanied by drenching sweat.
  - GI and nervous symp may present.
  - LNs enlarge, spleen becomes palpable.
  - jaundice may accompany hepatitis.
  - Deep pain + disturbances in motion esp in vertebral bodies → OM
  - A chronic stage can develop.

* Diagnosis:-
  - specimens: Blood {Biopsy (for culturing → lymph nodes or bone), serum (for serologic tests)}.
  - media: Trypticase soy agar w/ or w/o 5% sheep blood, Brain-heart infusion medium and Chocolate agar.
  - conditions: 8-10% CO_2, 35-37 °C, 3 wks to conclude -vity, should be cultivated during an acute phase or during recurrence.
    ↳ can be grown in 1 wk using automated blood culture systems.
  - urease (+).

- IgM ↑ during 1st wk and peak at 3 months. (may persist during chronic disease.)
- IgG and IgA ↑ 3wks after onset and peak at 6-8 wks, high during chronicity.
- IgG agglutinin titers > 1:80 → active infection
- ELISA more sensetive and specific than agglutination test esp in chronic disease

* Treatment:-
- Tetracyclines, rifampin, trimethoprim-sulfamethoxazole, aminoglycosides and some quinolones.
- Standard Tx → a tetracycline + streptomycin or gentamicin for 2-3 wks or rifampin for 6-8 wk.
- In case of endocarditis or neurological disease → Triple Therapy → doxycycline + rifampin + an aminoglycoside.

↱ MTBC → Tuberculosis, M. lepraе → Hansen's, M. ulcerans → necrotizing skin & soft tissue infections.

MAC and non-TB mycobacteria are ← **Mycobacteria** → rod-shaped, obligate aerobes
opportunistic in AIDs and other immunocompromised patients.
↳ carbolfuchsin stain

* Mycobacterium TB:-
- GI TB can be primary w/o pulmonary involvement or in the context of active pulmonary TB.
- Risk factors: HIV/AIDs, treatment w/ anti tumor necrosis factor agents and solid organs transplantation.
- immunosuppressants ↑ reactivitoin of latent TB.
- pathogenesis of GI TB:-
  1. Swallowing infected sputum in active pulmonary disease.
  2. Hematogenous or lymphatic spread from a distant focus.
  3. Direct extension from a contiguous site.
  4. ingestion of milk products infected w/ M. bovis.

- The entire GIT can be involved but the ileocecal region is the most common.
- Visceral organs TB may involve the liver, pancreas, GB, spleen, kidneys and adnexa.

- Caseating granulomas release organisms into the lymph
- Ulcerated mucosa → healing → strictures.

- Complications: bleeding, diarrhea, WT loss, obstruction, intussusception, perforation, stricturing disease and fistulae.
- Clinical manifestations of GI TB:-
  - no pathognomonic signs so it may mimic crohn's, colorectal cancer, appendicitis and inf conditions
  - presentation is chronic (wks to months).
  - most common symp is colicky and intermittent abdominal pain in the RLQ or periumbilical regions.
  - Other symp: anorexia, WL and fever.
  - Change in bowel habits with diarrhea more common than constipation.

- Culture and morphology:-
  - Coccoid and filamentous forms may be seen on media.
  - Gram invisible
  - ZN technique, preferred stains are fluorochrome stains such as auramine and rhodamine.
  - Non-selective and selective media used for culturing:-
    1. Semisynthetic agar media such as Middlebrooke 7H10 and 7H11
    2. Inspissated egg media such as lowenstein-Jenson (3-6 wks)
    3. Broth media such as Middlebrooke 7H9 and 7H12, eg (MGIT BACTEC) ← more rapid growth

* Diagnosis of GI TB

- High index of suspicion
- Biopsies via endoscopy → sent for Histology → PCR and acid-fast bacillus stain/culture.
  - PCR: high specificity and is more sensitive than acid-fast bacillus stain/culture
  - acid-fast bacillus stain/culture: low sensitivity but high specificity
- ideally acid-fast bacilli or caseous necrosis is present. (two low prevalence).
- Abdominal TB is a paucibacillary disease. (few bacteria are present).
- Tuberculin skin Test (TST) and Interferon gamma release assays (IGRAs) can't differentiate btw active and latent TB.
  - TST: False (-ve) are common
  - IGRAs: → such as Quantiferon TB gold plus or T-spotTB; help differentiate Crohn's from GI TB
- CT, MRI, PET scans, barium studies and endoscopic ultrasound provide supportive info but unable to establish the diagnosis.

* Treatment of GI TB:-

- First 2 months → 4 drug regimen (isoniazid, rifampin, pyrazinamide and ethambutol).
  - ↳ next 4 months → isoniazid and rifampin
- Disseminated disease requires prolonged therapy > 6 months
- Surgery might be needed is case of sig bleeding, obstruction, abscess formation and larg or drug-refractory fistulae.
- Prevention by ↓ from active pulmunary TB, treating latent TB in immunocompromised patients and avoid ingesting unpasteurized milk products.

Leptospira

- spirochete
- Tightly coiled, thin, flexible with one end forming a hook.
- actively motile
- seen using a dark-field microscope
- can survive for wks in water at alkaline pH
- grows best under aerobic conditions, 28-30°C in semisolid media such as Ellinghausen-McCullough-Johnson-Harris
- leptospira interrogans is the only path species (200 serovars).
- Causes leptospirosis which is a zoonosis

* Pathogenesis:-

- Enters through breaks in the skin and mucous membrane (ingestion is less important).
- incubation is 1-2 wks, infection is established is in parenchamous organs (liver and kidney).
- Hemorrhage and necrosis lead to organ dysfunction which results in jaundice, hemorrhage and nitrogen retention.
- The illness is biphasic, the 2nd phase develops when IgM titer rises.
  - ↳ manifests as aseptic meningitis w/ intense headache, stiff neck and pleocytosis of the CSF.
- could be mild or subclinical.
- Serovar-specific immunity follows infections, meaning reinfection w/ other serovars is possible.

* Diagnosis and treatment:-

- specimens: blood in a heparin tube, CSF, tissues, urine and serum (for agglutination tests).
  - antibodies appear 5-7 days and peak at 5-8 wks.
- Dark field or Giemsa stain of fresh blood may show leptospira in early infections
- Semisolid medium for culturing, growth is slow so culture should be kept for at least 8 wks.
- Diagnosis is confirmed serologically in most cases.
- Tx for mild disease: oral doxycycline, ampicillin or amoxicillin.
- Tx for moderate to severe disease: IV penicillin, ampicillin or ceftriaxone.

- infection is caused after contact with water or materials contaminated w/ animal excreta.
- prevention: rodent control, avoiding contaminated water and prophylaxis with 200mg doxycycline once weekly during heavy exposure.

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causes Q fever ↑ ↑ resistant to drying (survives for month in dried milk or feces.

may survive 60°C for 30 mins. ← Coxiella Burnetti → small obligate organism that only grows in cytoplasmic vacuoles.

phase I is the virulent infectious one, phase II is not infectious.
↳ occurs after serial passage in cell culture.

↳ doesn't stain w/ gram stain tho its membrane is similar to gram -ve bacteria.

*Pathogenesis:-
- Ticks transmit it to animals but uncommonly to humans.
- infection by inhalation of dust or aerosols from placenta, dried feces, urine or milk. (ingestion of unpasteurized milk is less common).
- Reservoirs: sheep, goats, cattle and parturient cats.

*Dignosis and treatment:-
- Acute Q fever resembles influenza, atypical pneumonia and hepatitis w/ antibodies to phase II antigens.
- Chronic Q fever lasts more than 6 months w/ infective endocarditis being the most common form. High antibodies to phase I antigens.
- Dignosis mainly be serology. (like leptospira).
- in case of culture -ve endocarditis PCR is useful.
- Tx of acute Q fever → doxycycline.
- Tx of Chronic Q fever → doxycycline + hydroxychloroquine for ≥ 18 months, valve replacement may be required.
- prevention → pasteurization at 71.5°C for 15 seconds.