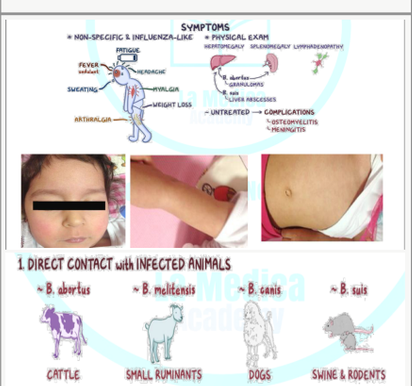
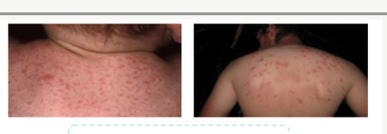
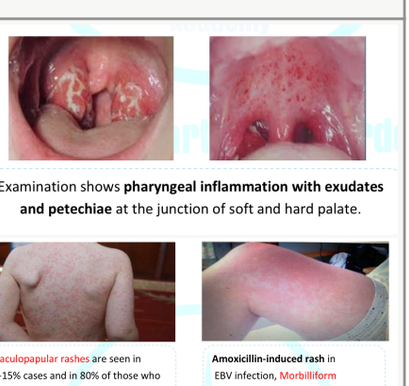
Non motile , non spores forming , urease and catalase positive.

	Brucellosis — the zoonotic master of disguise	Typhoid Fever — the systemic GI invader	Infectious Mononucleosis — immune response doing the damage
Core Identity	Think occupational + food-borne + intracellular. • Gram-negative aerobic coccobacillus • Facultative intracellular → chronicity + relapse • Humans = dead-end hosts • Childhood cases mostly from unpasteurized milk	Think human reservoir + fecal–oral + stepwise illness. • Caused by Salmonella typhi • Humans are the only reservoir • Chronic carriers matter epidemiologically	This is not the virus being mean. It's the immune system overreacting. • Caused by EBV (DNA herpesvirus) • Spread via saliva **"the kissing disease"** • Lifelong latency after infection
Epidemiology Clues	• Endemic where animal vaccination/pasteurization is weak • Males > females (exposure, not biology) • Children = 10–30% of cases	• Strongly linked to sanitation failure • Children <5 years → highest incidence & complications • Southeast Asia carries most of the global burden	**Who gets sick?** • Young children: usually silent • Adolescents/young adults: classic disease • >95% of humans eventually infected • Virus persists lifelong; intermittent contagiousness
Transmission (exams love this)	• Raw milk & dairy (main route in kids) • Aborted animal products • Skin/mucosal contact • Rarely undercooked meat	◦ Travel/work in endemic areas ◦ Handling Salmonella typhi in labs ◦ Close contact with infected/recently infected persons ◦ Drinking sewage-contaminated water • Long-Term Carriers:(be contagious with typhoid fever) ◦ Can shed S. typhi for ≥1 year without symptoms ◦ Post-recovery testing important to prevent spread	
Clinical Pattern	The illness whispers before it shouts. • Incubation: 2–4 weeks • Classic triad: Fever (often undulant) Arthralgia/arthritis Hepatosplenomegaly Children often show: • Poor appetite • Lassitude • Failure to thrive Bones/joints are frequent targets. CNS involvement is rare (~1%).	 **1st Week** • Symptoms: **Fever, malaise, anorexia, myalgia, headache, abdominal pain** • Early diarrhea → may be followed by **constipation** • Severe **lethargy** • Fever rises gradually; **classic stepladder pattern** is rare **2nd Week** • **High, sustained fever** • Fatigue, anorexia, cough, epistaxis • Abdomen: **hepatomegaly, splenomegaly, tenderness, distension** • Patient appears **acutely ill, disoriented, lethargic**, possible delirium/stupor • **Relative bradycardia** (pulse slower than expected for fever) • **Rose spots** appear in ~25% (day 7–10) **3rd Week** • Symptoms **gradually resolve in 2–4 weeks** if uncomplicated • **Malaise and lethargy** may persist **1–2 months** Paratyphoid Fever: milder, abrupt onset, more rash, fewer intestinal complications	**Classic Triad** • Fever • Pharyngitis • Generalized lymphadenopathy , **glandular fever (**name from the mononuclear lymphocytosi) • **Additional Symptoms:** • Headache, nausea, abdominal pain • Prodrome: 1–2 weeks • **PE** • Splenomegaly (50%), typically 2–3 cm below costal margin • Hepatomegaly (10%) • Sore throat with moderate–severe pharyngitis, tonsillar enlargement • Rash & eyelid edema
Diagnosis Logic	History + triad raises suspicion. Proof comes from: • **Culture (gold standard, slow but definitive)** • If culture negative → **Serology** – **SAT** pitfalls: false positives, prozone effect – EIA = most sensitive – PCR helps when available	• **Blood culture early (gold standard)** • Bone marrow culture = most sensitive (even after antibiotics) • Stool/urine cultures later (2nd–3rd week) • rose spot biopsy culture (63% sensitive) • **Widal Test:** Detect O & H antibodies (Tube agglutination, incubate 37°C overnight) • Testing for O agglutinins in Felix tubes, Chalk • Testing for H agglutinins in Dryers tubes, a narrow tube floccules at the bottom • **Serology**: Fluorescent antibody, ELISA **Lab hints:** • Anemia, thrombocytopenia , lymphopenia • Mild hepatitis • ALT:LDH ratio <9:1 → typhoid hepatitis	• **CBC**: lymphocytosis (10,000–20,000) • with 20–40% atypical lymphocytes • **Serology**: ◦ Heterophile antibodies ◦ EBV-specific: viral capsid IgM & IgG • **Throat swab to rule out Group A streptococcus** • Mild elevation of hepatic transaminases (~50%) • PCR for EBV DNA, CSF analysis if needed
Treatment (memorize cleanly)	• >8 years: Doxycycline + Rifampin × 6 weeks • <8 years: TMP-SMX + Rifampin × 6 weeks Human brucellosis is prevented in animals, not humans. Pasteurization, animal vaccination, public awareness.	• Fluoroquinolones (ciprofloxacin) if sensitive • Ceftriaxone or azithromycin if resistant • Duration: 14 days Chronic carriers may need prolonged antibiotics ± cholecystectomy. **Prognosis & Exam Traps** Relapse: 2–4% in children Chronic carriers: ≥3 months shedding S. typhi	**Famous Trap** Giving amoxicillin/ampicillin → rash in ~80% This is not a penicillin allergy. **Management** • Supportive only • No antivirals routinely • Steroids only for: – Airway obstruction – Hemolytic anemia – Severe thrombocytopenia No contact sports while spleen during first 2–3 weeks or is enlarged. • Excellent • Major symptoms: 2–4 week • Full recovery: ~2 months • Fatigue may linger weeks to months • Rare but feared: splenic rupture
Picture	 	 Rose Spots **Exam Tricks to Watch:** Rose spots appear ~day 7–10, not always present Relative bradycardia vs high fever ALT:LDH ratio for typhoid vs viral hepatitis Paratyphoid = milder, abrupt onset, more rash **Get vaccinated.**	 Examination shows **pharyngeal inflammation with exudates and petechiae** at the junction of soft and hard palate. Maculopapular rashes are seen in 3-15% cases and in 80% of those who have received **ampicillin or amoxicillin** **Amoxicillin-induced rash** in EBV infection, Morbilliform maculopapular rash on the leg.
	Incomplete treatment → osteomyelitis, meningitis, endocarditis. **Congenital brucellosis:** - Congenital brucellosis occurs using trans-placental transmission due to maternal infection during pregnancy or ingestion of the contaminated maternal secretions during birth and breastfeeding. - **The diagnosis** of brucellosis is made by culture or serology. - It may **lead to** spontaneous abortion, intrauterine fetal death, or delivery of an infected neonate.	**Complications** **GI**: bleeding, perforation **Cardiac**: myocarditis, endocarditis **Other**: pancreatitis, meningitis, osteomyelitis, pneumonia, urinary infections **Neuro**: confusion, hallucinations	**Complications** • Airway obstruction (enlarged tonsils/oropharyngeal lymphoid tissue) • Neurologic: meningitis, seizures, ataxia, Guillain-Barré, Reye syndrome, Alice in Wonderland syndrome • Cardiopulmonary: myocarditis, interstitial pneumonitis • Hematologic: hemolytic anemia, thrombocytopenia, neutropenia, aplastic anemia • Splenic: subcapsular hemorrhage, splenic rupture (especially with trauma) • Gastrointestinal: pancreatitis

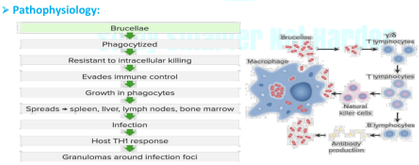
In our setting, the usual endemic lineup:

• Bacterial: Brucellosis, Typhoid

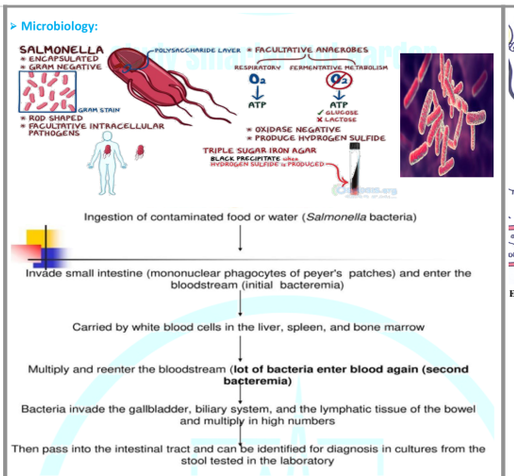
• Viral: Infectious mononucleosis (EBV)

• Parasitic: Leishmaniasis

➢ **Pathophysiology:**



➢ **Microbiology:**



Ingestion of contaminated food or water (*Salmonella* bacteria)

↓

Invade small intestine (mononuclear phagocytes of peyer's patches) and enter the bloodstream (initial bacteremia)

↓

Carried by white blood cells in the liver, spleen, and bone marrow

↓

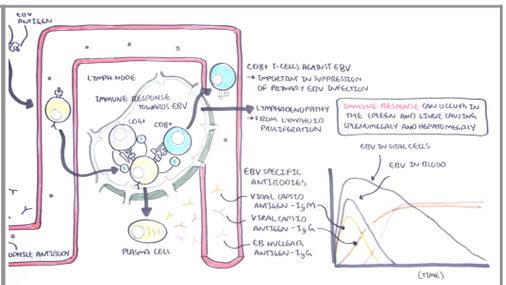
Multiply and reenter the bloodstream (**lot of bacteria enter blood again (second bacteremia)**

↓

Bacteria invade the gallbladder, biliary system, and the lymphatic tissue of the bowel and multiply in high numbers

↓

Then pass into the intestinal tract and can be identified for diagnosis in cultures from the stool tested in the laboratory



EBV Immune Response – Simplified

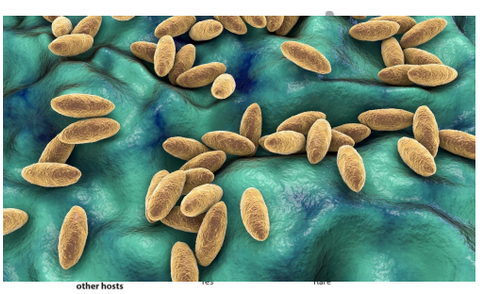
1. CD8+ T-cells (Cytotoxic T-cells):
   - Activated early during the incubation period → generally attack any infected B-cells.
   - Later, EBV-specific CD8+ T-cells expand closer to symptom onset.
2. CD4+ Helper T-cells:
   - Stimulate normal B-cells → transform them into plasma cells → produce antibodies → create long-term immune memory.
3. Antibodies:
   - First: Viral capsid IgM → remains in blood for ~4 months.
   - Next: Viral capsid IgG.
   - Finally: Nuclear IgG → indicates resolution of infection.
4. Clinical Manifestation:
   - Primary infection can be asymptomatic or very mild.
   - If symptoms appear → usually start ~6 weeks after infection due to immune response.

Pathophysiology (high yield)

- **Incubation : 2-6 weeks**
- **EBV infects B cells**

- **(primary EBV infection.) :**
- CD8+ T cells expand → atypical lymphocytes (Downey cells)
- Heterophile antibodies appear → diagnostic handle

- **Like other herpes viruses, EBV establishes lifelong latent infection** after the primary infection with frequent asymptomatic reactivations.



other hosts	Yes	Rare
Vaccine	Yes (S19, RB51, REV1)	No
Treatment	No	Yes