Viruses that Infect the Enteric Tract

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"Viruses that Infect the Enteric Tract" – Introduction

General Features

- Transmission: Fecal–oral route → entry via enteric tract.
- Diseases caused:
  - Local (GI): Norovirus, Rotavirus → diarrheal disease.
  - Systemic (extra-intestinal): Poliovirus, Coxsackievirus, Echovirus → CNS disease (e.g., meningitis, encephalitis).
  - Other Coxsackievirus diseases:
    - Hand, foot & mouth disease
    - Myocarditis

Classification

- Poliovirus, Coxsackievirus, Echovirus → belong to Enteroviruses (Picornavirus family).
- "Enterovirus" name significance:
  - Enteric tract = major site of replication.

- Feces = common source of infection & specimen for lab diagnosis.

- Note: Coxsackievirus & Echovirus can also replicate in upper respiratory tract → cause respiratory symptoms.

Structure

- All enteric viruses in this chapter: Naked nucleocapsid viruses (no envelope).

  - Clinical importance: Non-enveloped viruses are environmentally stable, resist drying, acids, detergents → survive in environment & transmit via fecal-oral route.

Related Viruses

- Hepatitis A & E viruses also enter via enteric tract → covered separately with hepatitis viruses.

Properties of Viruses Commonly Infecting the Intestinal Tract – Summary

1. Norovirus

- Virus Family: Caliciviruses

- Genome: Single-stranded RNA, positive polarity

- Virion RNA Polymerase: No
- Nucleocapsid: Icosahedral
- Envelope: No
- Number of Serotypes: Two or more

2. Rotavirus

- Virus Family: Reoviruses
- Genome: Double-stranded RNA; 11 segments
- Virion RNA Polymerase: Yes
- Nucleocapsid: Icosahedral
- Envelope: No
- Number of Serotypes: At least six

3. Poliovirus

- Virus Family: Picornavirus
- Genome: Single-stranded RNA, positive polarity

- Virion RNA Polymerase: No
- Nucleocapsid: Icosahedral
- Envelope: No
- Number of Serotypes: Three

4. Coxsackie Virus

- Virus Family: Picornavirus
- Genome: Single-stranded RNA, positive polarity
- Virion RNA Polymerase: No
- Nucleocapsid: Icosahedral
- Envelope: No
- Number of Serotypes: Many

5. Echovirus

- Virus Family: Picornavirus
- Genome: Single-stranded RNA, positive polarity

- Virion RNA Polymerase: No
- Nucleocapsid: Icosahedral
- Envelope: No
- Number of Serotypes: Many

Clinical Features of Viruses Commonly Infecting the Intestinal Tract - Summary

1. Norovirus

- Disease: Gastroenteritis
- Main Clinical Findings: Watery diarrhea
- Vaccine Available: No
- Antiviral Therapy: No

2. Rotavirus

- Disease: Gastroenteritis
- Main Clinical Findings: Watery diarrhea, especially in infants

- Vaccine Available: Yes
- Antiviral Therapy: No

3. Poliovirus

- Disease: Poliomyelitis
- Main Clinical Findings: Paralysis due to death of motor neurons
- Vaccine Available: Yes
- Antiviral Therapy: No

4. Coxsackie Virus

- Diseases & Findings:
    1. Hand, foot, and mouth disease → Vesicular lesions on hands, feet, and mouth
    2. Meningitis → Fever, headache, stiff neck
    3. Myocarditis → Congestive heart failure
- Vaccine Available: No

- Antiviral Therapy: No

S. Echovirus

- Disease: Meningitis
- Main Clinical Findings: Fever, headache, stiff neck
- Vaccine Available: No
- Antiviral Therapy: No

➢ Norovirus

Disease

- One of the most common causes of viral gastroenteritis:
    - Adults: Worldwide major cause.
    - Children (U.S.): Leading cause (since rotavirus incidence dropped after vaccination).
- Norwalk virus: Prototype Norovirus → first outbreak described in Norwalk, Ohio (1969).

Important Properties

- Genome: Non-segmented, ssRNA (+ sense).

- Capsid: Non-enveloped, icosahedral nucleocapsid.
- Virion lacks polymerase (replicates using host machinery).
- Morphology (EM features):
  - 10 prominent spikes.
  - 32 cup-shaped depressions ("calici" = cup).
- Antigenic variation:
  - ≥ 2 serotypes (exact number uncertain).
  - 6 genogroups → Genogroup II = most common in humans.

Replication

- Replicates similar to Poliovirus (another Picornavirus) → ssRNA (+) genome acts as mRNA directly on entry.

✅ Exam Tip Highlights

- *MCQ/Viva:* Which viruses are naked nucleocapsid & hence fecal-oral? → Norovirus, Rotavirus, Enteroviruses (Polio, Coxsackie, Echo), HAV, HEV.

- *Structure recall:* "Cup-shaped depressions" in Norovirus (hence genus name *Calicivirus*).

- *Concept link:* Non-enveloped → survives outside → fecal-oral transmission.

Norovirus – Transmission, Epidemiology, Pathogenesis, Clinical Features, Diagnosis & Prevention

Transmission & Epidemiology

- Mode: Fecal-oral route.

- Sources: Contaminated seafood, water, and fomites (door handles, surfaces).

- Settings of outbreaks:

    - Cruise ships (esp. Caribbean)
    - Schools, camps
    - Hospitals, nursing homes

- Person-to-person transmission common in group settings.

- Animal caliciviruses exist, but no zoonotic infection documented.

Features that enhance spread:

- Very low infectious dose.
- Prolonged shedding: Virus excreted in stool before symptoms & for weeks after recovery.
- Environmental resistance:
    - Resistant to chlorination & drying.
    - Infectious for days in water, food, and on surfaces.

Pathogenesis & Immunity

- Target site: Limited to intestinal mucosal cells.
- Effect: Watery diarrhea without blood or WBCs.
- Asymptomatic infections common (detected via antibodies).
- Immunity:
    - Short-lived after infection.
    - Reinfections possible.
- Epidemiology: New strains appear every 2–4 years → widespread outbreaks.

Clinical Findings

- Incubation: Short, rapid onset.
- Symptoms:
  - Sudden vomiting + watery diarrhea.
  - Low-grade fever, abdominal cramps.
- Stool & emesis: No blood.
- Duration: Self-limiting, 2–3 days.
- Complications:
  - None in healthy persons.
  - In immunocompromised: chronic gastroenteritis.
  - Some outbreaks: CNS symptoms (headache, meningismus, photophobia, obtundation).

Laboratory Diagnosis

- Gold standard: PCR on stool or vomitus.
- Practical approach: Usually clinical diagnosis in outbreaks.

Treatment & Prevention

- No specific antiviral or vaccine.

- Treatment:
    - Oral or IV rehydration → correct dehydration & electrolyte imbalance.
- Prevention:
    - Good personal hygiene (handwashing).
    - Public health measures: proper sewage disposal, disinfection of contaminated surfaces.

✅ Exam Tip Highlights

- *MCQ/Viva:* Which virus is the *most common cause of adult viral gastroenteritis worldwide? → Norovirus.
- *Remember:* Norovirus = resistant to chlorination & drying → survives on surfaces (fomites).
- *Important clinical point:* Norovirus gastroenteritis → watery diarrhea + vomiting without blood or WBCs (differentiates from bacterial dysentery).
- *Epidemiology Q:* Major outbreak settings = cruise ships & nursing homes.

➢ Rotavirus

Disease

- Major cause of viral gastroenteritis in young children.

- Important cause of infantile diarrhea worldwide (esp. severe diarrhea → hospitalization).

Important Properties

- Genome:
    - Segmented dsRNA genome (11 segments).
    - Unique feature: Only medically important human virus with segmented dsRNA.

- Structure:
    - Double-layered icosahedral capsid (no envelope).
    - Virion contains RNA-dependent RNA polymerase (essential, because host cells lack enzymes to synthesize mRNA from dsRNA).

- Serotypes: ≥ 6 serotypes of human Rotavirus.
    - Outer capsid glycoprotein = viral hemagglutinin = type-specific antigen.

- Induces protective antibody response.

- Zoonotic note: Many domestic animals infected with their own strains, but no cross-infection with humans.

Replicative Cycle

1. Attachment: Virus binds to β-adrenergic receptors on intestinal mucosal cells.

2. Entry: Virion enters cell.

3. Transcription:

    - Virion RNA-dependent RNA polymerase synthesizes mRNA from each of the 11 genome segments.
    - Process occurs in cytoplasm.

4. Translation: mRNAs → structural & non-structural proteins.

5. Genome replication:

    - A viral RNA polymerase synthesizes minus strands to form dsRNA genome.
    - Capsid proteins assemble around the genome.

6. Release: Virus exits by cell lysis (not budding).

Transmission & Epidemiology

- Mode: Fecal–oral route.
- Distribution: Worldwide.
- Age: By 6 years, almost all children have antibodies to ≥ 1 serotype → indicates widespread exposure.

✅ Exam Highlights

- *MCQ:* Only human virus with segmented dsRNA genome = Rotavirus.
- *Viva:* Why does Rotavirus carry its own RNA polymerase? → Host lacks enzyme to make mRNA from dsRNA.
- *Important point:* Capsid has double layer → gives environmental stability (important for fecal–oral spread).
- *Clinical relevance:* Leading cause of severe diarrhea in infants & young children.
- *Pathogenesis link:* Attachment to β-adrenergic receptor (unique detail).

Rotavirus – Pathogenesis, Immunity, Clinical Findings, Diagnosis & Prevention

Pathogenesis

- Site of replication: Mucosal cells of the small intestine.
- Mechanism of diarrhea:
  - Excess secretion of fluids & electrolytes into the bowel lumen.
  - Loss of salt, glucose, water → watery diarrhea.
  - No inflammation → diarrhea is non-bloody.
  - Likely mediated by stimulation of the enteric nervous system.
- Virulence determinants (from animal studies):
  - Certain genome segments control:
    - Tissue tropism
    - Inhibition of host RNA/protein synthesis

Immunity

- Protective immunity: Likely via intestinal IgA against specific serotypes.

- Neonatal protection: Colostrum IgA protects infants up to 6 months.

- Reinfection: Possible, especially with different serotypes.

Clinical Findings

- Symptoms:
    - Nausea, vomiting
    - Watery, non-bloody diarrhea

- Severity:
    - Most serious in young children → dehydration & electrolyte imbalance are major concerns.
    - Adults → usually mild illness.

Laboratory Diagnosis

- Most cases: Clinical diagnosis.

- Lab confirmation:
    - ELISA: Detects rotavirus antigen in stool.
    - PCR: Most sensitive method → detects viral RNA.
    - Serology: ≥ 4-fold rise in antibody titer confirms infection.

Treatment & Prevention

- Treatment:
    - No antiviral therapy.
    - Supportive care: Oral/IV rehydration to correct dehydration & electrolyte loss.
- Vaccines: Live oral vaccines available:
    1. Rotarix – live, attenuated, single human serotype G1.
    2. Rotateq – live reassortant, 5 serotypes (G1–G4, G9).
        - Outer surface protein from human virus inserted into nonpathogenic bovine strain → induces protective IgA.
- Vaccine caution:
    - History of intussusception → contraindicated.
    - Previous vaccine Rotashield withdrawn due to intussusception risk.
- Hygienic measures: Handwashing, proper sewage disposal.

✅ Exam Highlights

- *Pathogenesis:* Watery diarrhea is non-inflammatory → no blood/WBCs in stool.
- *Immunity:* Intestinal IgA = key protective factor; colostrum IgA protects newborns.
- *Vaccine:* Remember Rotarix = single strain GI, Rotateq = 5 reassortant strains; oral administration.
- *Lab Diagnosis:* ELISA common, PCR most sensitive.
- *Clinical relevance:* Severe in young children, mild in adults.

➢ Poliovirus

Disease

- Causes poliomyelitis.
- Key symptom: Paralysis (may be asymptomatic in most cases).

Important Properties

- Genome:

- Non-segmented, ssRNA (+).
- No polymerase in virion (replicates using host machinery after translation).

- Capsid: Non-enveloped, icosahedral nucleocapsid.

- Serotypes: 3 serotypes → each requires specific antibody for protection.

- Host range:

  - Primates only (humans + non-human primates).
  - Viral capsid binds to primate-specific receptor.
  - Purified viral RNA is infectious in non-primate cells (can bypass receptor).

Antigenicity:

- 3 serologic types based on capsid proteins.

- Little cross-protection → immunity requires antibodies to all three types.

Replicative Cycle

1. Attachment & Entry: Virion binds to specific cell receptors on the membrane → enters cell.

2. Uncoating: Capsid removed → RNA released.

3. Translation:

    - Genome RNA functions as mRNA.
    - Translated into a single large polypeptide (polyprotein) called noncapsid viral protein 00.

4. Polyprotein processing:

    - Viral protease cleaves polyprotein into:
        - Capsid proteins → form virion coat
        - Non-capsid proteins, including RNA polymerase for genome replication

5. Replication:

    - Synthesis of complementary (–) strand → template for new (+) strands.
    - Some (+) strands → mRNA for viral proteins.
    - Remaining (+) strands → progeny genomes.

6. Assembly: Genome + capsid proteins → progeny virions.

7. Release: Accumulate in cytoplasm → released upon cell lysis (no budding).

✅ Exam Highlights

- *Genome type:* ssRNA (+), non-segmented → similar to other Picornaviruses (Coxsackie, Echovirus).
- *Serotypes:* 3 → all three must be covered for complete immunity.
- *Replication:* Polyprotein strategy → hallmark of Picornaviruses.
- *Clinical link:* Paralysis → selective destruction of motor neurons (covered in pathogenesis).
- *Host range quirk:* Primate-specific receptor → explains human-only natural infection.

Poliovirus – Transmission, Epidemiology, Pathogenesis & Clinical Features

Transmission & Epidemiology

- Mode: Fecal-oral route.
- Replication sites: Oropharynx and intestinal tract.
- Host: Humans only.
- Historical epidemiology:

    - Pre-vaccine → summer/fall epidemics.
    - Post-vaccine → eradicated in US & Western Hemisphere.
    - Rare US cases:
        1. Exposure to virulent revertants from live vaccine.
        2. Unimmunized travelers exposed to wild-type virus abroad.

- Global status:
    - 1988: ~388,000 paralytic polio cases worldwide.
    - 2005: <2000 cases.
    - 2017: <100 cases, restricted to Afghanistan, Pakistan, Nigeria.

- WHO goal: Global eradication by 2005 (not yet achieved).

- Note: Smallpox = only fully eradicated human infectious disease.

Pathogenesis

- Primary replication: Oropharynx & small intestine, esp. lymphoid tissue.

- Spread:

    - Viremia → CNS
    - Retrograde transport along nerve axons.

- CNS tropism:

    - Preferentially infects motor neurons in anterior horn of spinal cord → flaccid paralysis.
    - Brainstem involvement → bulbar poliomyelitis → respiratory paralysis.
    - Rarely affects cerebral cortex.

- Mechanism of paralysis:

    - Neuron death → muscle paralysis.
    - Not due to direct muscle infection.

- Immune response:

    - Intestinal IgA + humoral IgG against specific serotype.
    - Infection → lifelong type-specific immunity.

Clinical Findings

- Range of illness:

    1. Asymptomatic infection (most common)

2. Abortive poliomyelitis – mild, febrile illness

3. Nonparalytic poliomyelitis – aseptic meningitis

4. Paralytic poliomyelitis – flaccid paralysis, possible brainstem involvement

- Incubation period: 10–14 days.

Details by form:

1. Abortive poliomyelitis:
    - Headache, sore throat, nausea, vomiting
    - Self-limiting

2. Nonparalytic poliomyelitis:
    - Aseptic meningitis → fever, headache, stiff neck
    - Usually resolves spontaneously

3. Paralytic poliomyelitis:
    - Flaccid paralysis of affected muscles
    - Painful muscle spasms
    - Brainstem involvement → respiratory paralysis
    - Spinal cord + brain parenchyma → meningoencephalitis or meningomyeloencephalitis

    - Motor neuron damage permanent, partial recovery possible via collateral nerve takeover

- Post-polio syndrome: Late deterioration of previously affected muscles, cause unknown.

- Fecal shedding: Virus excreted for several months; no permanent carrier state.

✅ Exam Highlights

- *Transmission:* Fecal-oral; human-only host.

- *CNS tropism:* Anterior horn motor neurons → flaccid paralysis.

- *Clinical spectrum:* Most infections asymptomatic; <1% develop paralysis.

- *Post-polio syndrome:* Occurs years after acute illness.

- *Global eradication:* Only 3 countries remain endemic (Afghanistan, Pakistan, Nigeria).

Poliovirus – Laboratory Diagnosis, Treatment & Prevention

Laboratory Diagnosis

- Methods:

    1. Virus isolation

        - From throat, stool, or cerebrospinal fluid (CSF)
        - Inoculation into cell cultures → cytopathic effect (CPE)
        - Neutralization with type-specific antisera confirms identity

    2. PCR-based assay: Detects poliovirus RNA in clinical samples.

    3. Serology: ≥4-fold rise in antibody titer indicates recent infection.

Treatment

- No antiviral therapy available.

- Supportive care:

    - Symptomatic relief (analgesics, fever control)
    - Respiratory support if bulbar involvement occurs
    - Physiotherapy for affected muscles

Prevention

Vaccines

1. Killed (inactivated) vaccine – IPV / eIPV

    - Induces humoral antibodies (IgG) → neutralizes virus in blood → prevents CNS infection.
    - Current US vaccine: Enhanced IPV (eIPV) → higher seroconversion + some mucosal IgA.
    - Administered 4 doses: 2m, 4m, 6–18m, 4–6y; booster recommended for adults traveling to endemic areas.
    - Preferred in the US due to no risk of vaccine-derived paralytic polio.

2. Live attenuated vaccine – OPV / Sabin

    - Oral administration → induces secretory IgA → interrupts fecal–oral transmission.
    - Contains all three serotypes.
    - Previously preferred in US due to oral delivery and mucosal immunity.

- Disadvantages:

    1. Rare reversion to virulence → vaccine-derived paralytic polio (VDPV), especially type 3.

    2. Can cause disease in immunodeficient

persons.

3. Gastrointestinal coinfections may limit vaccine virus replication.

4. Must be refrigerated to maintain potency.

- Global usage:
    - WHO (2016) → worldwide use of trivalent inactivated vaccine (IPV) only; OPV phased out to prevent VDPV outbreaks.

Other Preventive Measures

- Passive immunization:
    - Immune serum globulin for unimmunized exposed individuals.
    - Maternal IgG → protects newborns.
- Quarantine: Ineffective due to virus excretion before symptom onset and in asymptomatic cases.

Special Notes

- Vaccine history: Early vaccine lots contaminated with SV40 virus (monkey virus).
    - No increased cancer risk detected, though SV40

DNA occasionally found in some human cancers.
    - Modern vaccine cell cultures are carefully screened.

- Vaccine-derived poliovirus outbreaks: Occur in areas with low immunization coverage → controlled by OPV campaigns.

✅ Exam Highlights

- *Lab diagnosis:* CPE in cell culture + neutralization with type-specific antisera is classical method.

- *Treatment:* Supportive only; physiotherapy for muscle recovery.

- *Vaccines:*

    - IPV → safer, systemic immunity; eIPV = current US vaccine.
    - OPV → induces secretory IgA, interrupts fecal-oral spread, risk of VDPV.

- *Global eradication:* OPV phased out → prevent vaccine-associated paralytic polio.

- *Passive immunization:* Useful for unimmunized exposed individuals.

Poliovirus Vaccines – Key Features

Attribute	Killed (Salk / IPV)	Live (Sabin / OPV)
Prevents disease	✅ Yes	✅ Yes
Interrupts transmission	❌ No	✅ Yes
Induces humoral IgG	✅ Yes	✅ Yes
Induces intestinal IgA	❌ No	✅ Yes
Secondary protection via spread	❌ No	✅ Yes
Interferes with replication of virulent virus in gut	❌ No	✅ Yes
Reverts to virulence	❌ No	✅ Rarely
Coinfection with other enteroviruses may impair immunization	❌ No	✅ Yes

Can cause disease in immunocompromised	❌ No	✅ Yes
Route of administration	Injection	Oral
Requires refrigeration	❌ No	✅ Yes
Duration of immunity	Shorter	Longer

✅ Exam Highlights

- IPV: Safe, injected, protects against disease, but does not stop fecal-oral transmission.
- OPV: Oral, induces intestinal IgA, interrupts transmission, rare risk of reversion, longer immunity.
- Global strategy: IPV used in US; OPV phased out in many countries to prevent vaccine-derived poliovirus outbreaks.

➢ Coxsackievirus

📌 Diseases

- Group A

- Herpangina → fever, sore throat, vesicles on soft palate & uvula.
- Hand, Foot & Mouth Disease → vesicular rash on hands, feet, mouth (often in children).
- Acute Hemorrhagic Conjunctivitis.

- Group B

  - Pleurodynia (Bornholm disease) → fever + sharp chest pain due to intercostal muscle infection.
  - Myocarditis & Pericarditis → can be severe, especially in infants.
  - Myositis.

- Both Groups (A & B)

  - Aseptic (viral) meningitis → Coxsackie + Echovirus = ~90% of cases.
  - Common cold, pharyngitis, febrile rash.
  - Acute flaccid paralysis (resembles poliomyelitis).

📌 Important Properties

- Family: Picornaviridae (like Poliovirus, Echovirus).

- Genome: Non-enveloped, icosahedral, +ssRNA virus.

- Similar structure & replication to Poliovirus.

- Groups A vs. B classification → based on *pathogenicity in mice*:
    - Group A → widespread myositis & fatal flaccid paralysis.
    - Group B → less severe, but affects heart, pancreas, CNS, focal myositis.
- Serotypes: ≥24 of Group A, 6 of Group B.

📌 Replication Cycle

- Similar to Poliovirus:
    - Virus binds receptor → uncoating → RNA acts as mRNA.
    - Single polyprotein translated → cleaved into structural & nonstructural proteins.
    - Replication via a negative-strand RNA intermediate.
    - Virions accumulate in cytoplasm → released by cell lysis.

📌 Transmission & Epidemiology

- Route: Mainly fecal-oral, also respiratory aerosols.
- Site of replication: Oropharynx & intestinal tract.

- Host: Humans only.
- Seasonality: Summer & Fall outbreaks (similar to poliovirus).
- Epidemiology: Worldwide distribution.

✅ Exam Triggers (Spotting Clues in MCQs):

- Child with oral vesicles + vesicular rash on palms/soles → Hand, Foot & Mouth (Coxsackie A).
- Sharp chest pain (pleurodynia) or infant myocarditis → Coxsackie B.
- Summer viral meningitis outbreak → Coxsackie/Echovirus.

Coxsackievirus – Pathogenesis, Immunity, Clinical, Dx, Tx

📌 Pathogenesis

- Group A: Predilection for skin & mucous membranes → rash, vesicles (herpangina, HFMD).
- Group B: Involves visceral organs → heart, pleura, pancreas, liver.

- Both groups: Can affect meninges & anterior horn cells → aseptic meningitis, poliomyelitis-like paralysis.

- Spread: Replication in oropharynx & gut → viremia → dissemination.

📌 Immunity

- Type-specific IgG develops after infection → lifelong protection to that serotype.

- No cross-protection between different serotypes.

📌 Clinical Findings

🔷 Group A diseases

- Herpangina → fever, sore throat, tender vesicles in oropharynx.

- Hand-Foot-Mouth Disease (HFMD): vesicular rash on hands & feet + oral ulcers (children).

🔷 Group B diseases

- Pleurodynia (Bornholm disease / devil's grip): fever + sharp chest pain (myositis of intercostal muscles).

- Myocarditis & Pericarditis → fever, chest pain, heart failure; can progress to dilated cardiomyopathy →

may require transplant.

- Possible link to Type 1 Diabetes Mellitus (esp. Coxsackie B4).

🔷 Both Groups

- Aseptic meningitis (major cause along with echoviruses).
- Acute flaccid paralysis (resembles poliomyelitis).
- Respiratory infections, febrile rashes, pharyngitis.

📌 Laboratory Diagnosis

- PCR for viral RNA in CSF → best for rapid diagnosis of meningitis.
- Virus isolation: cell culture / suckling mice (takes days).
- Serology: rise in neutralizing antibody titers.

📌 Treatment & Prevention

- ❌ No antiviral drug available.
- ❌ No vaccine.

- ❌ No passive immunization recommended.

- Management: Supportive only (analgesics, fluids, management of complications).

✅ Exam Clues / Buzzwords

- Child with oral ulcers + rash on hands & feet → HFMD (Coxsackie A).

- Severe chest pain in summer + fever → Pleurodynia (Coxsackie B).

- Infant with myocarditis → think Coxsackie B.

- Summer viral meningitis outbreaks → Coxsackie/Echovirus.

➢ Echovirus & Other Enteroviruses

📌 Echovirus

- ECHO = *Enteric Cytopathic Human Orphan* (initially thought nonpathogenic).

- Now known to cause:

    - Aseptic meningitis (with Coxsackie → major cause).

    - URTI.
    - Febrile illness ± rash.
    - Infantile hepatitis.
    - Hemorrhagic conjunctivitis.

- Structure: Similar to enteroviruses (non-enveloped, ss+RNA, icosahedral).

- Serotypes: >30 known.

- Animal pathogenicity: Not pathogenic for mice (unlike Coxsackie), not disease-causing in monkeys (unlike polio).

- Transmission: Fecal-oral; worldwide distribution.

- Pathogenesis: Similar to other enteroviruses (replicate in oropharynx/GIT → viremia → systemic spread).

- Diagnosis: PCR (echovirus RNA detection in CSF).

- Serology: Limited value (many serotypes, no common antigen).

- Treatment/Prevention:

    - ❌ No antiviral therapy.

- ❌ No vaccine.

📌 Other Enteroviruses

🔹 Enterovirus 68 (EV68 / EVD68)

- Disease: Respiratory tract illness → mild cold to pneumonia & respiratory failure.
- Linked to acute flaccid paralysis (polio-like illness in children).
- Diagnosis: PCR.
- ❌ No treatment or vaccine.

🔹 Enterovirus 70

- Disease: Acute hemorrhagic conjunctivitis → petechial hemorrhages on bulbar conjunctiva.
- Recovery: Complete.
- ❌ No treatment.

🔹 Enterovirus 71

- Major cause of CNS disease → meningitis, encephalitis, paralysis.
- Other diseases: diarrhea, pulmonary hemorrhage,

HFMD, herpangina.

- Important in outbreaks of severe neurologic disease in children (Asia).

◈ Enterovirus 72 = Hepatitis A virus (HAV) → discussed separately.

✅ Exam Clues / Buzzwords

- Summer viral meningitis in children → Echovirus / Coxsackie.
- Acute hemorrhagic conjunctivitis → Enterovirus 70.
- Polio-like paralysis in children, esp. after respiratory illness → Enterovirus 68.
- HFMD outbreaks in Asia with severe encephalitis → Enterovirus 71.

➢ Case Scenarios for Exams

1. Norovirus

📌 *Case Scenario:*

A 22-year-old college student develops sudden onset of profuse watery diarrhea, vomiting, and abdominal cramps 24 hours after attending a cruise ship buffet.

Several of his friends are also ill. No blood or pus is seen in stool. Symptoms resolve within 2–3 days.

👉 *Key Clues*: Outbreak, cruise ship, short incubation, self-limited watery diarrhea.

2. Rotavirus

📌 *Case Scenario*:

A 1-year-old infant presents with fever, vomiting, and watery diarrhea for 2 days. The child is dehydrated (sunken eyes, dry mucous membranes) and has not received routine vaccinations. History reveals that several other children in the daycare center are sick.

👉 *Key Clues*: Infant, daycare, winter season, severe dehydration, unvaccinated.

3. Poliovirus

📌 *Case Scenario*:

A 7-year-old boy from an area with low vaccination coverage presents with fever, sore throat, and muscle pain. After a few days, he develops asymmetric flaccid paralysis of the right leg. CSF shows mild pleocytosis but no bacteria.

👉 *Key Clues*: Unvaccinated child, preceding fever, flaccid paralysis, poliovirus endemic area.

4. Coxsackievirus

📌 *Case Scenario (A – Hand, Foot, Mouth Disease)*:

A 5-year-old child presents with fever, painful oral ulcers, and vesicular rash on palms and soles. A few children in his kindergarten have similar lesions.

👉 *Key Clues*: Child, oral ulcers + vesicles on hands/feet, daycare outbreak.

📌 *Case Scenario (B – Coxsackie B: Myocarditis)*:

A 19-year-old college athlete develops fever, chest pain, and shortness of breath following an upper respiratory infection. ECG shows diffuse ST changes.

👉 *Key Clues*: Young adult, myocarditis/pericarditis after viral illness.

5. Echovirus

📌 *Case Scenario*:

A 9-year-old boy presents with fever, headache, vomiting, and neck stiffness. CSF examination shows

clear fluid, normal glucose, and lymphocytic predominance. No bacteria are cultured. Several similar cases have been reported from his school.

👉 *Key Clues*: School outbreak, aseptic meningitis, lymphocytic CSF, no bacteria.