

Poliovirus Infection in Children: A Comprehensive Review of Virology, Pathogenesis, Epidemiology and Global Eradication Efforts

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Abstract:

➤ Background:

Poliomyelitis, caused by *poliovirus*, remains a significant public health concern despite global eradication efforts. This review comprehensively examines the virology, pathogenesis, epidemiology, clinical manifestations, and eradication strategies for *poliovirus* infection in children.

➤ Methods:

We conducted a narrative review of peer-reviewed literature, WHO reports, and Global Polio Eradication Initiative (GPEI) publications.

➤ Results:

Poliovirus, a positive-sense, single-stranded RNA virus of the *Picornaviridae* family, has three serotypes (1, 2, 3). *Wild poliovirus type 2* was declared eradicated in 2015, and type 3 in 2019, leaving only type 1 circulating endemically in Afghanistan and Pakistan. The virus is transmitted primarily via the faecal-oral route, with children under five years being most susceptible. Approximately 75% of infections are asymptomatic; however, 1 in 200 infections results in irreversible paralysis, with 5–10% mortality among paralysed cases due to respiratory failure. Two effective vaccines exist: the inactivated *poliovirus* vaccine (IPV) and the oral poliovirus vaccine (OPV). While OPV has been instrumental in eradication efforts, rare cases of vaccine-derived polioviruses (VDPV) pose challenges to the endgame strategy.

➤ Conclusion:

Sustained vaccination coverage, enhanced surveillance, and eventual transition from OPV to IPV are critical for achieving and maintaining global polio eradication.

Keywords: *Poliovirus; Poliomyelitis; Children; Oral Polio Vaccine; Inactivated Polio Vaccine; Eradication; Vaccine-Derived Poliovirus.*

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I. INTRODUCTION

Poliomyelitis, commonly known as polio, is an acute infectious disease caused by the poliovirus, a member of the Enterovirus C species within the family *Picornaviridae*. The disease has afflicted humanity for millennia, with ancient Egyptian carvings depicting individuals with characteristic limb deformities suggestive of polio (Paul, 1971). Before the development of effective vaccines in the 1950s, polio was one of the most feared childhood diseases globally, causing hundreds of thousands of paralytic cases annually (Hogle et al., 1985).

Poliovirus is a remarkably simple yet highly infectious pathogen. It has a positive-sense, single-stranded RNA genome enclosed in a non-enveloped icosahedral protein capsid (Goodsell, 1998). The virus exhibits three distinct serotypes—types 1, 2, and 3—each with slightly different capsid proteins that define their antigenicity and receptor specificity (He et al., 2003). Notably, wild poliovirus type 1 (WPV1) is the most virulent and has caused most *paralytic cases* (Kew et al., 2005).

The clinical spectrum of poliovirus infection ranges from asymptomatic infection (approximately 75% of cases) to *mild febrile illness* (about 25%) and, in less than 1% of infections, paralytic poliomyelitis (Mueller et al., 2005). The paralytic form occurs when the virus invades the central nervous system and destroys motor neurons in the spinal cord, brainstem, or motor cortex, leading to irreversible flaccid paralysis (De Jesus, 2007). Among those who develop paralysis, 5–10% die when respiratory muscles become immobilized (WHO, 2024).

The development of polio vaccines stands as one of the greatest achievements in public health history. Jonas Salk developed the inactivated poliovirus vaccine (IPV) in 1955, followed by Albert Sabin's live-attenuated oral poliovirus vaccine (OPV) in 1961 (Sabin, 1956). The latter became the primary tool of the *Global Polio Eradication Initiative* (GPEI), launched in 1988, which has reduced global polio incidence by over 99% (GPEI, 2024). Today, *wild poliovirus* is endemic in only two countries—Afghanistan and Pakistan—and types 2 and 3 have been declared eradicated (GPEI, 2019).

However, challenges persist. Circulating vaccine-derived polioviruses (cVDPVs), arising from genetic reversion of the attenuated OPV strains, can cause paralytic disease in under-immunized populations (Kew et al., 2005). Additionally, the endgame strategy—including the phased withdrawal of OPV and transition to IPV-only schedules—requires careful coordination to maintain global immunity (WHO, 2014).

This comprehensive review aims to synthesise current knowledge on poliovirus virology, pathogenesis, epidemiology, clinical features, vaccination strategies, and the remaining challenges in achieving a polio-free world, with particular emphasis on children, who bear the greatest burden of disease.

➤ Highlights

- Poliovirus has three serotypes; only wild-type 1 remains endemic
- Children under 5 are most susceptible to infection and paralysis
- OPV has reduced global incidence by 99% since 1988
- VDPVs remain a challenge in under-immunized populations
- IPV-only schedules are recommended for the post-eradication era

II. VIROLOGY

➤ Viral Structure and Genome Organization

Poliovirus is a small, non-enveloped virus measuring approximately 30 nm in diameter with icosahedral symmetry (Hogle et al., 1985). The capsid is composed of 60 copies each of four structural proteins—VP1, VP2, VP3, and VP4—organized into a pseudo-T=3 symmetry (Carter & Saunders, 2007). The capsid encloses a single-stranded, positive-sense RNA genome of approximately 7,500 nucleotides (Kitamura et al., 1981).

The genome encodes a single polypeptide that is post-translationally cleaved by viral proteases (2A^{pro} and 3C^{pro}/3CD^{pro}) into mature functional proteins (De Jesus, 2007). These include:

- Capsid proteins (VP1–VP4): Structural components of the virion
- 2A^{pro}, 3C^{pro}, 3CD^{pro}: Proteases essential for polypeptide processing
- 3D^{pol}: RNA-dependent RNA polymerase responsible for genome replication
- VPg (3B): A small protein primer for RNA synthesis
- 2B, 2C, 3A, 3AB: Proteins involved in replication complex formation

The 5' untranslated region (UTR) of the poliovirus genome is exceptionally long (~700 nucleotides) and highly structured, containing an internal ribosome entry site (IRES) that allows cap-independent translation of the viral RNA (Jang et al., 1988). This feature enables the virus to hijack the host cell's translation machinery while inhibiting cellular protein synthesis (Attardi & Smith, 1962).

➤ Serotypes and Antigenic Variation

Poliovirus exists as three distinct serotypes: PV-1, PV-2, and PV-3. These serotypes are defined by antigenic differences in the capsid proteins, particularly VP1 (Jiang et al., 2007). Infection with one serotype does not confer immunity against the others; however, secondary infections with the same serotype are extremely rare (Carter & Saunders, 2007).

Table 1: Characteristics of Poliovirus Serotype

Serotype	Virulence	Last Wild Case	Eradication Status
Type 1	Most virulent	2024 (endemic)	Not eradicated
Type 2	Least virulent	1999 (India)	Declared eradicated (2015)
Type 3	Intermediate	2012 (Nigeria)	Declared eradicated (2019)

The mutation rate of poliovirus is relatively high, with a synonymous substitution rate of approximately 1.0×10^{-2} substitutions/site/year (Jorba et al., 2008). This genetic variability has implications for vaccine development and surveillance, as it necessitates continuous monitoring of circulating strains.

III. PATHOGENESIS

➤ Cellular Entry and Replication

Poliovirus infection begins with binding of the viral capsid to the poliovirus receptor (PVR), also known as CD155, an immunoglobulin-like receptor expressed on the surface of human cells (Mendelsohn et al., 1989). CD155 is found only on human cells and those of higher primates, explaining poliovirus's strict host specificity (Suzuki, 2006).

Following receptor binding, the virus is internalised via receptor-mediated endocytosis. A conformational change in the capsid facilitates the release of the viral RNA into the host cell cytoplasm (Brandenburg et al., 2007). Once released, the positive-sense RNA genome serves directly as mRNA for translation of the viral polypeptide by the host cell's ribosomes, guided by the IRES element (Pelletier & Sonenberg, 1988).

Replication occurs in cytoplasmic replication complexes. The viral RNA-dependent RNA polymerase (3D^{pol}) synthesises negative-sense RNA intermediates, which serve as templates for the production of multiple positive-sense progeny genomes (Louten, 2016). The VPg protein acts as a primer for both negative- and positive-strand synthesis (Murray & Barton, 2003). Assembly of progeny virions occurs in the cytoplasm, and cell lysis releases up to 10,000 infectious particles per infected cell (Jiang et al., 2007).

➤ Routes of Infection

Poliovirus is transmitted via the faecal-oral route, primarily through ingestion of contaminated food or water, or via person-to-person contact (WHO, 2014). The virus can also be transmitted through the oral-oral route via droplets or aerosols (ECDC, 2024). Infected individuals are most infectious from 7–10 days before to 7–10 days after symptom onset, but virus excretion in faeces can persist for up to six weeks (CDC, 2024).

Upon ingestion, the virus first replicates in the oropharyngeal and intestinal mucosa, followed by spread to regional lymph nodes. A primary viraemia occurs within 3–5 days, during which the virus disseminates to other tissues, including brown fat, reticuloendothelial tissue, and muscle.

➤ Neuroinvasion and Paralytic Disease

In less than 1% of infections, the virus enters the central nervous system (CNS) and replicates in motor neurons of the anterior horn cells of the spinal cord, brainstem, or motor cortex (Racaniello, 2006). The precise mechanism of CNS entry remains incompletely understood, but three hypotheses have been proposed (Ohka & Nomoto, 2001):

- Direct crossing of the blood-brain barrier via transient viraemia
- Retrograde axonal transport from infected peripheral tissues
- Transport within infected monocytes or macrophages (the "Trojan horse" mechanism)

Once inside motor neurons, the virus replicates extensively, causing inflammation, necrosis, and eventual destruction of these cells. This results in the characteristic flaccid paralysis, which is typically asymmetric and most commonly affects the lower limbs (Gómez Yafal et al., 1993). The extent of paralysis depends on the number and location of motor neurons destroyed (Mueller & Wimmer, 2003).

➤ Immune Evasion

- *Poliovirus Employs Several Strategies to Evade the Host Immune System (Racaniello, 2006):*
- ✓ Receptor binding sites in "canyons": The receptor-binding sites are located in deep "canyons" on the virion surface, which are too narrow for antibody access, protecting them from immune recognition (Carter & Saunders, 2007).
- ✓ Rapid replication: The virus replicates extremely quickly, overwhelming the host immune response before effective antibodies can be generated (Kew et al., 2005).
- ✓ Acid resistance: The viral capsid protects the RNA from the acidic environment of the stomach, allowing survival during gastrointestinal transit (Carter & Saunders, 2007).

Immunity following natural infection or vaccination involves both humoral and mucosal components. Secretory IgA antibodies in the gastrointestinal tract block viral replication, while serum IgG and IgM antibodies prevent spread to the CNS (Carter & Saunders, 2007).

IV. CLINICAL FEATURES

➤ *Spectrum of Disease*

The clinical manifestations of poliovirus infection range widely, from asymptomatic to life-threatening paralysis (Mueller et al., 2005).

Table 2: Clinical Spectrum of Poliovirus Infection

Category	Frequency	Characteristics
Asymptomatic	~75%	No symptoms
Abortive polio	~25%	Fever, sore throat, headache, nausea (2–3 days)
Nonparalytic polio	~1%	Meningitis symptoms: neck stiffness, photophobia, muscle pain
Paralytic polio	<1%	Asymmetric flaccid paralysis, most commonly of lower limbs
Bulbar polio	Rare	Paralysis of muscles innervated by cranial nerves; dysphagia, respiratory failure

➤ *Abortive and Nonparalytic Polio*

Abortive polio, occurring in approximately 25% of infections, presents as a mild, self-limited febrile illness with symptoms including fever, headache, malaise, sore throat, and gastrointestinal disturbances (He et al., 2003). Recovery is typically complete within 1–2 weeks (Mayo Clinic, 2024).

Nonparalytic polio (aseptic meningitis) affects about 1% of infected individuals. It presents with more pronounced symptoms, including severe headache, photophobia, neck and back stiffness, muscle pain, and hyperesthesia (Dunnebacke et al., 1969). While uncomfortable, nonparalytic polio does not result in permanent neurological deficits (CDC, 2024).

➤ *Paralytic Polio*

Paralytic poliomyelitis occurs in less than 1% of infections and represents the most severe form of the disease (WHO, 2014). The risk of paralysis increases with age; children under 5 are most affected, but adults have a higher risk of severe paralysis (GPEI, 2024).

- The disease typically progresses through three phases (Carter & Saunders, 2007):
- ✓ Minor illness phase (1–3 days): Fever, headache, and gastrointestinal symptoms
- ✓ Symptomatic period (1–3 days): An apparent recovery
- ✓ Major illness phase (paralytic phase): Fever returns, followed rapidly by asymmetrical, flaccid paralysis

Paralysis usually develops within 1–10 days of symptom onset and progresses for 2–4 days before stabilising (Jiang et al., 2007). The peak of paralysis occurs when fever subsides (Gómez Yafal et al., 1993). Muscles are typically weak, floppy, and poorly controlled, with reduced or absent deep tendon reflexes. Sensation remains intact (Wright et al., 2014).

➤ *Types of Paralytic Polio:*

- Spinal polio: Most common form, affecting motor neurons in the spinal cord. Paralysis is typically asymmetric and may involve one or more limbs, with lower limbs most frequently affected (Gómez Yafal et al., 1993).

- Bulbar polio: Affects the motor neurons of the brainstem, particularly the medulla oblongata. This results in paralysis of muscles innervated by cranial nerves, causing dysphagia, nasal speech, pooling of secretions, and dyspnea (Mueller et al., 2005). Bulbar polio has a high mortality rate.

- Bulbospinal polio: A combination of spinal and bulbar involvement, often the most severe form (Carter & Saunders, 2007).

➤ *Complications*

- Respiratory failure: Paralysis of the intercostal muscles and diaphragm impairs breathing. Historically, patients with respiratory paralysis were placed in "iron lungs" for respiratory support (Mayo Clinic, 2024). Even with modern ventilatory support, mortality among patients with respiratory involvement remains 5–15% (WHO, 2014).
- Post-polio syndrome (PPS): This is a poorly understood condition affecting polio survivor's decades after their initial infection. It is characterised by new-onset muscle weakness, fatigue, pain, and progressive muscle atrophy (ECDC, 2024). PPS is not infectious and does not involve viral replication but is thought to result from the degeneration of motor neurons that had previously compensated for initial damage (CDC, 2024).

V. EPIDEMIOLOGY

➤ *Historical Epidemiology*

In the pre-vaccine era, polio was a global disease causing devastating epidemics, particularly in industrialized countries (Paul, 1971). In the United States, annual cases of paralytic polio peaked in 1952 with 57,623 reported cases (Mayo Clinic, 2024). The disease primarily affected children, leading to its common name "infantile paralysis" (Ryan et al., 2004).

The epidemiology of polio shifted as sanitation improved. Paradoxically, better hygiene delayed initial infection to older ages, when the risk of paralytic disease was higher, leading to more severe epidemics (De Jesus, 2007).

➤ *Global Polio Eradication Initiative (GPEI)*

The Global Polio Eradication Initiative (GPEI) was launched in 1988 by the World Health Assembly, following the success of Rotary International's PolioPlus program (GPEI, 2024). At that time, polio paralysed more than 1,000 children worldwide every day, and the disease was endemic in more than 125 countries (GPEI, 2024).

The GPEI has Achieved Remarkable Progress:

Year	Estimated Cases	Countries Endemic
1988	350,000	125+
2000	800	30
2010	1,352	4
2020	140	2
2022	30	2

➤ *Current Status*

• *The 2014 Milestone: South-East Asia Region Certified Polio-Free*

On 27 March 2014, the World Health Organization (WHO) South-East Asia Region was officially certified polio-free by an independent commission. This was a historic achievement: the region, home to 1.8 billion people across 11 countries (including India, Indonesia, Bangladesh, and Thailand), became the fourth of six WHO regions to be certified. This success was particularly significant because India was once considered the most challenging place to end polio, having recorded its last case in January 2011. The certification represented the culmination of 17 years of effort, during which the polio programme conducted 189 nationwide campaigns and administered over 13 billion doses of oral polio vaccine.

However, the 2014 announcement was accompanied by a clear warning: progress was at risk unless polio was also stopped in the three remaining endemic countries: Afghanistan, Pakistan, and Nigeria.

Nigeria's Journey: From Endemic Status to Certification

Nigeria's path to certification was long and marked by significant setbacks, reflecting the complex challenges of reaching every child in a vast and diverse country.

• *The Road to Wild Poliovirus Eradication*

Eradication efforts in Nigeria began in earnest in 1996, but progress was hampered by multiple obstacles. A major crisis occurred in 2003 when a vaccine boycott in several northern states, fueled by unfounded rumors that the vaccine caused infertility, led to a dramatic resurgence of the disease. At one point, Nigeria was reporting an average of three new polio cases per day and became a primary exporter of the virus to other countries, including as far as Yemen and Indonesia.

Despite these challenges, a turning point came with the establishment of Emergency Operations Centres (EOCs), which allowed for real-time data analysis and rapid response. The engagement of traditional, religious, and civil society leaders also proved critical in building trust and increasing vaccine acceptance. These efforts led to a major milestone: on 25 September 2015, WHO removed Nigeria from the list of polio-endemic countries, as the country had gone a full 12 months without a case of wild poliovirus (the last case recorded on 24 July 2014). This was the first time Nigeria had successfully interrupted wild poliovirus transmission.

However, the victory was not yet complete. In 2016, after two years without a case, four new wild polio cases were reported, a devastating setback that delayed certification plans. This demonstrated the fragility of progress and the need for sustained vigilance.

• *Certification and the "Current Status"*

Nigeria's perseverance paid off. After years of sustained high-quality surveillance and vaccination campaigns with no new wild poliovirus cases, the African Regional Certification Commission for Polio Eradication (ARCC) declared Nigeria and the entire WHO African Region free of wild poliovirus on 25 August 2020.

The Current Challenge: Circulating Variant Poliovirus (cVDPV2)

The "current status" of polio in Nigeria (as of 2025/2026) is a story of "winning a battle, but the war is still on," as Professor Oyewale Tomori, the chair of Nigeria's Expert Review Committee on Polio Eradication, put it. While Nigeria is certified wild poliovirus-free, it continues to face outbreaks of a different form of the disease: circulating vaccine-derived poliovirus type 2 (cVDPV2).

cVDPV2 is a rare but serious consequence of using the live-attenuated oral polio vaccine (OPV). In communities with low immunization coverage, the weakened virus in the vaccine can circulate for a long time, mutate, and regain the ability to cause paralysis in children who are not vaccinated. This remains a significant public health threat, with 66 cases of cVDPV2 reported from 44 Local Government Areas in 12 states as of October 2025.

• *Key Strategic Responses:*

- ✓ Novel Oral Polio Vaccine (nOPV2): Nigeria has been a key adopter of the newer, more genetically stable nOPV2 to respond to these outbreaks. This vaccine is less likely to mutate into a dangerous form, representing a significant technological advancement.
- ✓ Integrated Surveillance: The country maintains a robust system of both Acute Flaccid Paralysis (AFP) surveillance and environmental surveillance (testing sewage water). The

National Polio Laboratory in Ibadan analyzes over 23,000 stool samples annually to track potential transmission.

- ✓ Renewed Commitment (by 2030): In 2025, the Nigerian government and partners like WHO reaffirmed their

commitment to ending all forms of polio by 2030, with large-scale vaccination campaigns aiming to immunize over 106 million children.

Table 3: Nigeria's Progress Toward Polio Eradication

Milestone	Status
WHO South-East Asia Region certified polio-free	Achieved 27 March 2014
Nigeria removed from polio-endemic list	Achieved 25 September 2015 (after 12 months without a case)
Nigeria & WHO African Region certified wild polio-free	Achieved 25 August 2020
Current Status: Circulating Variant Poliovirus Type 2 (cVDPV2)	Active challenge; 66 cases reported as of October 2025
Target for Ending All Forms of Polio	2030

Nigeria's success in eradicating wild poliovirus is a monumental public health achievement, but the persistence of cVDPV2 underscores the need for a continuous "risk-management mindset". Sustaining vaccination coverage, strengthening surveillance, and maintaining rapid outbreak response capabilities are essential to protecting children and securing a permanently polio-free future.

Table 4: WHO Region Certification Status

WHO Region	Certification Year	Status
Americas	1994	Polio-free
Western Pacific	2000	Polio-free
Europe	2002	Polio-free
South-East Asia	2014	Polio-free
Africa	2020	Polio-free
Eastern Mediterranean	—	Not certified

• Vaccine-Derived Polioviruses (VDPVs)

Circulating vaccine-derived polioviruses (cVDPVs) represent a significant challenge to the eradication endgame. These arise when the attenuated viruses in OPV revert to virulence through genetic mutations, particularly in under-immunized populations where the virus can circulate for extended periods (Kew et al., 2005).

• cVDPVs are classified as follows (GPEI, 2024):

- ✓ Type 1 cVDPVs: Most common, associated with outbreaks in several African countries
- ✓ Type 2 cVDPVs: Increasingly common, linked to areas with low OPV coverage
- ✓ Type 3 cVDPVs: Less frequent

The number of cVDPV outbreaks has increased in recent years, emphasising the need for improved vaccination coverage and enhanced surveillance (ECDC, 2024).

VI. VACCINATION

➤ Development of Polio Vaccines

The development of polio vaccines represents one of the greatest scientific achievements of the 20th century (Sabin, 1956).

➤ Inactivated Poliovirus Vaccine (IPV):

Developed by Jonas Salk in 1955, IPV consists of formalin-inactivated wild-type poliovirus strains (Mayo Clinic, 2024). The vaccine is administered via intramuscular injection and contains all three serotypes. IPV induces robust humoral immunity (serum IgG) but provides limited mucosal immunity in the gastrointestinal tract (Carter & Saunders, 2007). Since 2000, IPV has been the standard of care in the United States and many other developed countries (CDC, 2024).

➤ Oral Poliovirus Vaccine (OPV):

Developed by Albert Sabin in 1961, OPV contains live-attenuated strains of all three poliovirus serotypes (Sabin, 1956). Administered orally, OPV induces both systemic and mucosal immunity, providing superior protection against wild-type infection in the gut (Carter & Saunders, 2007). This characteristic, combined with low cost and ease of administration, made OPV the cornerstone of the GPEI (GPEI, 2024).

Table 5: Comparison of IPV and OPV

Feature	IPV	OPV
Route	Injection	Oral
Immune response	Humoral only	Humoral and mucosal
Intestinal immunity	Limited	Strong
Cost	Higher	Lower
Risk of VAPP/VDPV	None	Rare
Ease of administration	Requires trained personnel	Simple

➤ *Global Immunization Strategies*

➤ *The GPEI has utilised a multi-pronged immunization approach (GPEI, 2024):*

- Routine immunization: Integration of polio vaccine into national immunization schedules
- Supplementary immunization activities (SIAs): Mass vaccination campaigns targeting all children under 5
- National Immunization Days (NIDs): Synchronised campaigns reaching millions of children
- Surveillance: Active case finding and laboratory confirmation

➤ *Endgame Strategy*

The Polio Eradication Endgame Strategy has several key components (WHO, 2014):

- *Phased Withdrawal of OPV:*
- ✓ 2016: Switch from trivalent OPV (tOPV) to bivalent OPV (bOPV, types 1 and 3) globally
- ✓ Withdrawal of type 2 OPV

- *Introduction of IPV:*
- ✓ Routine administration of at least one dose of IPV in all countries
- ✓ IPV-only schedules in the post-eradication era

- *Enhanced Surveillance:*
- ✓ AFP surveillance
- ✓ Environmental surveillance
- ✓ Laboratory network strengthening

➤ *Vaccine Safety and Adverse Events*

• *Vaccine-Associated Paralytic Polio (VAPP):*

This occurs rarely (approximately 4 cases per 1,000,000 live births in OPV-using countries) in vaccine recipients or their contacts. The incidence is highest after the first dose (Kew et al., 2005).

• *Vaccine-Derived Polioviruses (VDPVs):*

As discussed above, these represent a rare but significant risk. Their occurrence is associated with low immunization coverage, allowing prolonged viral circulation and reversion to neurovirulence (GPEI, 2024).

• *Allergic Reactions:*

IPV contains trace amounts of streptomycin, polymyxin B, and neomycin; individuals allergic to these antibiotics may experience reactions (Mayo Clinic, 2024).

VII. SURVEILLANCE AND DIAGNOSIS

➤ *Clinical Diagnosis*

Polio is suspected in any child presenting with acute flaccid paralysis (AFP) in whom other causes have been excluded (WHO, 2014). Clinical features include:

- Asymmetric flaccid paralysis
- Normal sensation
- Preservation of consciousness
- Progression to maximum paralysis within 48–72 hours
- Fever at onset

➤ *Laboratory Diagnosis*

• *Virus Isolation:*

Stool samples are the specimen of choice for poliovirus isolation, as viral shedding in faeces persists for weeks after infection. Throat swabs may be positive only during the first week (CDC, 2024).

• *Molecular Methods:*

RT-PCR is the gold standard for poliovirus detection and typing. It provides rapid, sensitive, and specific results (ECDC, 2024).

• *Serology:*

Detection of poliovirus-specific antibodies can confirm infection in previously unvaccinated individuals (WHO, 2014).

• *Acute Flaccid Paralysis (AFP) Surveillance*

AFP surveillance is the cornerstone of polio eradication (GPEI, 2024). Key performance indicators include:

- ✓ Sensitivity: ≥ 1 AFP case per 100,000 children under 15 years annually
- ✓ Timeliness: $\geq 80\%$ of cases have adequate stool samples within 14 days of paralysis onset
- ✓ Completeness: $\geq 80\%$ of AFP cases investigated

• *Environmental Surveillance*

Testing of wastewater for poliovirus complements AFP surveillance by detecting virus circulation in communities (ECDC, 2024). This is particularly important in:

- ✓ Areas with low AFP surveillance quality
- ✓ Dense urban populations
- ✓ Regions with suspected persistent transmission
- ✓ Areas with high population movement

VIII. CHALLENGES TO ERADICATION

➤ *Operational Challenges*

• *Reaching Every Child:*

Insecurity, particularly in parts of Afghanistan, Pakistan, and previously in Nigeria, has hindered vaccination campaigns (GPEI, 2024). Attacks on health workers, cultural barriers, and geographic isolation remain significant obstacles.

- *Improving Surveillance:*

In areas with poor healthcare infrastructure, AFP surveillance may be inadequate, allowing undetected viral transmission (WHO, 2014).

- *Closing Immunity Gaps:*

Routine immunization coverage remains suboptimal in many regions, creating pockets of susceptible individuals (CDC, 2024).

➤ *Biological Challenges*

- *VDPV Emergence:*

As discussed, cVDPVs pose a continuing threat, particularly in areas with low OPV coverage. The risk of cVDPV outbreaks increases during the transition from OPV to IPV (Kew et al., 2005).

- *Chronic Carriers:*

Rare individuals with immunodeficiency can become chronic asymptomatic carriers, excreting VDPVs for years (GPEI, 2024).

- *Timing of Eradication Declaration:*

Eradication must be definitively confirmed through rigorous surveillance before vaccination can be withdrawn. Three years without WPV isolation in the presence of adequate surveillance are required for certification (WHO, 2014).

IX. TREATMENT AND MANAGEMENT

➤ *Supportive Care*

There is no specific antiviral treatment for polio; management is supportive (Mayo Clinic, 2024):

Intervention	Purpose
Bed rest	Reduce energy expenditure
Pain management	NSAIDs, analgesics for muscle pain
Hot moist packs	Control muscle spasms
Physical therapy	Prevent contractures and maintain function
Splints/braces	Maintain proper alignment
Ventilatory support	For respiratory paralysis
Nutritional support	Adequate calorie and fluid intake

➤ *Polio Syndrome Management*

Management of PPS focuses on (ECDC, 2024):

- Energy conservation strategies
- Pain management
- Assistive devices (braces, walkers, wheelchairs)
- Respiratory support
- Psychological support

- Pulmonary rehabilitation

➤ *Outbreak Response*

Any single case of polio is considered an outbreak requiring urgent response (GPEI, 2024):

- Immediate notification to WHO within 24 hours (per IHR 2005)
- Rapid investigation (within 48 hours)
- Supplementary immunization activities in affected and surrounding areas
- Enhanced surveillance to determine the extent of transmission

X. RECOMMENDATIONS

Based on the comprehensive review, the following recommendations are made:

➤ *For Parents and Communities:*

- Ensure complete childhood vaccination according to national schedules
- Seek early medical attention for any child with fever and weakness
- Maintain good hygiene practices, including safe water and sanitation
- Support community health workers and vaccination campaigns

➤ *For Healthcare Providers:*

- Maintain high vigilance for AFP cases and report immediately
- Strengthen routine immunization services
- Educate communities about polio and vaccine safety
- Participate in supplementary immunization activities

➤ *For Policymakers:*

- Increase funding for polio eradication activities
- Strengthen healthcare infrastructure in underserved areas
- Improve surveillance systems for early detection
- Develop strategies to address insecurity and access barriers
- Plan for the post-eradication era, including IPV transition

➤ *For International Organizations:*

- Continue technical and financial support for endemic countries
- Enhance environmental surveillance capabilities
- Develop novel vaccines and therapeutic options
- Support research on chronic poliovirus excretion

XI. CONCLUSION

Poliomyelitis, caused by poliovirus, has been a major cause of childhood disability for millennia. The development of effective vaccines transformed this once-dreaded disease into a potentially eradicable condition. The Global Polio Eradication

Initiative has achieved remarkable success, reducing global incidence by over 99% and eradicating wild poliovirus types 2 and 3.

However, the final steps of eradication present significant challenges. The emergence of vaccine-derived polioviruses in under-immunized populations, security issues hindering vaccination in endemic areas, and the need to transition from OPV to IPV without creating immunity gaps all require careful planning and implementation.

The eventual eradication of polio will represent one of the greatest public health achievements in history. Until that day arrives, sustained commitment from governments, international organizations, healthcare workers, and communities is essential. Every child vaccinated brings us one step closer to a polio-free world.

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CONFLICT OF INTEREST

The authors declare no conflict of interest.

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