Opinions and Recommendations of the Pediatric Infectious Diseases and Immunization Society

DOI: 10.5578/ced.20250412 • *J Pediatr Inf 2025;19(4):e285-e291*

Poliovirus from A to Z: A Journey from History to the Present Day, Epidemiology, Genetics, Pathogenesis, Nomenclature Terminology, Clinical Features, and Vaccination

A'dan Z'ye Poliovirüs: Tarihten Günümüze Yolculuk, Epidemiyoloji, Genetik, Patogenez, İsimlendirme Terminolojisi, Klinik Özellikler ve Aşılama

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Cite this article as: Yeşil E, Önal P, Özpınar Ş, Çomruk A, Seyhanlı D, Özdemirer Ü, et al. Poliovirus from a to z: A journey from history to the present day, epidemiology, genetics, pathogenesis, nomenclature terminology, clinical features, and vaccination. J Pediatr Inf 2025;19(4):e285-e291.

Abstract

Objective: Poliovirus is an enterovirus consisting of three serotypes that has caused serious epidemics throughout history. With the widespread use of vaccines, the global incidence has decreased significantly, and types 2 and 3 have been eradicated. The only subtype currently circulating is wild poliovirus type 1. Today, wild poliovirus is found only in Afghanistan and Pakistan, while cVDPV2 (circulating vaccine-derived poliovirus type 2) is the most common type worldwide. While most infections are asymptomatic or mild, a very small proportion can lead to paralytic polio. Diagnosis is made with stool samples in cases of acute flaccid paralysis, and treatment is supportive. Oral and inactivated polio vaccines are used to prevent the disease, and novel oral polio vaccine type 2 is used for cVDPV2 outbreak control when necessary.

Keywords: Vaccine, child, paralysis, poliovirus

Öz

Giriş: Poliovirüs, üç serotipten oluşan ve tarih boyunca ciddi salgınlara neden olmuş bir enterovirüstür. Aşıların yaygın kullanımıyla küresel insidans büyük ölçüde azalmış; Tip 2 ve Tip 3 eradike edilmiştir. Şu an dolaşımda olan tek alt tür, vahşi poliovirüs Tip 1'dir. Günümüzde vahşi poliovirüs yalnızca Afganistan ve Pakistan'da görülmekte olup, dünya genelinde en sık cVDPV2 (dolaşımda olan aşı türevi poliovirüs Tip 2) görülmektedir. Enfeksiyonların çoğu asemptomatik veya hafif seyrederken, çok küçük bir kısmı paralitik polioya yol açabilir. Tanı, akut flask paralizi vakalarında dışkı örnekleriyle konur; tedavi destekleyicidir. Hastalığın önlenmesinde oral ve inaktif polio aşıları ve gerektiğinde cVDPV2 salgın kontrolü için novel oral polio aşısı Tip 2 kullanılmaktadır.

Anahtar Kelimeler: Aşı, çocuk, paralizi, poliovirüs

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Received: 03.12.2025 **Accepted:** 21.12.2025
Available Online Date: 25.12.2025

History of Poliomyelitis

The history of poliomyelitis dates back to ancient times, with the earliest depictions found in Egyptian paintings and carvings from 1403-1365 BC, showing small children walking with canes and people having difficulty using their limbs (1). The first clinical description was made in 1789 by Michael Underwood, who referred to it as "weakness of the lower extremities." The clinical features of the disease were subsequently detailed by Jacob Heine in 1840 and Carl Oscar Medin in 1890, leading to it being named "Heine-Medin Disease"(2,3).

In 1905, Ivan Wickman predicted that the disease could be caused by an infectious agent, and in 1908, Karl Landsteiner and Erwin Popper determined that this infectious agent was a microorganism smaller than a bacterium (3). However, the poliovirus could not be seen until the electron microscope was invented. In 1948, John Enders, Thomas Weller, and Frederick Robbins succeeded in producing the poliovirus in living cells and were awarded the Nobel Prize in 1954 (3).

In 1955, Dr. Jonas Salk developed the first inactivated polio vaccine (IPV), followed by the live oral polio vaccine (OPA) developed by Dr. Albert Sabin in 1961 (3,4). The first outbreak of poliomyelitis occurred in 1894 in the Vermont region of the United States. More than 130 people were affected by this outbreak (5). In 1916, a major polio outbreak occurred in New York, and respiratory support devices known as "Iron Lungs" came into use during this period (4).

In 1988, the World Health Assembly established the Global Polio Eradication Initiative (GPEI). Created to stop the global circulation of poliovirus and increase vaccination rates worldwide, the GPEI is the most comprehensive infectious disease eradication initiative in history (3).

Epidemiological Characteristics of Poliovirus in World Health Organization Regions and Türkiye

Poliomyelitis primarily affects children under the age of five. According to the World Health Organization (WHO), even a single infected child poses a risk to all children worldwide. Therefore, effective surveillance and an effective immunization system are of vital importance in the global fight against the disease (6).

Before the introduction of vaccination, poliovirus affected almost all children. With the introduction of OPA and IPA, there was a rapid decline in the incidence of polio (7). Wild poliovirus (WPV) type 1 has historically been the primary cause of most polio cases worldwide and is currently the only subtype in circulation. WPV1 is still endemic in Pakistan and Afghanistan (3). The World Health Organization declared WPV2 eradicated in 2015 and WPV3 eradicated in 2019 (7).

Polio status in the Americas Region

Polio case rates in the United States dropped by 99% just six years after the first poliovirus vaccine was introduced. The last WPV case in the United States was reported in 1979. The last known case was a three-year-old child living in northern Peru in 1991. In 1994, the WHO Americas Region was declared polio-free (7,8). Based on the success in the Americas, the World Health Organization decided to eradicate WPV worldwide by 2000 as part of the Global Polio Eradication Initiative (3,7). With the launch of the Global Polio Eradication Initiative in 1988, approximately 350.000 poliovirus cases in more than 125 endemic areas declined to just 140 cases in 2020, occurring in only two countries (Afghanistan and Pakistan) (3). Although eradication is not yet possible, the inclusion of the polio vaccine in routine childhood immunization programs has resulted in a reduction of more than 99% in WPV cases worldwide since 1988 (3).

Polio status in the West Pacific Region

In 2000, it was announced that polio had been officially eradicated in 37 Western Pacific countries, including China and Australia. The last case of WPV in the Western Pacific region was a 15-month-old girl living near Cambodia in 1997 (3).

The European Region and Türkiye

Türkiye adopted the goal of eradicating polio by the year 2000 under the Global Polio Eradication Initiative in 1989, and significant progress has been made toward achieving this goal since then (9). Türkiye organized six National Immunization Days (NID) in 1995, 1996, 1997, 1998, 1999, and 2000 as part of the MECACAR Operation (Mediterranean, Caucasus, and Central Asian Republics). Within the scope of this operation, NIDs were synchronized in 18 geographically neighboring countries (9,10).

A total of 500 million children were vaccinated in 100 countries within the scope of the European region polio eradication program (3).

In 1997, a total of 141 acute flaccid paralysis (AFP) cases were reported from Türkiye, and 6 AFP cases were confirmed as polio with WPV1 isolation. Most recently, in 1998, 26 AFP cases were confirmed as polio with WPV1 isolation. No poliovirus cases have been isolated since then. These virologically confirmed cases were seen in patients from the province of Mardin. All six patients were between 9 months and 2 years of age; four patients were unvaccinated, and two had received only one dose of OPA (9,10).

Until 1997, clinical classification was used in our country. From this year onwards, virological classification was adopted, and to date, no WPV has been detected in stool samples collected for AFP surveillance (10).

In June 2002, the WHO European Region, including our country, was declared free of poliovirus. The last indigenous wild poliovirus case in this region was a 33-month-old unvaccinated child (Melik Minas) identified in Patnos/Ağrı, Türkiye, in November 1998.

Southeast Asia Region

On March 27, 2014, the WHO certified that the Southeast Asia Region had been eradicated of polio, meaning that wild poliovirus transmission had been stopped in this block of 11 countries stretching from Indonesia to India. The last case of polio in the region was detected in India in January 2011. With the addition of this region, it was accepted that 80% of the world's population now lives in polio-free areas (3,7).

African Region

The last case of WPV3 was reported in Nigeria in 2012 (3). In 2020, Africa became the fifth region to be certified polio-free (11). Both cases of polio detected in Malawi in February 2022 and Mozambique in May 2022 were traced back to Pakistan (12).

East Mediterranean Region

The endemic spread of wild poliovirus continues in Afghanistan and Pakistan, located in the Eastern Mediterranean region (3). In 2018, there were 21 cases of wild poliovirus in Afghanistan and 12 in Pakistan, while in 2020, this number rose to 56 cases in Afghanistan and 84 in Pakistan. While there were 6 cases each in Afghanistan and Pakistan in 2023, by 2024, the number of cases had risen again to 25 in Afghanistan and 74 in Pakistan (13).

When polio re-enters a country, the spread must be monitored and stopped within 12 months for that country to maintain its polio-free status. Failure to stop polio in these last remaining areas could lead to a global resurgence of the disease. Therefore, ensuring the complete and definitive eradication of polio is of critical importance.

Furthermore, in Syria, located in this region, 36 cases of wild poliovirus were detected in 2013 as a result of the deterioration of primary health care services and reduced vaccination coverage due to the internal unrest that began in 2011. Two cases were also reported from Iraq. In 2017, an outbreak caused by vaccine-derived poliovirus was confirmed in eastern Syria. These are the first outbreaks seen in Syria since 1999 (14).

Genetic Characteristics of Poliovirus and Pathogenesis

Polioviruses are classified as members of the Enterovirus C type within the Enterovirus genus of the Picornaviridae family, and there are three serotypes of polioviruses. These are non-enveloped, positive-sense, single-stranded RNA viruses that are stable under moderate environmental conditions.

The capsid of picornaviruses is an icosahedral structure composed of 60 protomers. Each protomer contains the structural proteins VP1, VP2, VP3, and VP4; VP1–VP3 are located on the surface, forming the antigenic properties, while VP4 is located inside the capsid. In mature poliovirus, the cleavage of the VP0 precursor into VP2 and VP4 is typical. The capsid proteins are built upon an eight-chain β-barrel core, and differences in the surface rings contribute to antigenic diversity among species (1).

The VP1–VP4 proteins are the structural proteins that form the capsid. The VP1 protein plays a critical role in the virus's binding to the host cell receptor (CD155) and in being the target of neutralizing antibodies. The VP1 gene is largely responsible for genetic diversity in poliovirus. Poliovirus binds to the host cell surface by binding to its own receptor, CD155 (15).

The replication of all picornaviruses occurs in the cell cytoplasm. The first step is binding to a cell surface molecule. After picornaviruses bind to their cellular receptors, the viral capsid is brought into the cell by endocytosis, and then the genome is released into the cytoplasm, where picornavirus replication occurs. When the positive-stranded viral RNA enters the cytoplasm, it is translated to provide the viral proteins necessary for genome replication and the production of new virus particles. The newly synthesized positive-stranded RNA combines with pentamers to form the infectious virus (16). The RNA functions directly like mRNA; that is, it undergoes translation immediately after entering the cell.

The nucleotide sequence in the VP1 region of the wild poliovirus has been shown to differ by more than 15% from the Sabin strain. The first poliovirus genotypes were also defined based on a difference of more than 15% in the 150-nucleotide region at the VP1/2A junction. Furthermore, it has been shown that wild-type polioviruses evolve during replication in healthy carriers, and the difference in VP1 can progress to 2.2% in six months (17).

In vitro, poliovirus replicates only in primate cells (human or primate). The viral cycle of poliovirus occurs entirely in the cytoplasm of the host cell. This cycle is one of the fastest known viral cycles and takes approximately eight hours at 37 °C in cell culture (18).

In poliovirus infection, the virus is taken orally and begins to replicate in the oropharyngeal and intestinal mucosa (19). The incubation period is usually 7–14 days (can vary from 2–35 days). Poliovirus replicates in gastrointestinal lymphatic tissues, and mild transient viremia occurs in most individuals. The virus spreads to systemic reticuloendothelial tissues (15). In humans, the CD155 protein has been detected in the intestinal epithelium, in the M cells of Peyer's patches, and in the germinal centers within Peyer's patches. Although the

mechanism by which poliovirus reaches the central nervous system is not fully understood, three possible routes have been identified. The first pathway involves the virus crossing from the blood into the central nervous system during viremia and neuroinvasion through the blood-brain barrier. The second pathway involves the virus infecting peripheral nerve endings and progressing toward the motor neurons of the anterior horn of the spinal cord via retrograde axonal transport. The third possible route is the transport of the virus to the central nervous system by infected monocytes or macrophages via a "Trojan horse" mechanism (20). Each of these mechanisms helps explain how the poliovirus can, albeit rarely, reach motor neurons and cause paralytic poliomyelitis (20).

In 4-8% of individuals, secondary, more pronounced viremia occurs and minor disease symptoms appear. Neurological symptoms are rare, with poliomyelitis developing in less than 1% of infected individuals. Therefore, poliomyelitis is a rare and complicating event that is not essential for poliovirus replication (15).

In neurological involvement, the poliovirus replicates within neurons; the most commonly affected region is the anterior horn cells of the spinal cord. Neurons in the medulla nuclei, cerebellar vermis, midbrain, thalamus and hypothalamus, pallidum, and motor cortex of the cerebrum may also be affected. Serum neutralizing antibodies develop approximately one week later and protect against paralysis but do not provide protection against reinfection. Immunity to poliovirus infection is serotype-specific, and there is no cross-protection. Immunity lasts a lifetime (5). The mechanisms by which poliovirus causes poliomyelitis are still not fully understood, and our knowledge largely comes from primate and mouse experiments (18).

Naming of Poliovirus Strains

Wild type poliovirus: It is the natural type of poliovirus that circulates naturally and historically causes paralytic polio in humans. Its genetic difference from the Sabin strain VP1 region is over 15%.

Vaccine-associated paralytic poliovirus (VAPP): It occurs as a side effect following OPA administration. It is a rare case of paralytic poliomyelitis that appears 4-40 days after OPA administration and lasts at least 60 days. Recipient VAPP may develop in the person who has just received the vaccine, and contact VAPP may develop in unvaccinated or immunocompromised individuals in the immediate environment who are exposed to the virus excreted from the body (21).

VAPP is a virus that has undergone a genetic change from the Sabin (OPA) vaccine strain that is mild enough to cause paralysis (<1%). VAPP cases do not usually cause epidemics, and the virus's ability to spread within the community is limited. It is quite rare, occurring in approximately one case per 2.7 million OPA doses (22).

When examining the relationship between vaccine strain serotypes and VAPP, type 3 has been more frequently associated with recipient VAPP. Type 2 has been observed more frequently in immunocompromised individuals and in cases of contact-related VAPP, while type 1 has been less frequently associated with VAPP cases (22).

Vaccine Derivative Poliovirus (VDPV): VDPV, oral polio vaccine virus strains are genetically differentiated to a certain degree.

- For Type 1 and Type 3 VDPVs: Differentiation of more than 1% in the entire VP1 genomic region (or ≥10 nucleotide changes),
- For Type 2 VDPVs: Differentiation of more than 0.6% in the entire VP1 genomic region (or ≥6 nucleotide changes).

Circulating VDPV (cVDPV): VDPV isolates with evidence of person-to-person transmission in the community are referred to as cVDPV. Previously, if VDPV genetically linked to at least two AFP cases had been isolated, it was called cVDPV. In the new classification implemented to increase the sensitivity of surveillance, the following are required to refer to VDPV as "circulating":

Genetically linked isolated VDPVs;

1. Isolation from at least two individuals (not necessarily AFP cases) without direct (i.e., household) contact is defined as cVDPV,
2. Isolation from one individual and one or more environmental surveillance samples is defined as cVDPV,
3. If isolated from two or more environmental surveillance samples, it is considered cVDPV if these samples were collected from more than one different environmental surveillance collection area (non-overlapping catchment areas) or were collected from the same area but at times more than two months apart.

Immunodeficiency-Related VDPV (iVDPV): Referred to the viruses isolated from individuals with evidence of primary immunodeficiency.

Ambiguous (aVDPV): It is a VDPV isolated from individuals without evidence of circulation or known immunodeficiency, or from environmental samples. A VDPV isolate should only be classified as 'uncertain' after additional field investigations (to determine whether it is a cVDPV or iVDPV) have been ruled out. If genetically linked isolates are subsequently found, an isolate classified as aVDPV may need to be reclassified as cVDPV (23).

Global Status of cVDPV Strains

VDPV cases now outnumber wild poliovirus cases. Transmission is limited in communities with high vaccine coverage. However, in communities with low coverage where IPV has not yet been introduced, the attenuated virus can continue to circulate and cause outbreaks (3,7).

Paralysis associated with cVDPV is primarily seen in Sub-Saharan Africa and Asia. First detected in an outbreak on the island of Hispaniola (North America) in 2001, cVDPV is responsible for most of the polio cases seen today (7). Forty-nine cVDPV1 cases have been reported in Mozambique, the Democratic Republic of Congo, and Madagascar, while 296 cVDPV2 cases have been reported in 20 other countries (7).

In the Philippines, the virus was detected in a 3-year-old girl in 2019, leading to an outbreak declaration following a single case, which was declared over in June 2021 (24). In December 2021, acute poliomyelitis infection was confirmed in an infant in Borneo/Malaysia, followed by three more cases, the last of which was detected in January 2022. The WHO declared the outbreak over in September 2022. In both outbreaks, the cases were classified as cVDPV (24). Although the number of cases in 2023 (524) decreased compared to 2022 (881), cVDPV cases were geographically more widespread in 2023 (24,25).

In 2022, an unvaccinated young adult from New York presented with acute flaccid paralysis (AFP), and stool testing identified VDPV2. Environmental testing of samples taken before and after the onset of the patient's illness detected VDPV2 in wastewater in surrounding counties. The virus is thought to have originated in a country that administers OPV but where the population remains unvaccinated (7).

In addition to this case, a case of polio in a young child in Gaza, sporadic cases of WPV polio in Africa, and the discovery of cVDPV in wastewater in many countries, including the United Kingdom and Israel, underscore the importance of maintaining high vaccination rates in all communities until polio is eradicated globally (7).

Clinical Characteristics of Polioviruses

Clinical findings

Approximately 90-95% of poliovirus infections are asymptomatic. Minor illness occurs in 4–8% of cases and presents with nonspecific complaints such as fever, fatigue, sore throat, loss of appetite, myalgia, nausea-vomiting, and diarrhea. The incidence of non-paralytic polio (aseptic meningitis form) is 1%. It may present with fever, fatigue, paresthesia, nausea, vomiting, headache, back and leg pain, and signs of meningeal irritation. At this stage, a cerebrospinal fluid examination may reveal mild pleocytosis. Non-paralytic polio may resolve within 1-2 weeks or progress to paralytic polio in a low percentage of cases.

Paralytic polio develops in only 0.1–0.5% of infections, but it is the most clinically serious form due to the risk of permanent sequelae. The underlying mechanism in the pathogenesis is the involvement of the anterior horn motor neurons of the spinal cord. Patients appear alert and restless. Fever is higher than in the abortive form (minor illness), and the patient may have severe muscle pain. Shortly before actual muscle weakness appears, superficial and deep tendon reflexes on the affected side often decrease or disappear. Often, following the initial period of illness, there is an asymptomatic interval lasting several days, followed by the reappearance of symptoms leading to paralysis.

Clinically, it is typically characterized by sudden onset, asymmetric, flaccid paralysis, intense muscle pain, and usually no sensory findings. Lower extremity involvement is more common than upper extremity involvement, and muscle atrophy develops progressively. Complete loss of strength in one or more extremities may occur within a few hours or days. Paralytic polio may present with bulbar, spinal, or bulbospinal involvement. Bladder and bowel atony may be present. Respiratory distress may occur due to involvement of the motor cranial nuclei in bulbar poliomyelitis or due to paralysis of the diaphragm and intercostal muscles in spinal involvement (1,15).

Diagnosis, notification, differential diagnosis and treatment

The diagnosis is made based on clinical presentation and virus isolation; however, magnetic resonance imaging and electromyography may be required to rule out differential diagnoses. Patients presenting with acute flaccid paralysis should have notification forms completed, and at least two stool samples, collected at least 24 hours apart starting from the onset of paralysis, should be delivered to the Ministry of Health within three days, maintaining the cold chain (26).

Differential diagnoses include postinfectious transverse myelitis, spinal mass, Guillain-Barré syndrome, botulism, inflammatory myopathy, motor neuropathies, myasthenia gravis, and acute intermittent porphyria (27). Due to the absence of a specific antiviral therapeutic agent, the treatment of poliomyelitis can be summarized as symptomatic support, pain control, respiratory support, and physical therapy (16,27).

Vaccination

Vaccination forms the basis of the fight against polio, with the main vaccines used being the inactivated poliovirus vaccine (IPV) and the oral poliovirus vaccine (OPV). In the current global strategy, while oral polio vaccine is being phased out, IPV has become the primary vaccination tool (28).

The inactivated poliovirus vaccine is administered parenterally and contains three serotypes inactivated with

formalin. IPV is administered intramuscularly and can be given in combination with vaccines such as diphtheria, tetanus, acellular pertussis, hepatitis B, and Haemophilus influenzae type B. The inactivated poliovirus vaccine provides systemic humoral immunity and prevents paralytic disease, but it cannot prevent fecal viral shedding because its ability to provide mucosal immunity alone is limited. IPV doses given to children who previously received OPV boost the mucosal immunity provided by OPV. Serious side effects other than pain at the injection site are rare.

The oral polio vaccine contains a live virus that has been weakened (attenuated) through repeated passages and, thanks to its ability to provide strong mucosal immunity, increases community immunity and is effective in controlling outbreaks. However, vaccine-associated paralysis (VAPP) or vaccine-derived poliovirus strains (VDPV) may rarely develop (29). Vaccine-derived strains emerge particularly in areas with low vaccination coverage and widespread fecal contamination.

Until April 2016, a trivalent oral polio vaccine containing Type 1, Type 2, and Type 3 Sabin virus was used. As of that date, the type 2 virus was globally removed from the vaccine content, and a bivalent vaccine containing type 1 and type 3 virus began to be used (23). In areas with low vaccination rates, the weakened Type 2 OPA virus circulated in the community for a long time and mutated, resulting in an increase in cVDPV2 cases.

Novel OPA Type 2 (nOPA2) is a new generation oral poliovirus vaccine with increased genetic stability, developed against cVDPV2 poliovirus outbreaks. This vaccine, which is on the World Health Organization's emergency use list, provides mucosal and humoral immune responses similar to those of the Sabin OPA2 vaccine. Field data show high immunogenicity and a good safety profile. In line with the global eradication goal, nOPA2 is recommended for selected use in outbreak control (14,23).

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