Introduction

**Poliomyelitis**, also called **infantile paralysis** is an acute viral infectious disease of the nervous system that usually begins with general symptoms such as fever, headache, nausea, fatigue, and muscle pains and spasms and is sometimes followed by a more serious and permanent **paralysis** of muscles in one or more limbs, the throat, or the chest. More than half of all cases of polio occur in children under the age of five. The paralysis so commonly associated with the disease actually affects less than 1 percent of persons infected by the poliovirus. Between 5 and 10 percent of infected persons display only the general symptoms outlined above, and more than 90 percent show no signs of illness at all. For those infected by the poliovirus, there is no cure, and in the mid-20th century hundreds of thousands of children were struck by the disease every year. Since the 1960s, thanks to widespread use of polio vaccines, polio has been eliminated from most of the world, and it is now endemic only in four countries: Nigeria, India, Afghanistan, and Pakistan. Approximately 1,000–2,000 children are still paralyzed by polio each year, most of them in India ("Polio", 2013).

There are three known serotypes (closely related though distinguishable forms) of wild poliovirus: PV1, PV2, and PV3. The most widespread serotype is PV1. PV2 likely has been eradicated; the last PV2 case was reported in 1999 in Uttar Pradesh, India. The third serotype, PV3, is close to eradication ("Polio", 2013).
Basic Characteristics of Poliovirus

Poliovirus is a human enterovirus and a member of the family of Picornaviridae. Poliovirus is composed of an RNA genome and a protein capsid. The genome is a single-stranded positive-sense RNA genome that is about 7500 nucleotides long. The viral particle is about 30 nanometres in diameter with icosahedral symmetry. Because of its short genome and its simple composition—only RNA and a non-enveloped icosahedral protein coat that encapsulates it—poliovirus is widely regarded as the simplest significant virus (Drutz & Ligon, 2000).

There are three poliovirus serotypes, referred to as type 1, type 2 or type 3. There is minimal heterotypic immunity between the three serotypes. That is, immunity to one serotype does not produce significant immunity to the other serotypes. The poliovirus is rapidly inactivated by heat, formaldehyde, chlorine, and ultraviolet light. Picornaviruses are stable at acid pH and are small, ether-insensitive viruses with an RNA genome (Centres for Disease Prevention and Control (CDC), 2011).

Pathology and Pathogenesis

The virus enters through the mouth, and primary multiplication of the virus occurs at the site of implantation in the pharynx and gastrointestinal tract. The virus is usually present in the throat and in the stool before the onset of illness. One week after onset there is fewer viruses in the throat, but virus continues to be excreted in the stool for several weeks (CDC, 2012c). Poliovirus replication
occurs in the alimentary tract, followed by a primary, transient presence of viremia. The virus
invades local lymphoid tissue, enters the bloodstream, and then may infect cells of the central
nervous system. Replication of poliovirus in motor neurons of the anterior horn and brain stem
results in cell destruction and causes the typical manifestations of poliomyelitis (CDC, 2011).

The primary determinant of infection for any virus is its ability to enter a cell and produce
additional infectious particles. The presence of CD155 is thought to define the animals and tissues
that can be infected by poliovirus. CD155 is found (outside of laboratories) only on the cells of
humans, higher primates, and Old World monkeys. Poliovirus is however strictly a human pathogen,
and does not naturally infect any other species (although chimpanzees and Old World monkeys can
be experimentally infected). The CD155 gene appears to have been subject to positive selection. The
protein has several domains of which domain D1 contains the polio virus binding site. Within this
domain 37 amino acids are responsible for binding the virus (Mueller & Wimmer, 2003).

Clinically, polio virus transmission is by faecal–oral or occasionally oral–oral routes and the
incubation period is commonly 6 to 20 days with a range of 3 to 35 days. The response to poliovirus
infection is highly variable and has been categorized on the basis of the severity of clinical
presentation (CDC, 2005; 2012c).

According to CDC (2005; 2012c), up to 95% of all polio infections are inapparent or asymptomatic. Estimates of the ratio of asymptomatic to paralytic illness vary from 50:1 to 1,000:1 (usually
200:1). Persons infected but without symptoms shed virus in the stool and are able to transmit the
virus to others.

Approximately 4%–8% of polio infections consist of a minor, nonspecific illness without clinical
or laboratory evidence of central nervous system invasion. This clinical presentation is known as
abortive poliomyelitis, and is characterized by complete recovery in less than a week. The spread and replication of the virus in other sites such as brown fat, reticuloendothelial tissue, and muscle causes secondary viremia and leads to the development of minor symptoms. Three syndromes observed with this form of poliovirus infection are upper respiratory tract infection (sore throat and fever), gastrointestinal disturbances (nausea, vomiting, abdominal pain, constipation or, rarely, diarrhea), and influenza-like illness. These syndromes are indistinguishable from other viral illnesses (CDC, 2012c).

Nonparalytic aseptic meningitis (symptoms of stiffness of the neck, back, and/or legs), usually following several days after a prodrome similar to that of minor illness, occurs in 1%–2% of polio infections. Increased or abnormal sensations can also occur. Typically these symptoms will last from 2 to 10 days, followed by complete recovery (CDC, 2012c).

Less than 1% of all polio infections result in flaccid paralysis. Paralytic disease occurs when the virus enters the central nervous system (CNS) and replicates in motor neurons within the spinal cord, brain stem, or motor cortex, resulting in the selective destruction of motor neurons leading to temporary or permanent paralysis. In rare cases, paralytic poliomyelitis leads to respiratory arrest and death. Paralytic symptoms generally begin 1 to 10 days after prodromal symptoms and progress for 2 to 3 days. Generally, no further paralysis occurs after the temperature returns to normal. The prodrome may be biphasic, especially in children, with initial minor symptoms separated by a 1- to 7-day period from more major symptoms. Additional prodromal signs and symptoms can include a loss of superficial reflexes, initially increased deep tendon reflexes and severe muscle aches and spasms in the limbs or back. The illness progresses to flaccid paralysis with diminished deep tendon reflexes, reaches a plateau without change for days to weeks, and is usually asymmetrical. Strength then begins to return. Patients do not experience sensory losses or changes in cognition. Many persons
with paralytic poliomyelitis recover completely and, in most, muscle function returns to some degree. Weakness or paralysis still present 12 months after onset is usually permanent (CDC, 2012c).

Paralytic polio is classified into three types, depending on the level of involvement. Spinal polio is most common, and during 1969–1979, accounted for 79% of paralytic cases. It is characterized by asymmetric paralysis that most often involves the legs. Bulbar polio leads to weakness of muscles innervated by cranial nerves and accounted for 2% of cases during this period. Bulbospinal polio, a combination of bulbar and spinal paralysis, accounted for 19% of cases. The death-to-case ratio for paralytic polio is generally 2%–5% among children and up to 15%–30% for adults (depending on age). It increases to 25%–75% with bulbar involvement (CDC, 2012c).

Persons with residual impairment following paralytic poliomyelitis can develop a condition called **post polio syndrome (PPS)**, after a period of prolonged stability (usually 30–40 years) (Howard, 2005) in 25% to 40% of these persons (CDC, 2012c). PPS is characterised by exacerbation of existing muscle weakness with new weakness or paralysis in previously unaffected muscles. Factors that increase the risk of PPS include, increased period of acute poliovirus infection, presence of permanent impairment after recovery from acute polio, and female sex (CDC, 2012c). It is believed that the pathogenesis of PPS is caused by the failure of oversized motor units created during the recovery process of initial paralytic polio. PPS is rarely life threatening but has a slow, step-wise, unpredictable course. PPS is not an infectious process and persons who develop the syndrome do not shed poliovirus (Howard, 2005; CDC, 2012c).

**Epidemiology**
According to World Health Organization (WHO) (2010), the incidence of poliomyelitis has been dramatically reduced worldwide, as a result of vaccination, with the WHO aiming to achieve cessation of all wild poliovirus transmission worldwide by 2013 through an intensified Global Polio Eradication Initiative. There have been imported poliomyelitis case reports in parts of Southeast Asia, Eastern Europe and Africa (WHO, 2011). In 1994, the continents of North and South America were certified to be free of polio, followed by the Western Pacific region (including Australia) in 2000 (WHO, 2000) and the European region in 2002 (WHO, 2002). In countries where the disease incidence is low but transmission is still occurring, poliomyelitis cases are seen sporadically or as outbreaks among non-vaccinated persons. Polio cases have decreased by over 99% since 1988, from an estimated 350 000 cases in more than 125 endemic countries then, to 223 reported cases in 2012. In the same year, there were still polio cases in Afghanistan, Nigeria and Pakistan (CDC, 2012c). They also stated that the most recent country to halt transmission was India, which on January 12, 2012, celebrated one full year with no new reported cases. In 2013, only parts of these three countries in the world remain endemic for the disease—the smallest geographic area in history—and case numbers of wild poliovirus type 3 are down to lowest-ever levels. Other countries also have cases of wild-type poliomyelitis from time to time, due to importation.

According to Global Polio Eradication Initiative (GPEI) (2010), the World Health Assembly has repeatedly identified Nigeria as the single biggest risk to global polio eradication. It is the only endemic country with ongoing transmission of both wild poliovirus (WPV) types 1 and 3. (Afghanistan had no WPV type 3 in 2011, and Pakistan’s last reported case was in June 2011). Multiple factors have hampered eradication efforts in Nigeria’s northern states. Many are embedded in the country’s border social-political dynamics, the enduring chasm between government and the governed, and a decentralized government system that has often neglected service delivery to marginalized
communities. Nonetheless, considerable progress has been made to overcome these challenges through the application of diplomatic pressure, incentives, new technologies, more comprehensive and culturally sensitive approaches, and renewed high-level political will.

Until global eradication of polio is achieved, all countries are at risk of polio infection. For example, in 2005, Indonesia had an imported case of polio, 10 years after the last case of indigenously acquired poliomyelitis was reported in the country. This imported case of polio caused a re-establishment of local transmission of the virus and resulted in a large polio epidemic, with more than 200 cases (Thorley et al., 2006). Also, a polio outbreak that emerged in April 2010 in Tajikistan was caused by a strain of PV1 that was very closely related to a strain isolated in Uttar Pradesh, India. Later that year the virus appeared in Kunduz province in northeastern Afghanistan, which shared a border with Tajikistan. Both Tajikistan and Kunduz province had been polio-free for more than a decade prior to the 2010 outbreaks. A sudden surge in polio cases and deaths in late October and early November 2010 in Congo (Brazzaville) raised further concern that poliovirus was spreading from polio-endemic regions. The outbreak in Congo was believed to have been caused by wild PV1 from India. Major challenges facing efforts to prevent the spread of the disease to polio-free countries included the hajj and ‘umrah pilgrimages to Mecca, each of which involved large-scale population movements of people from polio-endemic countries (“Polio”, 2013).

Mass vaccination against polio using intramuscular Salk inactivated polio vaccine (IPV-Salk) – named after Jonas Salk, first started in Australia in 1956. In 1966, IPV-Salk was replaced by Sabin oral polio vaccine (OPV-Sabin) –named after Albert Sabin, in the publicly funded immunisation program. OPV-Sabin is particularly suited to provide mass protection against wild-type polio. Continuing the polio vaccination program to maintain the current high coverage rate and adequate surveillance for cases of acute flaccid paralysis remain the best defence against the continuing threat of polio infection (Roche & Spencer, 2002).
Recognizing both the epidemiological opportunity and the significant risks of potential failure, the World Health Assembly in May 2012 adopted a resolution declaring the completion of polio eradication a programmatic emergency for global public health and called for the development of a comprehensive polio eradication and endgame strategy through 2018 to secure a lasting polio-free world. Subsequently, the three remaining endemic countries launched national polio emergency action plans, overseen in each case by the respective head of state, and the partner agencies of the GPEI also moved their operations to an emergency footing, working under the auspices of the Global Emergency Action Plan 2012-2013. By the start of 2013, the impact of the emergency approaches is being seen, with the lowest number of reported cases in fewer districts of fewer countries than at any previous time. Since then, the new Polio Eradication and Endgame Strategic Plan 2013-2018 has been developed, in consultation with polio-affected countries, stakeholders, donors, partners and national and international advisory bodies. The new Plan was presented at a Global Vaccine Summit in Abu Dhabi, United Arab Emirates, at the end of April 2013. It is the first plan to eradicate all types of polio disease simultaneously – both due to wild poliovirus and due to vaccine-derived polioviruses (WHO, 2013).
TABLE. Number of reported non-polio acute flaccid paralysis (NPAFP) cases and acute flaccid paralysis surveillance indicators among children aged 6–15 years, and oral polio vaccination history among children aged 6–35 months with NPAFP — Nigeria, January 2011–September 2012*

<table>
<thead>
<tr>
<th>Region/State</th>
<th>January–December 2011</th>
<th>January–September 2012</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No. of NPAFP cases</td>
<td>NPAFP rate†</td>
</tr>
<tr>
<td>High-risk northern states</td>
<td>2,411</td>
<td>8.6</td>
</tr>
<tr>
<td>Bauchi</td>
<td>213</td>
<td>8.6</td>
</tr>
<tr>
<td>Borno</td>
<td>148</td>
<td>6.5</td>
</tr>
<tr>
<td>Gombe</td>
<td>149</td>
<td>12.0</td>
</tr>
<tr>
<td>Jigawa</td>
<td>187</td>
<td>8.3</td>
</tr>
<tr>
<td>Kaduna</td>
<td>171</td>
<td>5.4</td>
</tr>
<tr>
<td>Kano</td>
<td>401</td>
<td>8.1</td>
</tr>
<tr>
<td>Katsina</td>
<td>173</td>
<td>5.7</td>
</tr>
<tr>
<td>Kebbi</td>
<td>205</td>
<td>12.1</td>
</tr>
<tr>
<td>Niger</td>
<td>250</td>
<td>11.9</td>
</tr>
<tr>
<td>Sokoto</td>
<td>190</td>
<td>9.9</td>
</tr>
<tr>
<td>Yobe</td>
<td>136</td>
<td>11.0</td>
</tr>
<tr>
<td>Zamfara</td>
<td>188</td>
<td>10.9</td>
</tr>
<tr>
<td>Other northern states††</td>
<td>1,000</td>
<td>9.4</td>
</tr>
<tr>
<td>Southern states§§</td>
<td>2,438</td>
<td>6.9</td>
</tr>
<tr>
<td>Total</td>
<td>5,849</td>
<td>7.9</td>
</tr>
</tbody>
</table>
† Data as of October 30, 2012.

† Per 100,000 children aged <15 years.

‡ Two stool specimens collected at an interval of ≥24 hours within 14 days of paralysis onset and properly shipped to the laboratory and arriving in good condition.

§ Includes cases pending final classification as of October 30, 2012.

** Annualized data.


Adapted from: (CDC, 2012c)

**FIGURE 1. Number of cases of wild poliovirus type 1 (WPV1), wild poliovirus type 3 (WPV3), and circulating vaccine-derived polio virus type 2 (cVDPV2), by month — Nigeria, January 2009–September 2012*
The figure above shows the number of cases of wild poliovirus type 1 (WPV1), wild poliovirus type 3 (WPV3), and circulating vaccine-derived polio virus type 2, by month, in Nigeria during January 2009-September 2012. During January-September 2012, 99 WPV (82 WPV1 and 17 WPV3) cases were reported, compared with 38 (28 WPV1 and 10 WPV3) cases during the same period in 2011.

FIGURE 2. Cases of wild poliovirus type 1 (WPV1), wild poliovirus type 3 (WPV3), and circulating vaccine-derived polio virus type 2 (cVDPV2),* by year — Nigeria, January 2011–September 2012†
The figure above shows cases of wild poliovirus type 1 (WPV1), wild poliovirus type 3 (WPV3), and circulating vaccine-derived polio virus type 2 (cVDPV2), by year, in Nigeria during January 2011-
September 2012. During 2011, a total of 62 WPV (47 WPV1 and 15 WPV3) cases were reported in Nigeria, compared with 21 (eight WPV1 and 13 WPV3) cases in 2010, an increase of 195%.


