

Poliomyelitis

Poliomyelitis (also referred to as *polio*) is an infection caused by the poliovirus, a virus of the Enterovirus genus, family Picornaviridae. Poliovirus is a single-stranded RNA virus with a protein capsid. In its most acute cases, it selectively destroys lower motor neurons of the spinal cord and brainstem which results in unilateral flaccid weakness or paralysis of the limbs. It may also cause death due to acute respiratory arrest. The illness can be characterized as a "remembered" disease, since it has been largely eradicated throughout the second half of the 20th century; however, the World Health Organization estimates there are still twenty to thirty million survivors alive. The global polio pandemic of the 1950's has had historical ramifications on the course of medicine, since it spurred the creation of the first Intensive Care Units and launched the first mass inoculation campaigns.

Epidemiology

Poliovirus, like all enteroviruses, is ingested through contaminated water or foods, proliferates in the gastrointestinal tract, and is then shed in the feces of the infected individual. It is, therefore, transmitted through the "fecal-oral route". The virus enters through the mouth and nose, multiplies in the throat and intestinal tract, and then is absorbed and spread through the blood and lymph system. The incubation time ranges from 5 - 35 days (average 7 - 14 days). In the United States, cases of wild-type poliovirus infections have not been reported in more than twenty years. The few cases that do occur are caused by the reversion to virulence in the live-attenuated Sabin polio vaccine. Wild-type poliovirus has been eliminated from Western Europe, Japan, and the Americas. However, it is still endemic in sub-Saharan Africa and southern Asia.

Pathogenesis

Poliovirus may follow one of several courses:

- asymptomatic infection (in 90-95% of cases);
- abortive infection;
- non-paralytic infection;
- paralytic poliomyelitis (in about 1% of cases).

The mechanism of its spread from the alimentary tract to other systems (e.g. CNS) has not yet been substantiated. However, in all cases of spread, a primary viremia is present after infection. The theories of spread include infection through infected monocytes crossing the blood-brain barrier, retrograde axonal infection of neurons contacting infected tissues in the periphery and transmitting it to the central nervous system, or simple direct passage of the virions through the blood-brain barrier.

Symptoms

Infections of the poliovirus have three patterns of infection. These are subclinical symptoms which may not be present or may only last for less than 72 hours), and clinical patterns which are further divided into non-paralytic and paralytic poliomyelitis. The symptoms for each of the patterns are:

Subclinical Infection Symptoms

Symptoms may be absent, or gone after first 72 hours. They include malaise, headache, sore throat, slight fever, vomiting.

Non-paralytic poliomyelitis

Symptoms include back pain or backache, diarrhea, excessive tiredness, fatigue, headache, irritability, leg pain (calf muscles), moderate fever, muscle stiffness, muscle tenderness and spasm in any area of the body, neck pain and stiffness, pain in front part of neck, pain or stiffness of the back, arms, legs, abdomen, skin rash or lesion with pain, vomiting.

Symptoms last 1-2 weeks.

Paralytic poliomyelitis

Usually starts with a fever 5-7 days before the appearance of other symptoms. Symptoms include abnormal sensations (but not loss of sensation) in an area, bloated feeling in abdomen, breathing difficulty, constipation, difficulty beginning to urinate, drooling, headache, irritability or poor temper control. Muscular complications include muscle contractions or muscle spasms in the calf, neck, or back, muscle pain, muscle weakness that is only on one side or worse on one side. The location of this pain depends on where the spinal cord is affected. This pain worsens into paralysis, and sensitivity to touch.

Postpoliomyelitis syndrome

About twenty to thirty five percent of patients who recover from paralytic poliomyelitis have new onsets of muscle weakness, pain, atrophy and fatigue 25 to 35 years after the onset of the acute infection.

Prognosis

Permanent weakness is observed in about two out of three patients affected with paralytic poliomyelitis. Patients who require mechanical ventilation (known as the iron lung in the pandemic era) rarely recover without some form of permanent disability.

Treatment, and prevention

No specific antiviral treatment is yet known for poliovirus, so treatment remains supportive and symptomatic. During the 1950's global pandemic, mechanical respiratory assistance was given through the *iron lung*. Vaccinations, therefore remain to be the most effective way for dealing with the disease.

Vaccines

The polio vaccine was developed by Jonas Salk in 1952, and introduced to the world in 1955. The oral vaccine was developed by Albert Sabin and licensed in the early 1960's. The discovery of these vaccines was critical to the eradication of this disease due to the fact that immunocompetent individuals do not have a long-term carrier state, the fact that polioviruses do not have a non-primate reservoir in nature, and that survival of the virus outside of a host is practically non-existent. Therefore, by providing the vaccine and the subsequent human-to-human transmission, poliomyelitis could be a key step in the disease's global eradication.

Salk Vaccine

The Salk vaccine, or inactivated poliovirus vaccine (IPV) is based on strains of the three known serotypes of the virus, and is cultured as a Vero cell line tissue culture (derived from monkey kidney tissue). The vaccine induces an IgG-mediated immunity in the blood, preventing polio from progressing to viremia (an obligatory condition to be met for the further infection of the host). The strains are then inactivated with formalin.

Oral Poliovirus Vaccine

The oral poliovirus vaccine (OPV) is the form of the vaccine used mostly during the eradication campaigns by the WHO and allied groups. This version of the vaccine is produced through passage of the virus through non-human cells at a sub-physiological temperature. This produces spontaneous mutations to the viral genome. Eventually, 57 nucleotide substitutions distinguish the viral strains found in the OPV from the Mahoney serotype. The other serotypes are also present in the OPV with similar mutation rates. The location of these mutations is a key factor to the attenuation of the virus: The mutations are found in the IRES, thus interfering with the ability of the virus to translate its RNA genome in the host cell. The strains found in the Sabin OPV are able to efficiently reproduce in the gut, however they are not able to do so in nervous tissue, thus making it safe for the recipient. Apart from its high effectiveness, the other major reason for the OPV to be chosen for wide distribution in mass vaccination campaigns is the elimination of the need for sterile syringes.

The Salk (killed polio) vaccine has no adverse effects, whereas the OPV may undergo reversion to a virulent form while it multiplies in the GIT and propagate to cause paralytic poliomyelitis. Although the wild-type poliovirus has been eradicated in Western Europe, Japan and the Americas.



Individual who had suffered from paralytic poliomyelitis



Iron Lung



Polio Vaccination in Sweden during the global pandemic of 1950's

Links

Bibliography

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