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Review Article

Poliomyelitis

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ABSTRACT

Poliomyelitis is a highly contagious disease caused by a virus belonging to the PICORNAVIRIDAE family. Post-polio syndrome survives paralytic poliomyelitis and is characterized by a neuromuscular symptom complex that leads to impaired physical function. To systematically review the effect of any treatment on post-polio syndrome compared with placebo, usual care or no treatment. Post-polio syndrome is characterized by a worsening of existing or new health problems, most commonly muscle weakness and fatigue, general fatigue, and pain after a period of stability following an acute post-polio infection. Patients with post-polio syndrome should be advised to avoid both inactivity and overloading of weak muscles.

INTRODUCTION

DEFINITION:

Polio is an acute viral infectious disease that spreads from person to person mainly through feces and mouth. Polio is an infectious disease caused by an intestinal virus that can affect nerve cells in the brain and spinal cord. The word polio comes from the Greek word's "polio" meaning "gray" and "myelon" meaning "core".

EPIDEMOLOGY:

Jacob Heine recognized poliomyelitis as a separate disease in 1840. The polio virus that causes it was identified in 1908 by Karl Landsteiner. Polio was one of the most feared childhood diseases of the 20th century. Polio occurs only in a few African and Asian countries. The first case was recorded in New York and the last case was recorded in the Philippines in 1993. By 1910, the number of polio cases in much of the world increased dramatically, mostly in cities during the summer months. About 90% of polio infections cause no symptoms, and when the virus enters the bloodstream, a sick person can experience a variety of symptoms. In approximately 1 percent of cases, the virus enters

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the central nervous system and destroys the motor neuron.

TYPES:

Polio is divided into three types:

1. Abortive poliomyelitis
2. Non-paralytic poliomyelitis
3. Paralytic poliomyelitis

1. Abortive poliomyelitis:

A low-grade poliovirus infection that causes 80-90% of clinically apparent polio cases in the United States, mostly in young children. (Recovery: less than 1 week).

SYMPTOMS:

Fever, malaise, headache, sore throat, vomiting, abdominal pain, loss of appetite.

2. Non-paralytic poliomyelitis:

A type of polio that does not cause paralysis.

SYMPTOMS:

Headache, nausea, vomiting, neck, back and leg pain and stiffness. (Recovery: 2-10 days after symptoms).

3. **Paralytic poliomyelitis**:

Poliovirus spreads along specific nerve fiber pathways, mainly by replicating and destroying motor neurons in the spinal cord, brainstem or motor cortex. This leads to paralytic poliomyelitis in different parts.

a. Spinal poliomyelitis:

Paralytic poliomyelitis, the most common form of spinal poliomyelitis, is caused by the virus invading the motor neurons of the anterior horn cells or the ventral gray matter of the spinal cord, which are responsible for moving muscles, including muscles. trunk, limbs and intercostal muscles. The virus attacks the motor neurons in the spinal cord and causes paralysis of the arms and legs and breathing difficulties. Invasion of viruses causes inflammation of nerve cells, which leads to damage or destruction of motor neurons, when neurons are destroyed, muscles no longer receive signals from the brain or spinal cord; without nerve stimulation, muscles atrophy, weaken, weaken and become poorly controlled and eventually paralyzed. The extent of spinal paralysis depends on the affected area of the cord, which can be cervical, thoracic or lumbar.

b. Polio bulb:

Up to 2% of paralytic polio develop bulbar poliomyelitis when the poliomyelitis invades and destroys the nerves in the bulbar region of the brainstem. The virus affects nerve cells for sight, vision, taste, swallowing and breathing. Destruction of these nerves weakens the muscles of the cranial nerves and causes difficulty in breathing, speaking and swallowing. Critical nerves include the glossopharyngeal nerve, which partially controls swallowing, and the vagus nerve, which sends signals to the heart, intestines, and lungs, and the accessory nerve, which controls movement of the upper neck.

c. Bulbospinal poliomyelitis:

About 19 percent of all cases of paralytic poliomyelitis have both bulbar and spinal symptoms. This subtype is called respiratory or bulbospinal poliomyelitis. The virus affects the upper part of the cervical spinal cord and the diaphragm becomes paralyzed. Critical nerves affected are the phrenic nerve, which leads the diaphragm to the lungs, and those that control the muscles involved in swallowing.

ETIOLOGY:

Polio is caused by infection with several families of enteroviruses known as "polio". It can also be caused by three serotypes of poliovirus ie. type-1, type-2, type-3. This group of RNA viruses colonizes the gastrointestinal tract, especially the oropharynx and intestine. The incubation period is 3-35 days, more often 6-20 days Poliovirus infects and causes disease only in humans. Shipping method: poliovirus is spread through feces and oral ingestion. Inhalation of droplets of respiratory tract secretions or entry through the conjunctiva may also be a possible route of entry for close contact with patients in the early stages of the

disease. The virus initially multiplies in the epithelial cells and lymphatic tissue of the gastrointestinal tract. It then spreads to the lymph nodes and enters the bloodstream, after further replication in the reticuloendothelial system, the virus re-enters the bloodstream and travels to the spinal cord and brain. The spread of the virus in feces is the reason why it is a highly contagious disease. The spread of the virus is observed 2-3 days before and 1 week after the appearance of symptoms. Spread is rapid in areas with poor sanitation, especially among immunocompromised people. The spread of the virus is mainly observed in warm regions during the summer months in temperatures.

Risk factors:

- Age: Children and the elderly.
- Living with an infected person.
- Weakened immune system.
- No polio vaccination.
- Extreme stress or strenuous activity.
- Travel to an area where there has been a polio epidemic.

LIFECYCLE:

The main stages of the poliovirus life cycle are:

1. Entering the host.
2. Attachment to the host cell.
3. RNA release.
4. Production of new virions.
5. Creation of new virions.
6. Attack on nerve cells.

Poliovirus enters the host through the mouth or orally with feces. The route that is more common is through the mouth to the feces. It is transmitted orally and reaches the intestine of the host. Many cells in the body have a poliovirus receptor (PVR) on their surface. This receptor is made up of a protein called immunoglobulin, and is where the polio virus binds to host cells. There are 3 different chains in PVR and poliovirus binds to the first of them. The virus injects its DNA by making a hole in the plasma membrane or enters the cell by endocytosis. When it first injects RNA into a cell, it makes a hole in the membrane and then injects the RNA. And when it enters the cell, it first removes its coat. Poliovirus is a positive-strand RNA virus. Thus, the genome inside the virus particle can be used as messenger RNA and immediately translated by the host cell. Upon arrival, the virus hijacks the translation machine of the cell, which prevents the protein synthesis of the cell, promoting the production of a virus-specific protein. Poliovirus mRNA is translated into a single long polypeptide. The polypeptide is cleaved, resulting in mature viral proteins. The positive RNA serves as a template for the synthesis of the complementary negative strand. When the polyprotein is cleaved, it forms the precursors p1, p2 and p3. P1 forms the capsid proteins and the other two precursors form the viral genome. Encapsulation of the genome causes the formation of new virions. Once these virions enter the bloodstream, they travel to the brain and cause polio.

PATHOPHYSIOLOGY:

Polio virus enters the body through the mouth and most often infects nearby cells, such as cells in the mouth, nose and throat. Poliovirus usually targets certain tissues of the central nervous system, such as:

- Anterior horn cells of the spinal cord
- Hypothalamus
- Thalamus
- Vestibular nuclei
- Deep brain nodes
- Reticular formation
- Cerebellar worm

It infects cells by binding to an immunoglobulin-like receptor known as CD155 on the cell surface. The most common course of infection is reproduction of the poliomyelitis virus in the cells of the gastrointestinal tract, followed by shedding the virus in feces. The poliovirus breaks down within about a week in the cells of the

gastrointestinal tract, from where it spreads to the tonsils (especially the follicular dental cells of the germinal centers of the tonsils). Intestinal lymphoid tissue, including M cells of liver spots and deep mesenteric lymph nodes of the cervical head, where it proliferates profusely. Virus particles have been identified:

- Rich cells
- Hepatic ileal spots
- Mementian lymph node .

The virus is then absorbed into the bloodstream, which is called "viremia". The presence of the virus in the bloodstream allows it to spread widely throughout the body. Poliomyelitis can survive for a long time and multiply in the blood and lymphatic fluids. In a small percentage of cases, it can spread and multiply in other areas, such as brown fat, retinal endothelial cells and muscle. Poliovirus replicates in monocytes, allowing secondary hematogenous spread. Rarely, it can progress and the virus can invade the central nervous system, causing a local inflammatory reaction. In most cases, it causes inflammation of the meninges, the layers of tissue surrounding the brain, known as non-paralytic aseptic meningitis. Poliomyelitis of the central nervous system can be characterized by the selective destruction of motor neurons. Depending on the location, motor neuron loss can cause focal or generalized symptoms. Although the mechanisms of spread of the virus in the central nervous system are not fully understood, the main hypotheses have been presented:

1. Poliovirus spreads directly from the circulation of the blood-brain barrier into the central nervous system, independent of cellular receptors.
2. Poliovirus is transported from peripheral muscles to the brain and spinal cord by retrograde axonal transport. An important explanation for the increased susceptibility to retrograde axonal transport in poliomyelitis-damaged muscle regions has been elucidated. At the neuronal synapse, the rate of endocytosis is related to the level of neuronal activity. Similarly, the level of neuronal activity and rate of endocytosis of motor neurons at the neuromuscular junction are related to the extent of muscle contraction. This explains the association between extreme exertion or muscle injury and the development of poliomyelitis in virile patients. Furthermore, since most CD155 receptors are transported back to the cell body, the virus is carried along, supporting the retrograde transport hypothesis. After the cell body of a neuron has formed, the shift from axoplasm to cytoplasm is thought to disrupt neuronal stability and kill the motor neuron. The death of a motor neuron paralyzes the corresponding muscle fiber.

- **SINGS AND SYMPTOMS:**

In its most severe forms, poliomyelitis can lead to paralysis and death. However, most patients with polio show no symptoms or remain visibly ill. When symptoms occur, they vary depending on the type of polio. Symptomatic poliomyelitis can be further divided into a mild form called non-paralytic or abortive poliomyelitis, and a severe form called paralytic poliomyelitis, which occurs in about 1 percent of cases. Many people who do not have paralytic polio make a full recovery. Unfortunately, people with paralytic polio usually develop permanent paralysis.

- **NON-PARALYSISPOLIOSYMPTOMS:**

These include fever, sore throat, headache, vomiting, fatigue, back and neck pain, stiffness in arms and legs, muscle aches and cramps, meningitis (inflammation of the membranes surrounding the brain).

- **SYMPTOMS OF PARALYTICPOLIO:**

Paralytic polio affects only a small proportion of those infected with polio. Paralytic polio symptoms often begin in the same way as non-

paralytic polio symptoms, but then progress to more severe symptoms, such as: * Loss of muscle reflexes * severe muscle pain and cramps * weak or weak limbs, often worse on one side of the body. Complications and post-polio syndrome: Post-polio syndrome describes a cluster of symptoms that affect up to 64% of all polio patients. It appears several years after polio has passed. On average, post-polio syndrome develops 35 years after infection. Signs and symptoms of post-polio syndrome include: * slowly progressing muscle and joint pain *muscle atrophy or contraction *exhaustion without reason *swallowing and breathing difficulties *suffering from the cold *sleep related problems such as apnea * concentration and memory difficulties *mood swings and depression post-polio syndrome is a slow, progressive disease. There is no cure, but it is not contagious or contagious.

- **SYMPTOMS IN CHILDREN:**

Most children with polio have no symptoms at all. This is called an undetected infection. Symptoms may be slightly different for each child. Symptoms are e.g. Fever, loss of appetite, upset stomach (nausea), vomiting, sore throat, malaise, constipation, abdominal pain, muscle pain in the neck, trunk, arms and legs, stiffness in the legs along the spine, muscle weakness all over, severe constipation , balding paralysis, muscle wasting, shortness of breath, weak cough, hoarse voice, difficulty swallowing, muscle paralysis which may be permanent, salivation, irritability and anger. Most paralyzed children regain some of their strength over time. Some children return to normal. Few children die from this disease.

- **DIAGNOSIS:**

Polio virus can be detected in samples from the throat, feces (feces), and sometimes CSF (cerebrospinal fluid) by isolating the virus from cell culture or detecting the virus by PCR (polymerase chain reaction). Diagnostic tests for polio include: * Medical control * Blood tests * Isolation and detection of viruses (cell culture) * Throat wash *CSF analysis * Polio virus fingerprinting medical examination: This includes a full system audit. The function of the respiratory muscles is examined, because poliomyelitis, which affects the spinal cord and brainstem, can affect the respiratory muscles. Muscle reflexes are tested. Abnormal reflexes are noted. There may be a stiff neck and stiff muscles of the back. There may be difficulty in bending the neck and difficulty in lifting the head or legs when lying flat on the back. Blood tests: Blood is tested for antibodies for polio virus. Antibodies are molecules that are produced by the body against an invading virus or bacteria. When a person is infected with polio virus, special tests can detect the levels of polio virus specific antibodies and confirm the diagnosis. Virus isolation and detection (cell culture): Poliovirus can be acquired from the stool or pharynx of people with suspected poliomyelitis. Virus isolation from CSF is diagnostic but rarely successful. If poliovirus is isolated from a person with acute flaccid paralysis, it must be further tested by oligonucleotide mapping or genomic sequencing to determine whether the virus is wild-type or vaccine-like. Currently, the generally preferred methods are human cell lines, such as human amniotic fluid cell line and human embryonic cell line. In India, polio laboratories work with the human rhabdomyosarcoma-derived cell line RD and the cell line L20B, which are highly specific for poliovirus.11 The growth of the virus is determined by its cytopathic effect on the cell lines. This usually happens within 7 days after vaccination. If cytopathic changes are seen only in the RD cell line, inoculate the L20B cell line to confirm the presence of poliomyelitis. The isolate is then subjected to neutralization tests using specific antisera for serotyping. Tests are also done to determine if the isolate is a wild strain or from the vaccine. These tests are called intertype

differentiation tests.11 They are based either on the principle of enzyme-linked immune sorbent assay or on hybridization methods. Gargling: The throat is washed and evaluated for virus. Washing liquids are incubated in culture medium in a favorable atmosphere. If the culture is positive for the virus, it can be seen under a microscope. Stool samples are also tested for polio. Virus isolation from cerebrospinal fluid (CSF) is diagnostic but rarely possible. Analysis of the cerebrospinal fluid: Cerebrospinal fluid bathes the spinal cord and brain. This CSF is sampled by lumbar puncture. A lumbar puncture means inserting a long, thin needle between the vertebrae. It aspirates a small amount of CSF, which is sent to a laboratory for evaluation. A routine CSF examination involves assessing the concentration of cells (white blood cells, blood, etc.) and sugar and other chemicals in the cerebrospinal fluid. There are usually 10-200 cells/mm3 and the cerebrospinal fluid may have a slightly higher protein content of 40-50 mg/100 ml. Poliovirus infection can increase the number of white blood cells and slightly increase the concentration of proteins in the cerebrospinal fluid. S.no Perception of features

1. Appearance Clear/slightly cloudy
2. Cells 0-5 cells/µl (less than 2 polymorphonucleocytes) Leukocytosis (mainly lymphocytes)
3. Proteins andlt;5 of serum protein Normal/slightly raised 100-300 mg/dL
4. Glucose andgt;60 % of serum glucose Normal Fingerprinting the polio virus.

Once the polio virus is isolated it is tested by a special test called oligonucleotide mapping (fingerprinting) or genomic sequencing. This is essentially looking at the genetic sequence of the virus to detect if the origin of the virus is “wild type” or “vaccine like”. Wild type virus naturally occurs in the environment and may occur as 3 subtypes – P1, P2 and P3. Vaccine like virus is derived after a spontaneous mutation of the genes of the virus in the polio vaccine.

TREATMENT:

There is no cure for polio, treatment is lessening severity of the symptoms (of weakness, paralysis). The goal of the treatment is to control symptoms while the infection runs its course as there are no specific treatment for the viral infection. People with severe cases may need life saving measures, especially breathing help. Symptoms are treated based on their severity treatment may include;

- Anti-biotics to prevent infections in weakened muscles.
- Pain relieving drugs(to reduce headache,muscle pain and spasms).
- Orthopedic surgery
- Portable ventilators to support breathing (long-term ventilation).
- Catheterization of an enlarged bladder may be necessary.
- People with paralytic polio may need hospitalization.
- Moist heat reduces pain and spasms. Medicines: Medical treatment for polio is based on the severity of the symptoms. This includes different groups of drugs such as
- Antibiotics such as ampicillin and amoxicillin.
- Pain relievers such as ibuprofen, acetaminophen, diclofenac, aspirin.
- Cholinergic drugs such as bethanechol
- Various vaccines.

AMPICILLIN :

- **Mechanism of action:**

By binding to specific penicillin-binding proteins (PBPs) located in the bacterial cell wall, ampicillin inhibits the third and final step of bacterial cell wall synthesis. Cell lysis is then mediated by autolytic enzymes of the bacterial cell wall, such as autolyses; it is possible that ampicillin interferes with the autolysin inhibitor.

- **Pharmacokinetics:**

- **Method of administration:**

Oral i.v. and i.m.

- **Absorption:**

Maximum plasma time: 1-2 hours (oral) **Bioavailability:** 30-40%

Distribution: Protein bound: 15-25% **Metabolism**: Max Elimination: Half-life: 1-1. 8 hours (normal renal function); 7-20 hours (anuria/end-stage renal disease)

Excretion: Urine (90% within 24 hours)

Side effects: Erythema, multiforme, scaly, dermatitis, rash, urticaria, fever, convulsions, black hairy tongue, diarrhea, enterocolitis, glossitis, nausea, oral candidiasis, pseudomembranous, colitis, stomatitis, vomiting, agranulocytosis, hematocytosis, anemia, anemiapuraxia increased aspartate aminotransferase- activity, interstitial nephritis, laryngeal stridor, serum sickness-like reaction.

- **Directions:** Genital infections, including gonorrhea.
  - Infections of the respiratory tract.
  - Infections of the gastrointestinal tract.
  - Bacterial meningitis/septicaemia.
  - Treatment of endocarditis.
  - *Prophylaxis of endocarditis (off label)
  - Soft tissue infections.
- **Contraindications:**

Dialysis, renal disease, renal failure, renal failure, asthma, carbapenem hypersensitivity, cephalosporin hypersensitivity, eczema, penicillin hypersensitivity, severe rash, urticaria.

- **Use:**

Prophylaxis of bacterial endocarditis, gonococcal infection - uncomplicated, intra-abdominal infection, shigellosis, sinusitis, pneumonia, skin or soft tissue infection, upper respiratory tract, septicemia, bacterial infection, urinary tract infection, peritonitis, bronchitis, bronchitis.

- **Drug interactions:**

a. **Abacavir-ampicillin** may slow the rate of excretion of abacavir, which may result in increased serum concentrations.
b. **Acetaminophen** - Acetaminophen can slow the elimination rate of ampicillin, which can lead to higher serum levels.
c. **Aciclovir** - Aciclovir can slow the rate of elimination of ampicillin, resulting in increased serum concentrations. * Dosage: Oral - 250-500 mg every 6 hours By the way - 1-2 g every 4-6 hours

AMOXIXILLIN:

The activity of amoxicillin in vitro is similar to that of ampicillin. Amoxicillin is effective against a wider spectrum of bacteria. The main difference between ampicillin and amoxicillin is that amoxicillin is slightly fatter soluble. As a result, amoxicillin can kill bacteria a little faster. Ampicillin is usually given by i.v. or i.m., while amoxicillin is the preferred oral medication because it is less likely to cause diarrhea and can be administered less frequently than ampicillin. * Dosage: Oral - child: 15-30 mg/kg Adults: 250-500 mg/kg Oral solution - 125mg/5ml-400mg/5ml Ibuprofen:

- **Mechanism of action:**

Inhibits prostaglandin synthesis in body tissues by inhibiting at least two cyclooxygenase (COX) isoenzymes, COX-1 and COX-2. Can inhibit chemotaxis, alter lymphocyte activity, reduce pro-inflammatory cytokine activity and inhibit neutrophil aggregation; these effects may contribute to anti-inflammatory effects.

- **Pharmacokinetics:**

Absorption: Absorbs quickly (85%). **Bioavailability:** 80-100%. Start time: 30-60 min. Duration: 4-6 h

Distribution: Protein bound: 90-99%. **Metabolism:** Rapidly metabolized in the liver (mainly by CYP2C9; CYP2C19 substrate) by oxidation to inactive metabolites. Elimination: Half-life: 2-4 hours (adults); 1.6 hours (children 3

months - 1 year; 35-51 h (3rd day), 20-33 h (5th day).

Excretion:

urine (50-60%; leg; 10% unchanged); rest with stool 24 hours inside.

Side effects:

Dizziness, epigastric pain, heartburn, constipation, nausea, rash, tinnitus, swelling, fluid retention, headache, vomiting.

VACCINE POLIO VIRUSES:

Monovalent oral polio vaccines (mOPV) types 1 and 2 were licensed in 1961, followed by type 3 mOPV in 1962 and trivalent OPV (tOPV) in 1963. Oral polio vaccine (OPV) contains live poliovirus strains (Sabin) derived from wild polioviruses that are attenuated by repeated cell passages, causing mutations that reduce their neurovirulence and infectivity. After receiving the OPV vaccine, live attenuated polioviruses replicate in the intestinal mucosa and lymphoid cells in the oropharynx and intestinal tract in the same manner as wild poliovirus infection. Vaccine viruses are excreted in the feces of the vaccinated person for a maximum of 6 weeks after taking the dose, and the greatest excretion is during the first 1-2 weeks after vaccination. Vaccine viruses can spread from the recipient to contacts. People who come in contact with vaccine feces can be exposed to the vaccine virus and become infected. Replication and faecal excretion of vaccine virus may occur after receiving a new dose of OPV vaccine, but the duration of excretion is usually short and the concentration of virus in the faeces is lower. The trivalent OPV vaccine largely replaced the IPV vaccine as the vaccine of choice in the United States and most other countries of the world until the late 1990s. The almost unique use of the tOPV vaccine led to the eradication of wild poliovirus from the United States in less than 20 years. However, one case of VAPP occurred for every 2–3 million doses of tOPV vaccine administered. The VAPP burden in industrialized countries led to the abolition of the OPV vaccine. In 1996, the Advisory Committee on Immunization Practices (ACIP) recommended increasing the use of IPV with sequential IPV followed by tOPV to reduce the incidence of VAPP. The sequential schedule eliminates VAPP among vaccine recipients by establishing humoral immunity against polio with inactivated polio vaccine before exposure to live vaccine virus. Because tOPV was still used for the third and fourth doses, the risk of VAPP still remains in contacts of vaccine recipients exposed to live vaccine virus in the feces of vaccine recipients. The sequential IPV-OPV polio vaccination program has been widely accepted by both providers and parents. Fewer cases of VAPP were reported in 1998-1999, suggesting the effect of increased use of the IPV vaccine. To promote the complete eradication of paralytic poliomyelitis in the United States in 1999, the ACIP recommended that only the IPV vaccine be used. Exclusive use of the IPV vaccine eliminates the spread of live vaccine virus and eliminates all indigenous VAPP viruses. Of the three types of wild poliovirus, type 2 was declared eradicated in 2015. To eliminate the risk of circulating VDPV type 2 (cVDPV2) infection, in 2016, all OPV-using countries simultaneously switched from the tOPV vaccine to the bivalent OPV (bOPV) vaccine, which contains only poliomyelitis types 1 and 3, in accordance to the Guidance of World Health Policy Ministry. organization All countries use one or more doses of IPV vaccine, either alone or in combination with bOPV vaccines. The WHO Director-General must approve the use of mOPV2 in response to cVDPV2 outbreaks; The mOPV2 Advisory Group provides recommendations for use. There are five combination vaccines that contain the IPV vaccine. DTaP-HepB-IPV (Pediarix), DTaP-IPV/Hib (Pentacel), DTaP-IPV (Kinrix), DTaP-IPV (Quadracel), and DTaP-IPV-Hib-HepB (Vaxelis) are licensed and available in the United States. Vaccination schedule and use:

The first dose of IPV vaccine can be given as early as 6 months of age, but is usually given at 2 months of age and the second dose at 4 months of age. The third dose should be given between the ages of 6 and 18 months. The recommended interval between doses of the main series is 2 months. However, if accelerated protection is required, the minimum interval between the first three doses of IPV vaccine is 4 weeks. The last dose of the IPV series should be given between 4 and 6 years of age and at least 6 months after the previous dose. A dose of IPV vaccine is recommended at age 4 or later, regardless of the number of previous doses. Shorter intervals between doses or starting the series at a younger age may result in lower seroconversion rates. Therefore, using the minimum age (6 weeks) and minimum dose intervals during the first 6 months is recommended only if the vaccine recipient is at immediate risk of exposure to circulating poliovirus (eg, during an outbreak or due to travel). . to a polio-endemic area). Combined vaccines:

DTaP-HepB-IPV (Pediarix)

The DTaP-HepB-IPV vaccine is approved for use as a three-dose series in children 6 weeks to 6 years of age. It is given to babies aged 2, 4 and 6 months. The minimum intervals for the DTaP-HepB-IPV vaccine are determined by the DTaP component. There should be at least 4 weeks between the three doses. Because the minimum age for the first dose of DTaP-HepB-IPV vaccine is 6 weeks, this vaccine cannot be used as a birth dose of hepatitis B (HepB) vaccine. The last dose of DTaP-HepB-IPV vaccine should be given at least 24 weeks of age, which is the minimum age to complete the hepatitis B vaccine series. Using the DTaP-HepB-IPV vaccine to administer 3 doses at 2, 4 and 6 months of age (based on the DTaP and IPV schedules) results in a 4-dose HepB vaccine series that is acceptable.

DTaP-IPV/Hib (Pentacel):

The DTaP-IPV/Hib vaccine is approved for use as a 4-dose series in children 6 weeks to 4 years of age. It is given to babies aged 2, 4, 6 and 15-18 months. Minimum intervals for DTaP-IPV/Hib vaccine are determined by DTaP component. There must be at least 4 weeks between the first three doses. Dose 4 must be separated from dose 3 by at least six months and should not be given before 12 months of age. If DTaP-IPV/Hib vaccine is used to administer 4 doses at ages 2, 4, 6 years and 15-18 months (based on DTaP and Hib schedules), then an additional booster dose with separate IPV or DTaP-IPV- vaccine the vaccine must be given between the ages of 4 and 6 years. This results in a 5-dose IPV vaccine series that is acceptable.

DTaP-IPV-Hib-HepB (inVaxel):

DTaP-IPV-Hib-HepB is approved for use as a three-dose series in children 6 weeks to 4 years of age. The minimum intervals for the DTaP-IPV-Hib-HepB vaccine are determined by the DTaP component. Because the minimum age for the first dose of DTaP-IPV-Hib-HepB vaccine is 6 weeks, this vaccine cannot be used as a birth dose of hepatitis B (HepB) vaccine. The last dose of DTaP-IPV-Hib-HepB vaccine should be given at least 24 weeks of age, which is the minimum age to complete the hepatitis B vaccine series. Using the DTaP-IPV-Hib-HepB vaccine to administer 3 doses at 2, 4, and 6 months (based on the DTaP and IPV schedules) results in a 4-dose HepB vaccine series that is acceptable.

DTaP-IPV (Kinrix):

DTaP-IPV (Kinrix) vaccine is approved only for DTaP vaccine dose 5 and IPV vaccine dose 4 for children 4 to 6 years of age whose previous DTaP vaccine doses were Infanrix and/or Pediarix dose 1, 2, and 3 and Infanrix dose 4 However, when DTaP-IPV (Kinrix) vaccine is administered to children who received a different brand of DTaP vaccine for previous doses of DTaP vaccine, or when the vaccine is administered as doses 1, 2, 3,

or 4 of the DTaP -vaccine. series or doses 1, 2 of the IPV vaccine series or 3 no repeat dose of DTaP-IPV (Kinrix) is required.

DTaP-IPV (Quadracel):

DTaP-IPV (Quadracel) vaccine is approved only for 5 doses of DTaP vaccine and 4 or 5 doses of IPV vaccine in children 4 to 6 years of age who have received 4 doses of Pentacel and/or Daptacel vaccine. However, if DTaP-IPV (Quadracel) vaccine is administered to children who received a different brand of DTaP vaccine for previous doses of DTaP vaccine, or if they were administered dose 1, 2, 3, or 4 of the DTaP vaccine series, or dose 1, 2, or 3 of the IPV series, it is not necessary to Repeat a dose of DTaP-IPV (Quadracel). Adult Polio Vaccination: Polio vaccination recommendations for these adults depend on past vaccination history and the duration of protection required. If an adult at increased risk of exposure to polio has never received polio vaccine or has no written evidence of polio vaccination, primary immunization with IPV is recommended. The recommended schedule is 2 doses 1-2 months apart, the third dose is administered 6-12 months after the second dose. The minimum interval between doses 2 and 3 is 6 months. In some cases, time does not allow to fulfill this schedule. If the need for protection is 8 weeks or more, 3 doses of IPV vaccine should be administered at least 4 weeks apart. If there are 4-8 weeks before the need for protection, two doses of IPV vaccine should be given at least 4 weeks apart. If it is less than 4 weeks until protection is needed, a single dose of IPV vaccine is recommended. In all cases, the remaining vaccine doses should be administered at later recommended intervals if the individual is still at increased risk. Immunogenicity and vaccine efficacy: The IPV vaccine is very effective in producing immunity against polio and protection against paralytic polio. At least 90% of vaccine recipients develop protective antibodies against all three types of polio after 2 doses, and at least 99% are immune after 3 doses. The IPV vaccine prevents wild poliovirus from entering the recipient's central nervous system (CNS) and thus prevents paralysis. Protection against paralytic disease correlates with the presence of antibodies after vaccination. The IPV vaccine appears to induce less local gastrointestinal immunity than the OPV vaccine. Those who have received the IPV vaccine usually do not shed the virus in the nasopharynx, but instead excrete the virus in their feces when exposed to wild or vaccine polio. The duration of virus shedding and the amount of virus in feces are similar in IPV-vaccinated individuals to that of unvaccinated individuals when they are not exposed to live polio (vaccine or wild). The duration of immunity with IPV is not precisely known, although it is likely to provide lifelong immunity after a complete series. The OPV vaccine is very effective in producing immunity against poliovirus. Due to serotype interference during intestinal replication, a single dose of tOPV confers immunity to all three vaccine viruses in approximately 50% of recipients. The OPV vaccine induces local intestinal immunity, which reduces virus shedding after reinfection with the same serotype of polio and reduces potential transmission. Subsequent doses cause less interference with intestinal reproduction, and 3 doses confer immunity to all three polio types in more than 95% of recipients in industrialized countries. As with other live virus vaccines, immunity to oral poliovirus vaccine is likely to be lifelong.

Vaccination contraindications and precaution:

As with other vaccines, a severe allergic reaction (anaphylaxis) to any component of the vaccine or after a previous dose is a contraindication to further doses. A patient's moderate to severe acute illness (with or without fever) is considered a precaution against vaccination, although people with mild illness can be vaccinated. Because IPV

contains small amounts of streptomycin, neomycin, and polymyxin B, allergic reactions are possible in people sensitive to these antibiotics. People with allergies that are not anaphylactic, such as skin contact sensitivity, can be vaccinated. Contraindications for combination vaccines containing IPV include those for individual component vaccines (eg, DTaP, hepatitis B), but the specific ingredients may vary.

Pregnancy: Pregnancy is a precaution for the IPV vaccine. Although adverse effects of the IPV vaccine on pregnant women or their fetuses have not been documented, vaccination of pregnant women should be avoided for theoretical reasons. However, if a pregnant woman is at increased risk of infection and needs immediate protection against polio, the IPV vaccine can be administered according to the schedule recommended for adults.

Vaccine Safety: In prelicensing studies of enhanced IPV, local reactions were mild and transient. Participants reported induration (18%), pain (13%) and erythema (3.2%) within 48 hours after vaccination. Systemic reactions reported included fever (38% reported temperature ≥ 39°C), irritability, drowsiness, irritability, and crying. Study participants received DTP concurrently with IPV and therefore these systemic reactions could not be attributed to a specific vaccine. However, the frequency and severity of these reactions were comparable to those reported with DTP ALONE.

In countries where the IPV schedule is followed, no risk of serious adverse events has been observed. After the increased use of IPV in the United States, a review of the vaccine adverse event reporting system between 1991 and 1998 showed no increase in the reporting of poliovirus vaccine-related adverse events associated with increased use of IPV. In addition, symptom clustering was comparable for IPV and OPV. VAPP occurs very rarely after OPV vaccination. The mechanism of VAPP is thought to be a mutation or reversion of the attenuated vaccine polio to a more neurotropic form. Recovery is thought to occur in almost all vaccine recipients, but rarely leads to paralysis. The resulting paralysis is identical to that causeds by wild poliovirus. The IPV vaccine does not contain live virus, so it cannot cause VAPP. The risk of VAPP is 7-21 times higher with the first dose than with any other dose in the series. VAPP is more likely to occur in people 18 years of age or older than in children, and almost 7,000 times more often in people with certain types of immunodeficiency, especially B-lymphocyte disorders (such as agammaglobulinemia and hypogammaglobulinemia) that reduce immune system synthesis. . immunoglobulins. VDPVs are genetically distinct forms of vaccine strains. VDPVs develop as a result of long-term replication of OPV-containing vaccine strains in an immunocompromised individual or in a poorly vaccinated community and have restored the neurovirulence and infectivity of natural poliomyelitis. The risks and manifestations of paralysis caused by VDPV are similar to those of wild poliovirus of the same serotype. Outbreaks of circulating VDPV have caused more than 1,200 cases of paralytic polio between 2000 and 2019, and as of 2017 have outnumbered cases of wild polio.

CONCLUSION

Poliomyelitis cannot be curable but can be preventive by vaccines.

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