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POLIO AN OVERVIEW

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ABSTRACT

Poliomyelitis is an infectious disease with acute and persistent faccide paralysis is caused by poliovirus. Postpolio syndrome (PPS) may be affect survivors of paralytic poliomyelitis. it is characterised by complex of neuromuscular symptoms leading to a decline in physical functioning. The standard schedule for polio virus immunization remains 4 doses of inactivated polio virus vaccine at 2,4, and 6 through 18months and 4 throughb 6 years of age. poliomyelitis is a disease that became a public health issue at the beginning of 20th century and was more or less eliminated in developed countries by early 1970s. the global polio eradication initiative was launched in 1988 with the aim of eliminate paralytic poliomyelitis. today, as progress is made towards the goal of global eradication of poliomyelitis to wild polioviruses, all the developing countries where OPV is used face the risk of vaccine which associated paralytic poliomyelitis.

KEYWORDS: Poliomyelitis, Immunity, Vaccine, Herbal medicine, Immunization, Polio Eradication.

INTRODUCTION

Poliomyelitis is an viral, infectious disease. It spread from one person to another person, Polio is a contagious disease caused by an intestinal virus that may attack nerve cells of the brain and spinal cord. The Symptoms of poliovirus is fever, headache, sore throat, and

vomiting. In some cases developed neurological complications, stiffness of the neck and back, weak muscles, pain in the joints, and paralysis in muscles. Polio can be spread through contact with eachother faces, the airborne droplets, in food, or in water. The virus enters to the body by mouth or nose, and travels through the intestines. and Then it enters the bloodstream. The anti-polio and antibodies are produced in most of cases. That stops the progression of the polio virus. The individual gains permanent immunity against the disease.[1] poliovirus is used as model, because it is large body of research data exists the physical, chemical, and biological properties of the virus, vaccination is available, and it is easy to culture as compared to other viruses. The poliomyeliyis originates from the Greek Word "Polio" meaning "Grey" and "Myelon" meaning "Marrow". it is an infectious disease cause by poliovirus a member of genus enterovirus, belonging to the picornaviridae family.[2] Approximately 90% of poliovirus infections cause no symptoms at all, affected individuals which can exhibit a range of symptoms. In about 1% of cases, the virus enters the central nervous system, and they infected and destroying motor neurons, leading to muscle weakness and acute paralysis.[3] poliovirus infection occurs by a fecal and oral route the host ingests the virus, which can replicate in the alimentary tract. The virus is then shed in the feces. Most polio infections are asymptomatic.in about 5 percent of cases, the virus replicates in other tissues. The paralytic poliomyelitis occurs in less than 1 percent of cases. Polio virus composed of an RNA genome and a protein capsid. The genome is a single-stranded positive RNA genome that is about 7500 of neucleotides long. The viral partical is about 30nm in diameter with the symmetry. Polio virus was first isolated in 1909 by karl landsreiner and Erwin popper.

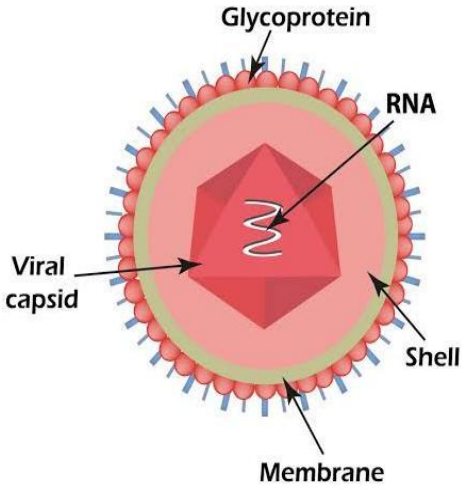

Fig. 1: Structure of Polio Virus.

Poliomyelitis was first distinguished by Jakob Heine in 1840. It is causative agent, poliovirus, was identified in 1908 by Karl Landsteiner. While the major polio epidemics were unknown before the late 19th century, polio virus was one of the most childhood diseases of the 20th century. By 1910, many countries experienced a dramatic increase in polio cases, and epidemics became regular events. Most of in cities during the summer months. These epidemics which can left thousands of childrens and adults paralyzed provided the impetus for a "Great Race" towards the development of a vaccine. Developed in the 1950s, polio vaccines are credited with reducing the global number of polio cases per. Enhanced vaccination efforts by Rotary International, the World Health Organization, and UNICEF Must result in global eradication of the disease.[4] Polio epidemics have thousands of people, mostly young children; the disease has caused paralysis and death for much of human history. Polio had existed.

Fig. 2: Jonas Salk-Inventor of Polio Vaccine.

for many years quietly as an endemic pathogen until the 1880s, when major epidemics began to occur in Europe; soon after, wide read epidemics appeared in the United States.[5] the virus may spread in to the posterior horn cells, motor neuron of the thalamus and hypothalamus. histological appearance of the affected brain cells shown vacuolation. widespread muscular atrophy occurs leading to flaccid paralysis. postpolio syndrome can occurs 25 to 30 years after the initial paralytic attack.[6]

CAUSES

Poliomyelitis is caused by infection with a member of the genus. Enterovirus known as poliovirus. this group of RNA viruses colonize the gastrointestinal tract specifically the pharynx and the intestine. The incubation time ranges from 3 to 35 days. The common span of 6 to 20 days polio virus infects and causes disease in humans. Its structure is very simple, composed of a single (+) sense of RNA genome enclosed in a protein shell called a capsid. In addition of protecting the virus's genetic material, capsid proteins enable poliovirus to infect

certain types of cells. There are 3 types of poliovirus have been identified poliovirus type 1 (Polio virus 1), type 2 (Polio virus 2), and type 3 (Polio virus 3) it is slightly different to the capsid protein. All three are produce the same disease symptoms. Polio virus 1 is the most commonly encountered form, and the one most closely associated with paralysis.[7] The Individuals who are exposed to the virus, either through infection or by immunization with polio vaccine and develop immunity. In immune individuals, IgA antibodies against poliovirus are present in the tonsils and gastrointestinal tract, and they are able to block virus replication; IgG and IgM antibodies against polio virus can prevent the spread of the virus to motor neurons of the central nervous system (CNS). Infection or vaccination with one serotype of poliovirus does not provide immunity to the other serotypes, and full immunity requires exposure to each serotype.[8]

TYPES OF POLIO

1-Paralytic polio

1% of poliovirus spreads along certain nerve fiber pathways, replicating and destroying motor neurons within the spinal cord, brain stem, and motor cortex. It leads to the development of paralytic polio. The various forms of which spinal polio, bulbar polio and bulbospinal polio. It vary only the amount of neuronal damage and inflammation. That occurs, the region of the CNS affected. the neuronal cells lesions within spinal ganglia it also occur in the reticular formation, vestibular nuclei, cerebellar vermis, and deep cerebellar nuclei. Inflammation associated with nerve cell. Destruction often alters the colour and appearance of the grey matter in the spinal column; causing it to appear reddish and swollen other destructive changes associated with paralytic .It occur in the forebrain region, specially the hypothalamus and thalamus.[9] The symptoms of paralytic polio include high fever, headache, and the back and neck stiffness. weakness of various muscles, sensitivity to touch, to swallowing, muscle pain, loss of superficial and deep reflexes, ittitability, paresthesia (pins and needles), constipation, or difficulty urinating. The Paralysis generally develops 1 to 10 days after early symptoms begin, progresses for 2 to 3 days, it is usually complete by the time the fever breaks.[10] The likelihood of developing paralytic polio increases with age, as does the extent of paralysis. In children, non-paralytic meningitis is the most likely consequence of CNS involvement, and paralysis occurs in only one in 1000 cases. In adults, paralysis occurs in one in 75 cases[11] paralysis of one leg is most common in under five years of children's, and in adults paralysis of the chest and abdomen also it affecting all four limbs is more likely. paralysis rates also depending on serotypes of the infecting polio virus, the highest rate of

paralysis 1 in 200are associated with polio virus type 1 and the lowest rates 1 in 2,000 are associated with polio virus type 2.[12]

Spinal polio

Spinal polio is the most common form of paralytic poliomyelitis, the results from viral invasion of the motor neurons of the anterior horn cells, and the ventral (front) grey matter section in the spinal columns, which are responsible for movement of the muscles, including those of the trunk, limbs and the intercostals muscles. Virus can causes inflammation of the nerve cells, it leading to damage or destroying the motor neuron ganglia. When the spinal neurons die and degeneration takes place, leading to weakness of those muscles formerly innervated by the now-dead neurons. The destruction of nerve cells, the muscles no longer receive signals from the brain and spinal cord, without nerve stimulation, the muscles atrophy, becoming weak, floppy and poorly controlled, and finally completely paralyzed. Progression to maximum paralysis is rapid (2 to 4 days), and it usually associated with fever and muscle pain. Deep tendon reflexes are also affected, and are usually absent or diminished; sensation in the paralyzed limbs, however, is not affected.[13] The extent of the spinal paralysis is depends on the region of the spinal cord affected, which may be cervical, lumbar or thoracic. The polio virus may affect muscles on both sides of the body, but more often the paralysis is asymmetrical. Any combination of limbs may be affected one leg, one arm, or both legs and both arms.[14]

Bulbar polio

In about 2% of cases of paralytic polio, bulbar polio occurs when poliovirus invades and destroys the nerves within the bulbar region of the brain stem. In The bulbar region the white matter pathway that connects to the cerebral cortex to the brain stem. The destruction of these nerves weakens the muscles supplied by the cranial nerves, it producing symptoms of encephalitis, and they causes difficulty in breathing, speaking and swallowing. Critical nerves affected the glossopharyngeal nerve, they are partially controls swallowing and functions in the throat, tongue movement and taste. The vagus nerve, which sends signals to the heart, intestines, and lungs. The accessory nerve, which controls upper neck movement. Due to the effect on swallowing, secretions of mucus, may build up in the airway they causing suffocation. Other signs and symptoms include facial weakness it caused by destruction of the trigeminal nerve and facial nerve, which innervate the cheeks, tear ducts, gums, and muscles of the face. In the ther structures; double vision; difficulty in chewing; and abnormal

respiratory rate, depth, and rhythm, which may lead to respiratory arrest. The Pulmonary edema and shockare also possible, and may be fatal.[15]

Bulbospinal polio

The approximately 19% of all the paralytic polio cases have both bulbar and spinal symptoms; this subtype is called respiratory or bulbospinal polio. the virus affects the upper part of the cervical spinal cord (cervical vertebrae C3 through C5), and paralysis of the diaphragm occurs. The critical nerves affected the phrenic nerve, which drives the diaphragm to inflate the lungs, and those that drive the muscles needed for swallowing. By destroying these nerves, form of polio affects breathing making it difficult for the patient to breathe without the support of a ventilator. It leads to paralysis of the arms and legs and it may also affect swallowing and heart functions.[16]

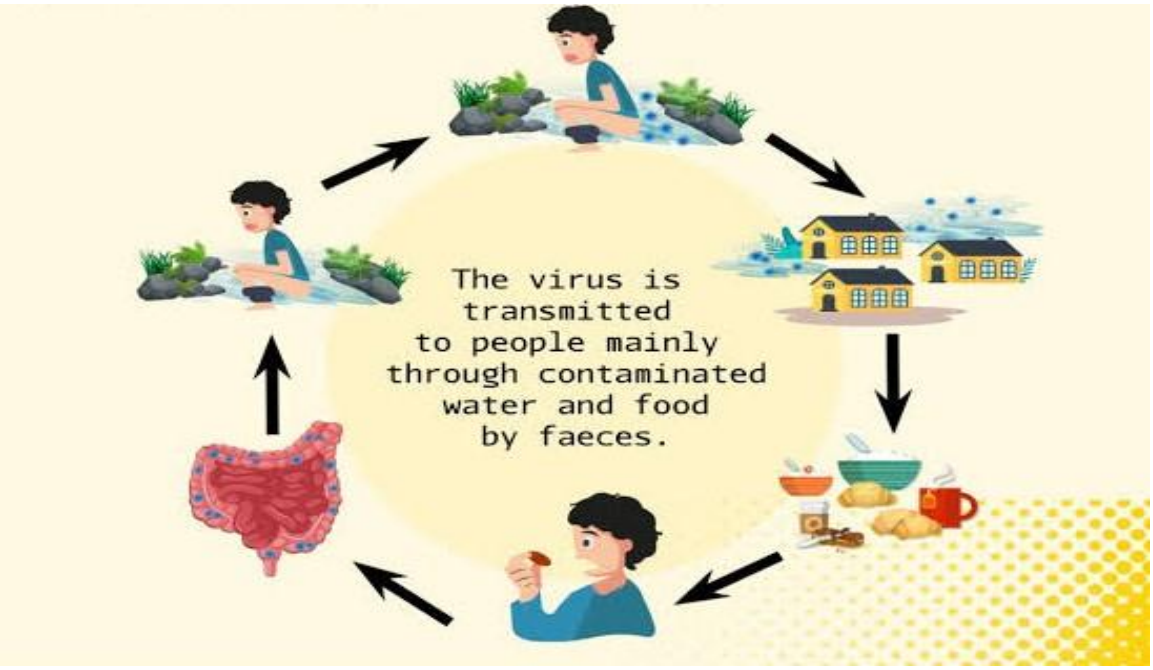

Fig. 3: Transmission of Polio Virus.

PATHOPHYSIOLOGY

Poliovirus enters the body through the mouth, they can infecting the first cells with which it comes in contacts to the pharynx and intestinal mucosa. It gains entry by binding to an immunoglobulin-like receptor, it is known as the poliovirus receptor or CD155. and the cell membrane. The virus then attacks the host cells of own machinery, and they begins to replicate. Poliovirus is divides within the gastrointestinal cells for about a week, from where it spreads to the tonsils specifically the follicular dendritic cells residing within the tonsilar

germinal centers, In the intestinal lymphoid tissue including the M cells of Peyer's patches, and the deep cervical and mesenteric lymph nodes, where it multiplies abundantly. The virus is subsequently absorbed into the bloodstream.[17] In the presence of virus the bloodstream enables to widely distributed throughout the body. The Poliovirus can survive and multiply within the blood and lymphatics for long periods of time, sometimes as long as 17 weeks. In a small percentage of cases, virus can spread and replicate in other sites, such as brown fat, the reticuloendothelial tissues, the muscles. This sustained replication causing a major viremia, and it leads to the development of minor influenza, like symptoms. Rarely, this may progress and the virus may invade the central nervous system, provoking a local inflammatory response. In most cases, this causes a self-limiting inflammation of the meninges, the layers of tissue surrounding the brain, which is known as non-paralytic aseptic meningitis. Penetration of the Central nervous system, provides Unknown benefit to the virus; it is quite possibly an incidental deviation of a normal gastrointestinal infection. The mechanisms by which poliovirus spreads to the Central nervous system are poorly understood, but it appears to be primarily a chance event largely independent of the age, gender of the individual.[18]

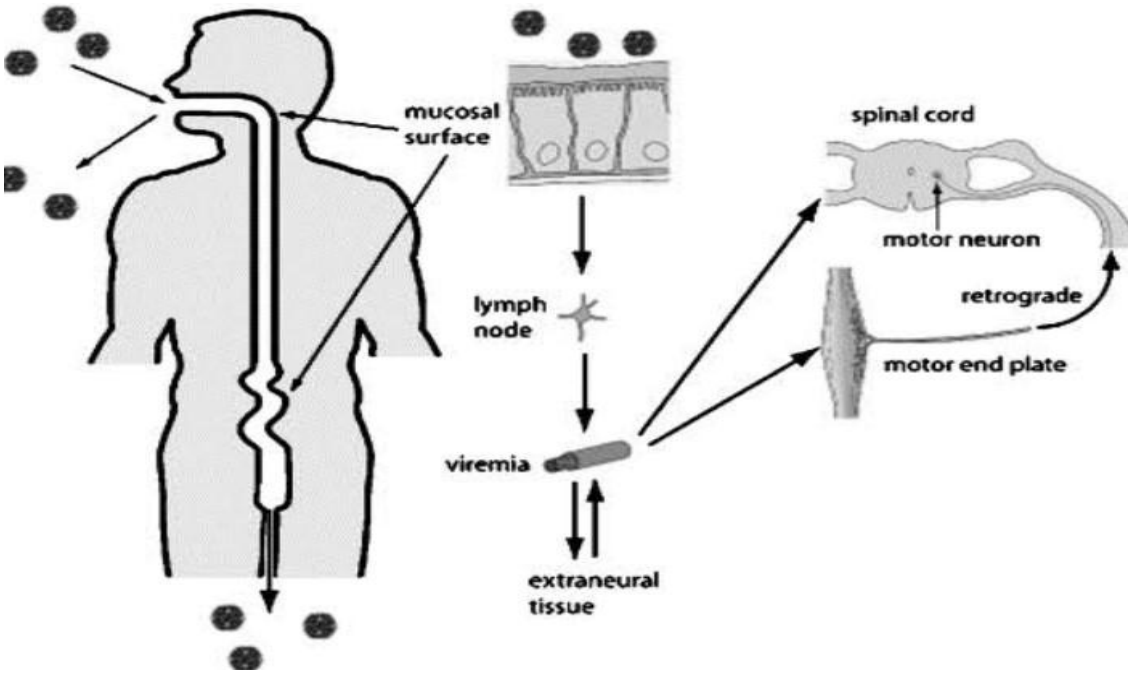

Fig. 4: Pathogenesis Of Polio Virus.

POLIO VACCINE

Polio vaccines are used all over the world to combat poliomyelitis or polio. The first was developed by Jonas Salk and first tested in 1952. Announced to the world by Salk on April

12, 1955, it consists of an injected dose of inactivated poliovirus. the oral vaccine was developed by Albert Sabin using attenuated poliovirus. A Human trials of Sabin's vaccine began in 1957, and it was licensed in 1962.[19] Because there is no long term carrier state for poliovirus in immune competent individuals, polioviruses have not primate reservoir in nature. survival of the virus in the environment for an extended period of time appears to be remote. Therefore, the interruption of person to person transmission of the virus by vaccination is the critical step in global polio eradication. There are two polio vaccines have eliminated polio from most countries in the world. It reduced the worldwide incidence from an estimated 350,000 cases in 1988 to 1,652 cases in 2007.[20]

Storage condition: Polio virus store in -200C for 24 months, 00C for 6 months and 40C for 3 months.[21]

TYPE OF VACCINE

1. Inactivated vaccine

In the University of Pittsburgh the Jonas Salk was first developed the first effective vaccine in 1952, but it would require years of testing. To encourage patience, Salk went on a CBS radio to report a successful test on a small group of children and adults on March 26, 1953, and two days later the results were published in JAMA.[22] The inactivated poliovirus vaccine, or Salk vaccine, is based on three wild, virulent reference strains, Mahoney (type 1 poliovirus), MEF-1 (type 2 poliovirus), and Saukett (type 3 poliovirus),they grown in a type of monkey kidney tissue culture, which are then inactivated with formalin. The injected Salk vaccine are confers IgG-mediated immunity in the bloodstream. in which they prevents polio infection from progressing to viremia and protects the motor neurons, thus they eliminating the risk of bulbar polio and post-polio syndrome. in Beginning of February 23, 1954, the polio vaccine was tested at Arsenal Elementary School and the Watson Home for Children in Pittsburgh, Pennsylvania. Salk's vaccine was used in a test called the Francis Field Trial, by Thomas Francis, the largest medical experiment in history. Some test began with 4,000 children at Franklin Sherman Elementary School in McLean, Virginia, and would eventually involve 1.8 million children, in 44 states from Maine to California.[23] By the conclusion of the study, roughly 440,000 received one or more injections of polio vaccine, in about 210,000 children received a placebo, consisting of harmless culture media, and 1.2 million of children received no vaccination and they served as a control group. Who would then be observed to see, if any contracted polio. The results of the field trial were announced on April 12, 1955.

The Salk vaccine had been 60–70% effective against polio PV1 (poliovirus type 1), over 90% effective against PV2 and PV3, and 94% effective against the development of bulbar polio. Soon after Salk's vaccine was licensed in 1955, the children's vaccination campaigns were launched. In the United States following a mass immunization campaign promoted by the March of Dimes, the annual number of polio cases fell from 35,000 in 1953 to 5,600 by 1957. By 1961 only the 161 cases were recorded in the United (US).[24] An enhanced potency polio virus was licensed in the United States in November 1987, and is currently the vaccine of choice in the United States(US). The first dose of polio vaccine is given shortly after birth of child; usually between 1-2 months of age. a second dose is given at 4 months age of child. The timing of the third dose depends on the vaccine formulation but should be given between 6-18 months age of child. A booster vaccination is given at 4 to 6 years of age, for a total of four doses at before school entry. Most of the countries, a fifth vaccination is given during teens. Routine vaccination of adults 18 years of age and older in developed countries is not necessary neither recommended because most adults are already immune and have a very small risk to wild poliovirus in their home countries.[25] In 2002, a 5-component (pentavalent) combination vaccine called as Pediarix. they containing inactivated polio vaccine was approved for use in the United States(US). The vaccine also contains combination of diphtheria, tetanus, and cellular pertussis vaccines and a pediatric dose of hepatitis B vaccine. In the UKIPV is combined with diphtheria, Tetanus, and pertussis type b vaccines. When the current formulation of IPV is used, 90% or most of individuals develop protective antibody to all three serotypes of poliovirus. After two doses of inactivated polio vaccine, and at least 99% are immune to poliovirus following three doses. The duration of immunity induced by inactivated polio vaccine is not known with certainty, although a complete series is thought to provide protection for many years.[26]

2. Oral vaccine

Oral polio vaccine is produced by the passage of the virus through non-human cells at a sub-physiological temperature, which produces spontaneous mutations in the viral genome.[27] the Oral polio vaccines was developed by several groups, one of which was led by Albert Sabin. And other groups, led by Hilary Koprowski and H.R. Cox, developed their own attenuated vaccine strains. In 1958, the National Institutes of Health created a special committee on live polio vaccines. The various vaccines were carefully evaluated for their ability to induce the immunity to polio, while retain a low incidence of neuropathogenicity in monkeys. in Large scale clinical trials performed in the Soviet Union in late 1950s — early 1960s by Mikhail

Chumakov and his colleagues demonstrated safety and high efficacy of the vaccine. Based on these results, the Sabin strains were chosen for worldwide distribution.[28] There are 57 nucleotide substitutions which distinguished the attenuate Sabin 1 strain from its virulent parent, two nucleotide substitutions attenuate the Sabin 2 strain, and 10 substitutions are involved in attenuating the Sabin 3 strain. The primary attenuating factor common to the all three Sabin vaccines is a mutation located in the internal ribosome entry site of virus's (IRES). which alters stem loop structures, and they reduces the ability of poliovirus to translate its RNA template within the host cell. The poliovirus in the Sabin vaccine replicates very efficiently in the gut, the primary site of infection and replication, but is unable to replicate efficiently within nervous system tissue. oral polio vaccine also proved to be superior in administration, eliminating the need for sterile syringes and making the vaccine more suitable for mass vaccination campaigns. Oral polio vaccine also provided long lasting immunity than the Salk vaccine.[29] In 1961, type 1 and 2 monovalent oral poliovirus vaccine was licensed, and in 1962, type 3 monovalent oral polio vaccine was licensed. In 1963, trivalent oral polio vaccine was licensed, and became the vaccine of choice in the United States and most the other countries in the world, largely replacing the inactivated polio vaccine. A second wave of mass immunizations leads to a further dramatic decline in the number of polio cases. Between 1962 and 1965 about 100 million Americans received the Sabin vaccine. The result was a substantial reduction in the number of polio disease cases; even from the much reduced levels following the introduction of the Salk vaccine.[30] Oral polio vaccine is usually provided in v10-20 doses of vaccine. A single dose of oral polio vaccine usually in two drops contains 1,000,000 infectious units of Sabin 1 polio virus vaccine, 100,000 infectious units of the Sabin 2 strain, and 600,000 infectious units of Sabin 3. The vaccine contains small traces of antibiotics neomycin and streptomycin but it does not contain preservatives. One dose of oral polio vaccine produces immunity to all three poliovirus serotypes. in approximately 50% of recipients. Three doses of live attenuated oral polio vaccine produce protective antibody to all three poliovirus. in more than 95% of recipients. Oral polio vaccine produces excellent immunity in the intestine, the primary site of wild poliovirus entry, which helps prevent infection with wild virus in areas where the polio virus is endemic. The polio virus used in the vaccine is shed in the stool and can be spread to others within a community, resulting in protection against poliomyelitis even in individuals who have not been directly vaccinated. Inactivated polio vaccine produces less gastrointestinal immunity than doe's oral polio vaccine, and primarily acts by preventing the virus from entering the nervous system. In regions without the wild poliovirus the inactivated

polio vaccine is the vaccine of choice. In the higher incidence in the region of higher incidence of polio, and thus the different relative risk between the efficacy and reversion of the vaccine to a virulent form, live vaccine is still used. The live virus also has stringent requirements for storage and transport. Which is a problem in some remote areas? As with other live-virus vaccines, immunity initiated by oral polio vaccine is probably lifelong.[31]

3. Latrogenic (Vaccine induced) polio

The major concern about the oral polio vaccine is known as ability to revert to a form that can achieve neurological infection and cause paralysis. The Clinical disease, including paralysis, caused by vaccine derived poliovirus, is indistinguishable from that caused by wild polio viruses. This is believed that a rare event, but outbreaks of vaccine are associated paralytic poliomyelitis have been reported, and it tends to occur in areas of low coverage by oral polio vaccine, because the oral polio vaccine is itself protective against the related outbreak strain.[32] As the incidence of wild polio diminishes, nations transition from use of the oral vaccine back to the injected vaccine because the direct risk of iatrogenic polio due to oral polio vaccine outweighs the indirect benefit of immunization via subclinical transmission of oral polio vaccine. When inactivated polio vaccine is used, reversion is not possible but there remains a small risk of clinical infection upon exposure to reverted oral polio vaccine or wild polio virus. At the mid of 1950s the polio vaccine are widespread use. The incidence of polio disease declined rapidly in many industrialized countries. The use of oral polio vaccine was discontinued in the United States in 2000 and in 2004 in the UK, but it continues to be used around the globe. The rate of vaccine associated paralytic poliomyelitis varies by region but is generally about 1 case per 750,000 vaccine recipients.[33] Vaccine associated paralytic polio is more likely to occur in adults than in children. In immunodeficiency in children, the risk of vaccine associated paralytic polio is almost 7,000 times higher, particularly for persons with B-lymphocyte disorder, which can reduce the synthesis of protective antibodies. The World Health Organization considers the benefits of vaccination to much outweigh the risk of vaccine derived polio. Outbreaks of vaccine derived polio have been stopped by multiple rounds of high-quality vaccination, in order to immunize the entire population.[34] Outbreaks of vaccine associated paralytic polio occurred independently in Belarus in 1965-66, in Canada 1966–68, in Egypt 1983–1993, in Hispaniola 2000 -2001, in philippines 2001, in Madagascar 2001–2002, and in Haiti 2002, where political strife and poverty have interfered with vaccination efforts.[35] In 2006 an outbreak of vaccine-derived poliovirus occurred in China. Cases have been reported from Cambodia (2005–2006), Myanmar (2006–2007), Iran

(1995, 2005–2007), Syria, Kuwait and Egypt. Since 2005, The World Health Organization has been tracking the vaccine caused polio in northern Nigeria caused by a mutation in live oral polio vaccines.[36]

Epidemiology

As a result of vaccination, there has been a dramatic reduction in the incidence of poliomyelitis globally. However, the disease still remains endemic in four countries – Nigeria, India, Pakistan and Afghanistan. In other countries also have cases of wild type poliomyelitis from time to time, due to importation. These efforts have reduced the number of annual diagnosed cases by 99%; from an estimated 350,000 cases in 1988 to a low of 483 cases in 2001, after which it has remained at a level of about 1,000 cases per year (1,606 in 2009).[37] The World Health Organization aimed to eradicate poliomyelitis by the year 2005. Although this was not successful, world health organization is still hopeful that polio eradication will be achieved by 2010 or soon after, primarily through 'national polio days' where all children in a certain region are given oral polio vaccine. Poliomyelitis has been a notifiable disease in Australia since 1922. The highest recorded incidence of poliomyelitis in the country 39.1 per 100,000 populations was in 1938. The last polio epidemic in Australia was in 1961–1962.[38] Mass vaccination against polio using intramuscular Salk inactivated polio vaccine first started in Australia in 1956. In 1966, inactivated polio vaccine-Salk was replaced by Sabin oral polio vaccine in the publicly funded immunisation program. Oral polio vaccine is particularly suited to provide mass protection against wild-type polio. As expected, good vaccine coverage with oral polio vaccine over several years lead to cessation of indigenous transmission of wild polio virus infections in Australia.[39] in Australia the last reported case of locally acquired wild type polio in 1972. In 1986, there was a case of poliomyelitis reported in a 22 year old that was initially thought to be wild-type virus, but was later confirmed as a vaccine like strain. 8-10 In October 2000, Australia together with the other 36 countries in the Western Pacific Region was declared 'polio free' by the world health organization. This certification of 'polio-free' status is confirmation of interruption of indigenous poliovirus transmission and containment of wild polio virus in the country.[40] Until global eradication of polio is achieved, all countries are at risk of polio infection. In 2005, the 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 polio virus and resulted in a large polio epidemic, with more than 200 cases.[41] As of 2012, polio remains endemic in only three

countries: Nigeria, Pakistan, and Afghanistan, although it continues to cause epidemics in other nearby countries due to hidden or re-established transmission. For example, despite eradication 10 years prior, an outbreak was confirmed in China in September of 2011 it involving a strain prevalent in neighbouring Pakistan. Since in January 2011, there was no reported cases of the disease in India, and hence in February 2012, the country was taken off the world health organization list of polio endemic countries. It is reported, if there are no cases of polio in the country for two more years, it will be declared as a polio-free country.[42]

LABORATORY DIAGNOSIS

1. Viral Isolation:- Poliovirus may be recovered from the stool or pharynx from a person with presumed poliomyelitis. The Isolation of virus from the cerebrospinal fluid (CSF) is diagnostic but is rarely accomplished. If poliovirus is isolated from a person with acute flaccid paralysis, it must be tested further, using oligonucleotide mapping or genomic sequencing, to determine if the virus is wild-like or vaccine-like.

2. Serology:- Neutralizing antibodies appear early and may be at high levels by the time the patient is hospitalized and, therefore, a 4-fold rise may not be demonstrated.

3. Cerebrospinal Fluid:- The CSF in poliovirus infection usually contains an increased number of white blood cells (10 to 200cells/mm3, primarily lymphocytes) and a mildly elevated protein from 40 to 50 mg/100 ml.

PREVENTION

1. Passive Immunization:- In 1950, William Hammon at the University of Pittsburgh purified the gamma globulin component of the blood plasma of polio survivors. Hammon proposed the gamma globulin, which are contain antibodies to poliovirus, could be used to halt poliovirus infection, prevent disease, and reduce the severity of disease in other patients who had contracted polio virus. The results of large clinical trial be promising, the gamma globulin was shown to be about 80% effective in preventing the development of paralytic poliomyelitis. It was also shown to reduce the severity of the disease in patients who developed polio. The gamma globulin approach was later deemed impractical for widespread use, however, due to large part to the limited supply of blood plasma, so the medical community turned it focus to the development of a polio vaccine.[43]

2. Vaccine:- There are Two types of vaccine are used throughout the world to combat polio. Both types induce immunity to polio, efficiently blocking person-to-person transmission of wild poliovirus, thereby protecting both individual vaccine recipients and the wider

community (so-called herd immunity).[44] The first candidate polio vaccine, based on one serotype of a live but attenuated virus, was developed by the virologist Hilary Koprowski. In February 27, 1950, the Koprowski's prototype vaccine was given to an eight year old boy. The Koprowski continued to work on the polio vaccine throughout the 1950s, leading to large scale trials in the then Belgian Congo and the vaccination of seven million children in Poland against serotypes polio virus type 1 and polio virus type 2 between 1958 and 1960.[45] The second inactivated polio virus vaccine was developed in 1952 by Jonas Salk at the University of Pittsburgh, and announced to the world on April 12, 1955. The Salk vaccine, or inactivated poliovirus vaccine, is based on poliovirus grown in a type of monkey kidney tissue culture, which is chemically inactivated with formalin. After two doses of inactivated polio vaccine, 90% or more of individuals develop protective antibody to all three serotypes of poliovirus, and at least 99% are immune to poliovirus following three doses.[46] Subsequently, Albert Sabin developed another live, oral polio vaccine (OPV). It was produced by the repeated passage of the virus through nonhuman cells at sub physiological temperatures. The attenuated poliovirus in the Sabin vaccine replicates very efficiently in the gut, the primary site of wild poliovirus infection and replication, but the polio vaccine strain is unable to replicate efficiently within nervous system tissue. A single dose of Sabin's oral polio vaccine produces immunity to all three poliovirus serotypes in about 50% of recipients. there are Three doses of live-attenuated oral polio vaccine produce the antibody to all three poliovirus types in more than 95% of recipients. In Human trials of Sabin’s vaccine began in 1957, and in 1958 it were selected, in competition with the live vaccines of Koprowski and other researchers, by the United States National Institutes of Health. Licensed in 1962, it rapidly became the only polio vaccine used worldwide.[47]

Fig. 5: Prevention of Polio Virus.

NATIONAL IMMUNISATION PROGRAM (NIP)

Children:- Children are recommended a primary course of 3 doses of an IPV-containing vaccine at 2, 4 and 6 months of age and a booster dose at 4 years of age. The recommended interval between 2 doses is 2 months, but, for catch up, the minimum interval can be in 1 month. In the Australia, a 3-dose primary schedule and a booster at 4 years of age provides adequate protection. Therefore, for those children who have received a complete course of polio vaccine during childhood, a further booster dose is not required later in life unless they are at increased risk of infection as below.

Adults: - The schedule for unvaccinated adults is 3 doses administered at intervals of 1–2 months.

Booster Doses:- A booster dose is not required for fully vaccinated children or adults unless they are at increased risk of infection, such as travelling to areas or countries where poliomyelitis is epidemic healthcare workers, including laboratory workers, in possible contact with poliomyelitis cases. For such exposed to a continuing risk of infection, booster doses are desirable every 10 years. There is no cure for polio disease. The focus of modern treatment has been providing relief of symptoms, speeding recovery and preventing complications. Supportive measures include antibiotics to prevent infections in weakened muscles, analgesics for pain, nutritious diet and moderate exercise. Treatment of polio often requires long-term rehabilitation, including physical therapy, braces, corrective shoes and, in

some cases, orthopedic surgery.[48] Portable ventilators may be required to support breathing. Historically, negative-pressure ventilator, more commonly called an iron lung, it was used to artificially maintain respiration during an acute polio infection until a person could breathe independently (generally about one to two weeks). Today, many polio survivors with permanent respiratory paralysis use modern jacket type negative pressure ventilators worn over the abdomen and chest.[49] The Other historical treatments for polio including hydrotherapy, electrotherapy, massage and passive motion exercises, and surgical treatments, such as tendon lengthening and nerve grafting. The Devices.

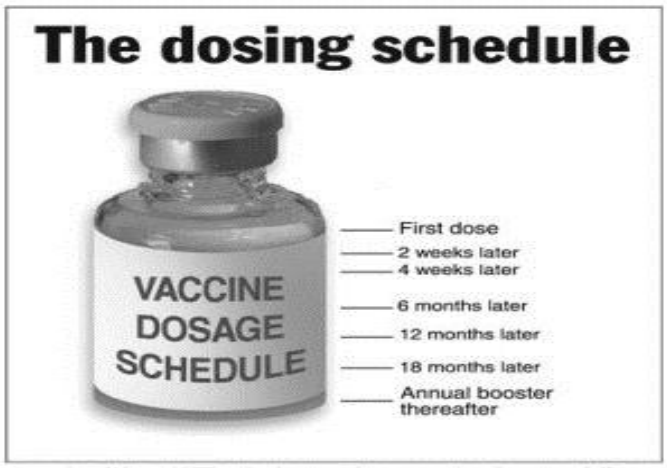

Fig. 6: The Dosing Schedule of Polio Virus.

Such as rigid braces and body casts which tended to cause muscle atrophy due to the limited movement of the user were also touted as effective treatments.

Natural Ayurvedic Treatment of Polio:- Polio is a very deadly virus and though partly eradicated through an extensive worldwide immunization programme it is still around and wreaking its deadly havoc upon parts of the population. It is a highly infectious disease and enters the body through the mouth and goes onto attack the central nervous system. The cause of that despite extensive medical research has yet to be found. Though many who catch the virus survive most that do are left with a form of paralysis, usually in an arm or a leg.

it is unable to offer a cure, Ayurvedic can alleviate the pain experienced by sufferers and can also the recovery greatly.

Ayurvedic Cause of Polio:- Paralysis or more specifically polio is caused according to Ayurvedic when there is a divergence in vata-pitta-kamba which affects the dhosa (energies

of the body). This physiological shift causes toxins (ama) to build up and attack the nervous system. To be treated positively Ayurvedic says that there must be a re-balancing of these within the body. To achieve this state the following methods should be applied.

Ayurvedic Remedies For Polio:- As previously stated any form of the Ayurvedic programme can not cure polio. That takes two Herbal remedy and intensive Ayurvedic massaging. The herbal remedies can eradicate strengthen the immune system and internal disorder.

The Massage alleviates the ongoing pain and any paralysis that may have occurred. the instance intensive massages repair the wasted and dead muscle. Massaging also allows the affected area to receive nutrients which can expel toxins.

Massage Applied To Remedy Polio In Ayurvedic:- As possibly befits the remedy for such a deadly serious disease like polio, Ayurvedic prescribes arguably its' most important massage form in the treatment of the disease, the method known as Navarakizhi and pindasvedka. In This treatment revitalized mainly the skin and it is also used with Ayurvedic anti-ageing treatments. However in achieving this it also rejuvenates dead and wasted muscles which are why it is used as a remedy for paralysis, which is why it used on polio sufferers.

The Revitalizing Agent In Pindasvedka Or Navarakizhi:- Pindasvedka or Navarakizhi is a simple remedy consisting of medicated rice. The rice provides carbohydrates which rejuvenate the affected areas when applied. Pindasvedka or Navarakizhi is applied in Ayurveda. IN this a fine cloth of the medicated rice solution is placed upon the patients' body. It creates gradual warmth and is applied until the patient builds up a great sweat. The act of sweating expels the toxins. It is mainly massage based. But the imbalances caused by the pitta-vatta-kamba shift that disrupts the internal systems of a polio patient can be put right by ingesting certain herbs and spices. They can be added to cooking and will aid the healing processes of paralysis sufferers.

Herbs and spices used in the Ayurvedic cure for polio.

1-Fennel

2-Ginger

3-Black pepper

4-Coriander

5-Licorice

CONCLUSION

The above review revels that the rehabilitation in patients with post-polio syndrome should take the multi professional and multidisciplinary approach, with an emphasis on physiotherapy, including enhanced & individually modified physical activity, and muscle training. Patients with post-polio syndrome should be advised to avoid both inactivity and over use of the weak muscles.

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