Poliovirus and Poliomyelitis Vaccines: A Review

Faten S. B. Gazzaz


This paper discusses poliomyelitis, a viral disease with serious sequelae, that remains a health problem in Saudi Arabia. The points discussed are related to aetiology, isolation of the viral agent, laboratory diagnosis, epidemiology and clinical manifestations of poliomyelitis. Special emphasis is placed on the types of poliomyelitis vaccines, their advantages and disadvantages. The purpose of this article is to call attention to this important disease and to stress the need for conducting more studies in order to eradicate this virus from our community.

Poliomyelitis is an acute infectious disease that in its serious form, affects the central nervous system. The destruction of motor neurons in the spinal cord results in flaccid paralysis. However, only a small number of infections are clinically apparent. Within a period of 1 year (September 1985 to September 1986), 150 poliomyelitis patients were reported to have visited the Riyadh Medical Rehabilitation Center.1 Of the 150 patients, six (4%) were <1–5 years of age, 24 (16%) were 5–10 years, 83 (55.3%) were 10–20 years, 30 (20%) were 20–30 years and seven (4.7%) were 30–40 years. There was a slight male preponderance, with a ratio of 58.6% males to 41.3% females. The majority of patients presented with lower limb paralysis—approximately 92% of the patients. Poliomyelitis is endemic in Saudi Arabia and most of the surrounding countries.1

Aetiology
Poliomavirus was discovered in 1909 by Landsteiner and Popper. There are three serologically distinct groups of polioviruses which have characteristics in common that place them into the Picornaviridae family. They are small, 25–30 nm in size, have icosahedral symmetry, with 32 capsomeres; they are ether-sensitive, single-stranded RNA-containing viruses.2,3 Poliovirus attaches to specific cell receptors. The presence of these receptors controls the susceptibility to the infection by the virus. The human chromosomal location of the genes controlling this receptor is 19q13-qter. Some viral isolates can be identified as vaccine-like, and others as wild-like.

Isolation of the Viral Agent and Laboratory Diagnosis
Stool samples or rectal swabs are the specimens1 of choice for isolation of enteroviruses; cerebrospinal fluids and/or throat swabs can also be used.3,4 The three types of polioviruses grow readily in primary monkey kidney cells,2 and/or human cell lines.2,5 These consist of rhesus monkey kidney (Rh MK), or African green monkey kidney (GMK), human diploid fibroblast (HDF), WI-38, human embryonic kidney (HEK) and/or Hep-2 cell lines, depending upon what is used.4 The optimum temperature for growth is 37°C. The observation for cytopathic effect (CPE) in fluid cultures is the most sensitive and rapid method for recognition of enterovirus infection in cell cultures. Extensive cell destruction usually occurs 24–48 h after inoculation into the cell systems.4 For serological diagnosis, two paired sera (acute and convalescent) are needed.3 A four-fold (or more)
increase in antibody titre by complement fixation (CF), or particularly, by neutralizing antibodies means infection.²

**Epidemiology**

In addition to their pathogenicity in humans, the polioviruses produce paralysis in monkeys.⁴ They are generally transmitted by human carriers by direct or indirect faecal–oral spreads. Flies have been found to be contaminated by the virus in heavily infected areas, and may pick it up from sewage.²³ Poor personal hygiene, inadequate disposal of human faecal wastes, poor sewage management, and cross-contamination of water supplies and food are reflected in widespread dissemination of wild virus and maintenance of poliomyelitis in the population.

Wild poliovirus disease is exceedingly uncommon in immunized populations;² when it occurs, it tends to affect immigrants incubating the disease, non-immunized groups within the larger population, and sporadic, local, endemic foci following introduction of a case.²

**Manifestations**

The incubation period of poliovirus from contact to initial clinical symptoms is 8–12 days. Infection by one of the polioviruses may result in asymptomatic infection, or an influenza-like illness, termed abortive poliomyelitis and characterized by fever, malaise, anorexia, headache, sore throat and abdominal or muscular pain, which usually subsides or leads to non-paralytic disease with aseptic meningitis. Either of these may develop into paralytic poliomyelitis, which may also occur as a first phase, usually with flaccid paralysis, but brain stem invasion may cause spasticity of non-paralysed muscles.²⁵ Recovery may take 6 months and residual paralysis can occur. The virus first multiplies in the tonsils, lymph nodes of the neck and Peyer’s patches of the intestines.² The central nervous system (CNS) may be invaded through the bloodstream, rather than along peripheral nerves, as was once believed. Some picorna-viruses cause paralysis not distinguishable on the basis of clinical features from paralytic poliomyelitis.

**Vaccines**

There are two types of vaccines available:²⁵

- **Inactivated virus vaccine (Salk)**

  This is a virus grown in cell culture and killed by formalin.

  Its advantages are that it is safe (no living virus); there is no back mutation; there is no problem using the vaccine in immunocompromised patients; it can be incorporated in mixed vaccine preparations; it is both effective (greatly reduces numbers of cases) and useful in tropical areas where live virus vaccine is interfered with by other viruses.

  Its disadvantages are that it needs boosters and is more expensive. Also, as virulent polio is used in its preparation there is the possibility of accidents.

- **Live attenuated virus (Sabin)**

  This is grown in human diploid cell cultures.

  Its advantages are that it is easily administered by mouth, relatively inexpensive and results in secretion of IgA into the gastrointestinal tract (GIT), preventing re-infection. It gives life-long immunity; herd immunity is certain.

  Its disadvantages are that as it is a live virus there is the possibility of back mutation; it spreads to household contacts and the community; in certain areas such as the tropics there is a poor take (interference by other viruses). It is also contra-indicated in immunocompromised patients. There is evidence of excretion of the virus by recently immunized health care workers.

**Discussion**

The poliomyelitis vaccine available in Saudi Arabia is usually the Sabin oral polio vaccine which is a stabilized suspension of types 1, 2, and 3. Live attenuated poliomyelitis viruses (Sabin strains), are obtained by propagation of the viruses in monkey kidney tissue cultures.²⁵ The antibodies formed following oral immunization with the Sabin polio vaccine are primarily IgA antibodies limited to the GIT.⁶ On the other hand, the antibodies synthesized following parenteral immunization with the killed poliovirus (Salk vaccine), are primarily IgG antibodies, localized in the circulation. Thus parenteral immunization with the killed virus results in immunity to systemic infection with the poliovirus, but does not prevent the establishment of the carrier state for a limited time. On the other hand, oral immunization with the live, attenuated virus, results in long-term secretory IgA synthesis and prevention of the carrier state, but no systemic immunity.⁶ The oral poliomyelitis vaccine (OPV) consists of live viruses, which replicate in the human intestinal tract and are excreted from the body for several weeks.²⁵ The unavoidable spread of infectious, attenuated vaccine virus through pharyngeal and faecal excretion can effectively immunize unvaccinated individuals who come in close contact with the vaccinees.⁵ The oral vaccine provides a substantial public health benefit, by protecting a number of people who were never intentionally vaccinated.

The IgA proteins are the predominant immunoglobulins in secretions bathing mucosal surfaces.⁶ These tissues are frequently in direct continuity with, and exposed to, the external environment and, therefore, provide the portals of entry and the first line of defence for the penetration and invasion.
of the GIT by pathogenic microorganisms. The antibacterial and antiviral protection afforded by secretions is directly attributed to their content of specific IgA antibodies, although they also contain small quantities of IgG and traces of IgM antibodies. The ratio of IgA antibodies to IgG antibodies in secretions is usually the reverse of that which exists in the blood. Recent research confirms that the attenuated OPV virus in some cases undergoes genetic changes while in the human intestinal tract, regaining much of the neurovirulence it carried before attenuation.

Weiss called attention to vaccine-induced injuries and the war against polio. Because of the increasing availability of polio vaccines, the World Health Organization is trying, by the year 2000, to eradicate the disease, which leaves 250,000 individuals paralysed each year. The greatest drawback of the oral vaccine among the more developed countries is the low, but significant rate of serious paralytic complications associated with its use.

A new vaccine, combining E-IPV (enhanced inactivated poliovirus vaccine), originally developed by Jonas Salk and given by injection, with the traditional diphtheria-pertussis-tetanus (DPT) vaccine is under development in Canada and elsewhere, potentially simplifying delivery of the injectable E-IPV. With no serious side-effects associated with the E-IPV, the question emerges: is it necessary that the ideal immunization to poliovirus should consist of initial parenteral immunization with the killed virus, followed by oral immunization with the attenuated virus?

According to the report of the Institute of Medicine (IOM), of the National Academy of Sciences, a vaccine schedule with two or more doses of killed virus E-IPV, followed by booster doses of the oral vaccine would certainly reduce and possibly eliminate, cases of vaccine-associated paralysis in OPV recipients and their contacts. For now, the IOM report concludes, no change in the current policy of primary reliance on OPV is recommended. However, a mixed schedule of DPT, E-IPV and OPV should be taken into consideration and is to be expected within 2–5 years.

**Conclusion**

Large-scale studies will have to be conducted in Saudi Arabia to determine the efficiency of the currently used oral vaccine (Sabin), and also to draw up a schedule that would provide maximum protection against all the component antigens, to boost our continuously improving health care. Laboratory investigations of genetic susceptibility may be able to identify those individuals who are at greater risk of paralysis, either from natural disease, or oral poliovaccine, and also evaluate the roles of the different immunoglobulins to both vaccines in the provision of resistance.

**References**