

Polio: Is It Really Gone?

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ntil the development of widespread immunization programs, poliomyelitis, with its disabling effects, was a disease that struck fear and caused devastation throughout the world. Since the advent of the two immunizations against poliovirus in the 1950's and 1960's, there has been a dramatic decline in the incidence of *poliomvelitis* in Canada and the remainder of the western hemisphere. Despite successes in our country, global organizations such as the World Health Organization (WHO) continue to be frustrated in their pursuit of worldwide eradication of the virus. Polio has made

Poliovirus is a riborucleie acid (RNA) non-enveloped *enterovirus*. There are three serologic types, P1, P2 and P3, based on difference ince there ince t serotypes, protection from the disease requires antibodies against each of the three types.

How is Poliovirus transmitted?

Poliovirus is traditionally transmitted via the fecal-oral route. As a result, poor sanitation in underdeveloped areas of the world plays a very important role in maintaining the momentum of epidemics. Its prevalence in underdeveloped

countries also puts travellers to those areas at risk of infection. Most cases in North America are the result of infection from the live attenuated vaccine or oral polio vaccine (OPV). This can happen as a result of the attenuated virus reverting to a more virulent strain, but more commonly in immunocompromised patients receiving the live vaccine; there have been 11 reported cases of vaccine-associated paralytic polio (VAPP) in Canada since 1980. The risk of developing VAPP is about 1 per 2.5 million doses administered, but is 2000 times higher among immunodeficient patients.

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s'Although *poliomyelitis* is the most publicized and most feared outcome of poliovirus infection, asymptomatic infection is the most common course (80% to 90%). In 1% to 5% of cases, patients will develop non-paralytic polio (or abortive *poliomyelitis*): following a three day to six day incubation period, these patients will present with flu-like symptoms of:

- fever,
- malaise,
- sore throat,
- anorexia,
- myalgia and ٠
- headache.

This condition will typically resolve within three days.



Non-paralytic aseptic meningitis

Another 1% to 10% will develop non-paralytic aseptic meningitis. These patients will experience similar symptoms and exhibit similar signs to those with non-paralytic polio, but will also experience signs and symptoms of meningeal inflammation, such as neck and back stiffness and they can also experience muscle spasms. Examination of the cerebrospinal fluid (CSF) of these patients reveals:

- lymphocytic pleocytosis,
- normal glucose level,
- normal or slightly elevated protein levels.

Paralytic poliomyelitis

The least common presentation is that of paralytic poliomyelitis. After replication in the oropharynx and intestinal tract, the virus spreads hematogenously to the central nervous system (CNS). In the CNS, poliovirus preferentially replicates in the motor neuron cell bodies. Infection of these cells causes paralysis. After one day to several days, signs of aseptic meningitis are followed by severe back, neck and muscle pain and by the development of muscle weakness. In some cases, the disease appears to be biphasic, with aseptic meningitis followed first by apparent recovery, but then (one day or two days later) by the return of fever and the development of paralysis. Since the motor nerve cell bodies are affected, sensation remains normal, although patients may describe sensory symptoms. Most patients will recover some function within weeks-to-months of initial infection, but unfortunately, approximately two-thirds of patients will have permanent neurologic sequelae. Paralytic polio has traditionally been classified into three

categories, that of spinal polio, bulbar polio and bulbospinal polio.

- 1. Spinal polio: This category involves the infection of the motor neuron cell bodies in the spinal cord found in the anterior horn that primarily controls skeletal and respiratory muscles. Resultant weakness is typically asymmetric and more proximal than distal and the legs are the most commonly involved limbs. Although any patient can present with any degree of disability, infected children tend to have more isolated paralysis (*e.g.*, only one limb), while adults tend to have more generalized manifestations. Examination reveals typical lower motor neuron signs, such as:
 - weakness,
 - decreased or absent reflexes,
 - fasciculations and
 - decreased muscle tone.
- 2. Bulbar polio: Infection of the cranial nerve cell bodies can lead to:
 - dysphagia,
 - difficulty in handling secretions,
 - dysphonia or
 - respiratory insufficiency (due to aspiration, involvement of the respiratory centre in the medulla or paralysis of the phrenic nerve).

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Dr. Embil is a Consultant, Infectious Diseases and Associate Professor, University of Manitoba. He is also the Medical Director, Infection Prevention and Control Program, Health Science Centre and Winnipeg Regional Health Authority, Winnipeg, Manitoba. Severe medullary involvement may even lead to circulatory collapse. This can clearly be a life-threatening condition that may require indefinite respiratory support.

3. Bulbospinal polio: This category is simply a combination of spinal and bulbar polio.

What is post-polio syndrome?

Post-polio syndrome (PPS) presents as a new, but insidious onset of symptoms 10 years to 40 years after the initial infection. Symptoms include:

- weakness in either previously affected limbs or in limbs not thought to have been originally involved,
- general fatigue or exhaustion with minimal activity,
- myalgia,
- arthralgia,
- dysphagia,
- respiratory problems,
- sleep-related problems, (e.g., sleep apnea) and
- intolerance to cold.

Risk factors include the female gender and increasing time from acute infection. Although the pathophysiology is unclear, PPS is thought to be due to progressive dysfunction and loss of motor neurons that compensated for the neurons lost during the original infection and not to persistent or reactivated poliovirus infection.

How is polio diagnosed?

In addition to the clinical findings described above, the physician can seek to isolate the virus in a suspected case. The following cultures should be sent for poliovirus testing:

- throat,
- stool,
- serum and
- CSF.

Where available, polymerase chain reaction (PCR) analysis of these samples can be used to identify the virus, as it is highly sensitive (95%) and specific (80%) and is generally quicker than culture testing. In addition to confirming the diagnosis, these results can help to identify the source of the infection as either from a live (OPV) vaccine or from a wildtype virus. Polio is a reportable disease and any positive test results should be forwarded to the physician's provincial Ministry of Health.

The risk of developing VAPP is about 1 per 2.5 million doses administered, but is 2000 times higher among immunodeficient patients.

How is polio treated?

There is no cure for polio and as a result, supportive therapy is the only available option. The patient's requirements will dictate the therapy required, which can range from:

- physiotherapy,
- special braces and slings for affected limbs to
- ventilatory support for respiratory weakness.

The latter requirement spawned the now-infamous iron lung: weighing as much a small car,



the iron lung was effective at providing respiratory support but was clearly very restrictive.

How is polio prevented?

There are two polio vaccines available: the liveattenuated vaccine given orally (OPV) and the inactivated polio vaccine (IPV) given intramuscularly. Both vaccines offer protection against all three serotypes of the poliovirus.

IPV

Each vaccine has its advantages and disadvantages. IPV is considered to be safer to the recipient due to its inactivated form. However, it is somewhat less effective (90% after two initial doses given at least six weeks apart, but up to 100% effective with a booster six months to 12 months later). It also provides IgG humoral immunity, but very little to no IgA mucosal (or intestinal) immunity. As a result, it cannot prevent transmission. Although neither proven effective, nor recommended by Health Canada, booster IPV shots are frequently given to immunized patients who will be travelling to endemic areas.

OPV

OPV has the benefit of interrupting the spread of the virus to others by conferring intestinal (IgA, mucosal) immunity to the recipient. It provides lifelong immunity and can be given very easily since it is a single dose by mouth. The main disadvantage lies in the fact that it is a live-attenuated virus that can, in some cases, infect the recipient or the recipient's contacts. Another disadvantage when considering immunization in the developing world is the need for the vaccine to be refrigerated.

Table 1		
Current Health Canada immunization guidelines against poliovirus		
Age group	Immunization schedule	Comments
Children	2 months	• The 6 month immunization is not required,
	4 months	but can be given for convenience.
	6 months	
	18 months	
	4 years to 6 years	
Unimmunized adults	2 doses given at 4 week-	Routine immunization of unimmunized adults
	to-8 week intervals	is not considered necessary, due to neglibible
		risk of exposure to wild virus.
		 Immunization of adults is only recommended
		in those who are unimmunized and are at
		increased risk of exposure to the poliovirus
		(such as travellers to endemic areas,
		laboratory or healthcare workers).

Poliovirus

Because of the virtual eradication of polio in North America and the persistent risk of VAPP, OPV is no longer used in either Canada or in the US. IPV is the vaccine of choice (Table 1).

Conclusion

Through widespread vaccination, the general population may have forgotten its terrifying renown, but polio remains a very disabling and potentially life-threatening disease that with continued effort, could be completely eradicated. Physicians need to ensure vaccination of their patients and remember that the disease continues to exist. Continued preventative measures can help prevent North American outbreaks.

Additional reading

- Kidd D, Williams A, Howard RS: Classical diseases revisited poliomyelitis. Postgrad Med J 1996; 72(853):641-7.
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