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Poliomyelitis, Disease Overview

Many polio survivors now experiencing the late effects of polio have asked for information about the poliovirus and acute poliomyelitis. The following article is copied with permission from "Information on Poliomyelitis and Poliovirus Vaccine Live Oral Systemic." All rights reserved. ©1999 The United States Pharmacopeial Convention, Inc.

Poliomyelitis is a contagious disease occurring worldwide. It is caused by three types (serotypes 1, 2, and 3) of poliovirus, which is an enterovirus. The three serotypes are not cross-protective, which means that the individual must develop immunity to each type for complete protection against the disease.[1,2] In countries where poliomyelitis is endemic, the disease often is caused by poliovirus serotype 1, less frequently by poliovirus serotype 3, and least frequently by poliovirus serotype 2.[1]

Poliomyelitis can be transmitted directly by fecal-oral contact or indirectly by contact with infectious saliva or feces (or by contaminated sewage or water).[79,86] Polioviruses enter the mouth and replicate in the oropharynx and intestinal tract.[1,2] From there, the viruses are carried by the blood stream into the central nervous system (CNS), resulting in cell destruction of the motor neurons of the anterior horn and the brain stem.[1,2] (The exact mechanism by which the CNS becomes infected, however, remains uncertain and controversial. A study involving transgenic mice expressing the human poliovirus receptor suggested that poliovirus spreads from muscle to CNS by means of peripheral nerve muscle fibers, rather than directly from the blood stream.)[3] Motor function of the individual is therefore impaired while the sensory function remains unaltered.[1,2]

Paralytic symptoms usually occur 7 to 21 days from the time of initial infection (range is from 4 to 30 days). The period of communicability starts after viral replication, continuing as the virus is excreted in oral secretions and feces. Communicability ends when replication and excretion of virus cease, which usually occurs 4 to 6 weeks after infection. More than 90% of susceptible contacts become infected after household exposure to the wild poliovirus.[1]

Poliomyelitis can be diagnosed by recovery of polioviruses from throat secretions in the early phase of illness (first week), from feces (often for several weeks), and rarely from the cerebrospinal fluid (CSF). Virus isolates are classified as either wild-type (naturally occurring strains) or vaccine-like. Diagnosis also can be established by serologic testing to demonstrate seroconversion. Laboratory findings may include a normal or mildly elevated white blood cell count and CSF findings that are indistinguishable from other viral causes of aseptic meningitis.[4]

About 95% of poliomyelitis cases are asymptomatic and can be recognized only by the isolation of the virus from the feces or oropharynx or by a rise in antibody titer; however, these inapparent infections are still considered contagious.[2] Abortive poliomyelitis occurs in about 4 to 8% of infections and its manifestations include fever, headache, sore throat, listlessness, anorexia, vomiting, and abdominal pain. Neurologic examination is normal. The illness lasts from a few hours to about 2 to 3 days and is clinically indistinguishable from other viral infections; it can be suspected clinically during an epidemic. Nonparalytic poliomyelitis has more severe systemic manifestations than the abortive type, and with positive signs of meningeal irritation that make it clinically indistinguishable from aseptic meningitis caused by other enteroviruses.[4]

Paralytic poliomyelitis can be classified as spinal, bulbar, or spinobulbar disease. The development of paralysis is rapid (about 2 to 4 hours), is usually accompanied by fever and muscle pain, and rarely progresses after the patient's temperature has returned to normal. Spinal paralysis is usually asymmetric and more severe proximally than distally. Deep tendon reflexes are absent or diminished. Bulbar paralysis may affect respiration and swallowing. Many patients recover some muscle function after the acute episode. Prognosis can be firmly assessed usually within 6 months after the onset of paralytic manifestations.[1]

Paralytic poliomyelitis may be confused with Guillain-Barré syndrome; in the latter, (a) the muscle weakness is more symmetric and ascending, with loss of sensation in about 80% of cases; (b) paresthesia is common; and (c) CSF findings consist of high protein content with normal or minimal

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pleocytosis.[4] Other than Guillain-Barré syndrome, atypical/typical presentation of poliomyelitis may be mistaken for other clinical entities such as transverse myelitis, traumatic neuritis, or other paralytic conditions.[5,74]

Risk factors for paralytic poliomyelitis include larger inocula of poliovirus, increasing age, pregnancy, strenuous exercise, tonsillectomy, and administration of intramuscular injections while the patient is infected with the virus, either during outbreaks or when intramuscular injections are given within 30 days after administering oral poliovirus vaccine.[1,6,7,8,9] In developing countries, other factors to consider include a compromised environment (because of poor sanitation and high population density) that is a potential source of endogenous foci of poliovirus activity and the poor immune status of the community due to inadequate nutrition.[80,81]

Poliomyelitis confers type-specific lifelong immunity. Carrier states (asymptomatic persons excreting poliovirus for more than 6 months after infection) are rare and have been reported only in immunodeficient persons.[1]

A late-onset syndrome (post-polio syndrome) has been reported with increasing frequency among people, occurring 30 to 40 years after the patients contracted wild poliovirus infection in childhood. The cause is unknown but probably is related to the aging or death of nerves and muscles that were compensating for the original damage. Patients experience muscle pain and exacerbation of existing muscle weakness. Risk factors for developing the post-polio syndrome include (a) increasing length of time since acute poliovirus infection, (b) presence of permanent residual impairment after recovery from the acute illness, and (c) female sex.[1,2,10,74,77]

Severe poliomyelitis can result in lower limb deformity such as flexion contracture of the knee or lateral rotation deformity of the tibia, leading to impaired mobility.[82,83] Other complications of poliomyelitis may include impairment of respiration due to paralysis of the respiratory muscles, airway obstruction due to involvement of cranial nerve nuclei or lesions of the respiratory center. Myocarditis, gastrointestinal problems (hemorrhage, paralytic ileus, gastric dilatation), and urinary tract infections also have been reported.[4]

Management of poliomyelitis is supportive and symptomatic, since antiviral agents specific for the treatment of this illness are not available. Patients with abortive or mild nonparalytic poliomyelitis may require only bed rest for several days. Analgesics, antipyretics, or hot, moist packs applied to muscles may be helpful. During active myelitis, rest on a firm bed is advisable. Physical therapy is very important in the management of paralytic poliomyelitis during the convalescent period.[4] ■

References

1. Advisory Committee on Immunization Practices. Poliomyelitis prevention in the United States: introduction of a sequential vaccination schedule of inactivated poliovirus vaccine followed by oral poliovirus vaccine. *MMWR Morb Mortal Wkly Rep* 1997; 46(RR-3): 1-25.

2. Grabenstein JD. Poliovirus: which vaccine when? *Hosp Pharm* 1997; 32(6): 866-80.

3. Ren R, Racaniello VR. Poliovirus spreads from muscle to the central nervous system by neural pathways. *J Infect Dis* 1992; 166(4): 747-52.

4. Modlin JF. Poliovirus. In: Mandell GL, Bennett JE, Dolin R, editors. *Principles and practice of infectious diseases.* New York: Churchill Livingstone Inc; 1995. P. 1613-20.

5. Sabin AB. Paralytic poliomyelitis: old dogmas and new perspectives. *Rev Infect Dis* 1981; 3: 543-64.

6. Sutter RW, Patriarca PA, Suleiman AJ. Attributable risk of DTP (diphtheria and tetanus toxoids and pertussis vaccine) injection in provoking paralytic poliomyelitis during a large outbreak in Oman. *J Infect Dis* 1992; 165(3): 444-9.

7. Wyatt HV. Mothers, injections and poliomyelitis. *Soc Sci Med* 1992; 35(6): 795-8.

8. Strebel PM, Ion-Nedelcu N, Baughman AL, et al. Intramuscular injections within 30 days of immunization with oral poliovirus vaccine: a risk factor for vaccine-associated paralytic poliomyelitis. *N Engl J Med* 1995; 332: 500-6.

9. Wright PF, Karson DT. Minimizing the risks associated with the prevention of poliomyelitis. *N Engl J Med* 1995; 332: 529-30.

10. Ramlow J, Alexander M, LaPorte R, et al. Epidemiology of the post-polio syndrome. *Am J Epidemiol* 1992; 136(7): 769-86.

74. Panel comment, 10/98.

77. Reviewers' consensus, 10/98.

79. Knolle H. Transmission of poliomyelitis by drinking water and the problem of prevention. *Gesundheitswesen* 1995; 57(6): 351-4.

80. Swartz TA. Basic conditions for the eradication of poliomyelitis: indications for a common prescription. *Public Health Rev* 1993/94; 21: 157-60.

81. Panel comment, 11/98.

82. Asirvatham R, Watts HG, Rooney RJ. Rotation osteotomy of the tibia after poliomyelitis: a review of 51 patients. *J Bone Joint Surg* 1990; 72(3): 409-1 1.

83. Parekh PK. Flexion contractures of the knee following poliomyelitis. *Int Orthop* 1983; 7(3): 165-72.

86. van der Avoort HG, Reimerink JH, Ras A, et al. Isolation of epidemic poliovirus from sewage during the 1992-3 type 3 outbreak in The Netherlands. *Epidemiol Infect* 1995; 114(3): 481-91.

The complete monograph entitled "Information on Poliomyelitis and Poliovirus Vaccine Live Oral Systemic" was developed with the support of the United States Agency for International Development. The document is available in French, Portuguese, Russian, and Arabic, in addition to the original English. To receive a copy (please indicate which language), contact Nancy Blum, MPH, MA, US Pharmacopeia, 12601 Twinbrook Parkway, Rockville, MD (Maryland) 20852 (301-816-8385, nlb@USP.org).