Table 1. Summary of clinical manifestations of poliovirus infection[6,7,12-19]

Clinical manifestation	Frequency	Presenting symptoms/signs	Diagnosis	Prognosis
Asymptomatic infection	75–95% of infection	Asymptomatic or minor malaise.	Detected through isolation or polymerase chain reaction (PCR) of poliovirus from faeces or oropharynx, or through serology testing during poliovirus epidemic or outbreaks, as symptoms/presentation are not specific to poliovirus infection.	Complete recovery without complications
Abortive polio/ minor illness	4–24% of infection	Transient illness with nonspecific symptoms including fever, malaise, drowsiness, nausea, anorexia, vomiting, constipation, headache and sore throat; normal neurological examination.	Detected through isolation or PCR of poliovirus from faeces or oropharynx. CSF biochemical markers are similar to those found in viral meningitis. CSF can have increased leucocytes with a high ratio of polymorphonuclear cells to lymphocytes during early disease. CSF total protein can also be elevated at an average of 40–50mg/dL. CSF glucose level is usually normal. Unlike other viral causes of aseptic meningitis, poliovirus is rarely isolated from CSF.	High mortality for bulbar polio or spinal-bulbar polio involving the medulla, leading to cardio-respiratory compromise and death; high mortality for polioencephalitis. Permanent weakness in two-thirds of patients. Complete recovery is less likely if presenting symptoms of paralysis are severe.
Nonparalytic polio/ aseptic meningitis	1–4% of infection	Symptoms are similar to abortive polio but with the addition of meningeal irritation, neck stiffness, severe headache and pain over the limbs, back and neck that develop 1–2 days later. The illness lasts for up to 2 weeks.		
Paralytic polio	<1% of infection	<u>Spinal paralytic polio</u> Initial period of minor illness followed by a brief symptom-free period of up to 10 days and subsequent rapid onset of acute flaccid paralysis (AFP), loss of deep tendon reflexes, paraesthesia, fever, headache, neck stiffness and cerebrospinal fluid (CSF) pleocytosis. Lower motor neuron signs appear with classical asymmetrical distribution of flaccid paralysis. Lower limbs are more commonly affected than upper limbs. Sensory involvement is very rare. <u>Bulbar paralytic polio</u> Paralysis of cranial nerves especially the ninth and tenth cranial nerves innervating the soft palate and pharynx, without limb weakness. Patients can present with dysphagia, dyspnoea and pooling of saliva. <u>Polioencephalitis</u> Agitation, confusion, disturbances of consciousness, seizures, autonomic dysfunction and upper motor neuron signs.		
Post-polio syndrome (PPS)	Around 50% of survivors of paralytic polio	A non-infectious syndrome can occur in survivors of paralytic polio from 8–71 years later. New gradual onset of muscle weakness, pain and fatigue can occur in the same muscles that were affected during the course of paralytic polio, or previously unaffected limb muscle groups can also be involved in PPS.	No specific test for diagnosis. Most experts have validated the diagnostic criteria by Halstead et al.[19] PPS is a diagnosis of exclusion and all potential medical or surgical causes must be excluded before diagnosis of PPS is made.	Varied prognosis. In some cases, affected persons are not severely handicapped and symptoms stabilised over time. In severe cases, PPS can lead to skeletal deformities, affecting a person's ability to perform simple tasks of daily living.

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