Pathology and Pathogenesis

Poliovirus is extremely infectious, but is usually benign, with 90 to 95 percent of cases being asymptomatic. In four to eight percent of cases there is a non-specific viral syndrome and only one to two percent of cases are associated with paralysis. The rate of paralysis varies with the strain of the virus and the individual's age, with the likelihood of paralysis increasing with age. In children, paralysis occurs in 1/1000 cases, while in adults, paralysis occurs in 1/75.[5] Polio is predominantly a disease of the very young, with 70 to 90 percent of cases occurring in those younger than 3 years.[4]

The poliovirus is a positive single stranded RNA enterovirus (picornavirus) and has three antigenically distinguishable types: poliovirus numbers I (responsible for approximately 85 percent of paralytic cases), II and III. None provide cross-immunity to the others.[1] Polio type infections can also be caused by other enteroviruses, a group that consists of the polioviruses, Coxsackie A (A7, A9) and B (B2 to B4) viruses (types 1, 2, 4, 6, 7, 9, 11, 16, 18, 30) ECHO viruses and enteroviruses 70 and 71.[6]

The poliovirus accesses the body orofaecally. After passing through the stomach, the virus reaches the intestine where it establishes itself in the cells of the intestinal lining. Viruses multiply in the lymphoid tissues of the oropharynx and intestine during the one- to three-week incubation period.[7] In most cases, the virus is present in the oropharyngeal secretions for one to two weeks and is excreted in the stools for several weeks to months. From the lymphoid tissues of the oropharynx and intestine, the virus may move into the regional lymph nodes and from there into the blood, causing a transient viremia. If the virus accesses the circulatory system and a viremia results, the central nervous system is exposed to the infection and acute paralytic polio may result.[1]

Poliovirus has a predilection for the motor neurons of the anterior horn of the cervical and lumbar regions of the spinal cord, which can result in the cell death or injury of these motor neurons. Following death of the anterior horn cells, wallerian degeneration results and the muscle fibres associated with those neurons become "orphaned" (denervated), producing clinical weakness.[8] The localisation and degree of paralysis depends on the site and the severity of neuronal lesions.[7]

Studies undertaken more recently have shown that the involvement of the central nervous system is more diffuse than simply the motor neurons.[9-11] Lesions are also observed in the intermediate and posterior grey columns, and occasionally in the dorsal root ganglia. In the brainstem, the reticular formation and most of the nuclei of cranial nerves can be involved. In the cerebral cortex, neuronal lesions are usually mild and restricted to the precentral gyrus, thalamus, hypothalamus and the globus pallidus.[1]