**Poliomyelitis and Acute Flaccid Paralysis**

* Poliovirus infection is **subclinical in 90–95%** of cases.
* it causes **nonspecific** febrile illness in about 5% of cases and **aseptic meningitis** or **paralytic disease** in 1–3%.
* In endemic areas, older children and adults are immune because of prior unapparent infections.
* Occasional cases in the developed countries occur in patients who have traveled to foreign countries; most cases were in immunodeficient patients who received the poliovirus vaccine or were exposed to recent vaccinees.
* Severe poliovirus infections rarely follow oral poliovirus vaccination as a result of "reversion" of the vaccine virus. The incidence of **vaccine associated paralytic poliomyelitis (VAPP)** in the United States has been 1:750,000 and 1:2.4 million doses for the first and second dose of oral polio vaccine (OPV), respectively. Although rare, VAPP has been more common than wild-type poliomyelitis in the United States since the 1980s. This led to changes in the recommended immunization regimen, substituting inactivated polio vaccine (IPV) for OPV.

**Clinical Findings:**

The initial symptoms are fever, myalgia, sore throat, and headache for 2–6 days. In less than 10% of infected children, several symptom-free days are followed by recurrent fever and signs of **aseptic meningitis**: headache, stiff neck, spinal rigidity, and nausea. Mild cases resolve completely.

In only 1–2% of children does high fever, severe myalgia, and anxiety portend progression to loss of reflexes and subsequent flaccid paralysis. Sensation remains intact, although **hyperesthesia** of skin overlying paralyzed muscles is common and "pathognomonic".

Paralysis is usually "asymmetric". Proximal limb muscles are more often involved than distal, and lower limb involvement is more common. **Bulbar involvement** affects swallowing, speech, and cardiorespiratory function and accounts for most deaths. Bladder distention and marked constipation characteristically accompany lower limb paralysis. Paralysis is usually complete by the time the temperature normalizes. Weakness often resolves completely. Atrophy is usually apparent by 4–8 weeks. Most improvement of muscle paralysis will take place within 6 months.

**Laboratory findings:**

In patients with **meningeal symptoms**, the CSF contains:

 1) up to several hundred leukocytes (mostly lymphocytes) per microliter,

2) the glucose level is normal, and

3) protein concentration is mildly elevated.

Poliovirus is easy to grow in cell culture and can be readily differentiated from other enteroviruses. It is rarely isolated from spinal fluid but is often present in the throat and stool for several weeks following infection. Paired serology is also diagnostic. Laboratory methods are available to differentiate wild from attenuated vaccine isolates.

**Differential diagnosis**

**Aseptic meningitis** due to poliovirus is indistinguishable from that due to other viruses.

**Paralytic disease:**

**1. Neurotropic virus infections:**

1. Rabies virus: onset of paralysis: Mo- yr; progression of paralysis: acute, symmetric, ascending; sensory signs and symptoms with reduction or absence of deep tendon reflexes.

2. Varicella-zoster virus: Exanthematous vesicular eruptions with incubation period 10- 21 days; Acute, symmetric, ascending with sensory signs and symptoms.

**2. Guillain–Barré syndrome (GBS):** is an acute polyneuropathy, a disorder affecting the peripheral nervous system. Ascending symmetric paralysis (weakness beginning in the feet and hands and migrating towards the trunk) is the most typical symptom, and some subtypes cause change in sensation or pain, as well as dysfunction of the autonomic nervous system. It can cause life-threatening complications, in particular if the respiratory muscles are affected or if the autonomic nervous system is involved. The disease is usually triggered by an infection (e.g. respiratory tract infection).

The diagnosis is usually made by nerve conduction studies and with studies of the cerebrospinal fluid (minimal pleocytosis, "high protein" concentration in the CSF –*albuminocell dissociation*).

**3. Acute traumatic sciatic neuritis:** Intramuscular gluteal injection; acute, asymmetric weakness; hours to 4 days after the trauma; sensory signs and symptoms with reduction or absence of deep tendon reflexes.

**4. Acute transverse myelitis:** Preceding Mycoplasma pneumonia, Schistosoma, other parasitic or viral infection. Acute, symmetric hypotonia of lower limbs; progression of the paralysis: hours to days; sensory signs and symptoms with reduction or absence of deep tendon reflexes. CSF is usually normal.

**5. Spinal cord compression**: by space-occupying lesion or trauma. Back pain, local spinal tenderness. Acute, symmetric hypotonia of lower limbs; sensory signs and symptoms with reduction or absence of deep tendon reflexes.

**6. Toxin –induced neuropathy:**

1. Diphtheria (*Corynebacterium diphtheriae*): sensory signs and symptoms with reduction or absence of deep tendon reflexes.

2. Botulism (*Clostridium botulinum*): Abdominal pain, diplopia, loss of accommodation, mydriasis. Incubation period 18-36 hr. Rapid, "descending", symmetric paralysis. **No** sensory signs and symptoms or reduction or absence of deep tendon reflexes.

3. Tick bite paralysis: Ocular symptoms; Latency period 5-10 days; Acute, symmetric, ascending paralysis with reduction or absence of deep tendon reflexes, but no sensory signs and symptoms.

**7. Diseases of the neuromuscular junction:**

Myasthenia gravis: Weakness, fatigability, diplopia, ptosis, dysarthria. Multifocal weakness. No sensory signs and symptoms or reduction or absence of deep tendon reflexes.

**8. Disorders of muscle:** e.g. Polymyositis: there is neoplasm or autoimmune disease. The onset: subacute. Proximal muscle involvement → distal ones. Progression of the disease: Weeks to months with reduction or absence of deep tendon reflexes, but no sensory signs and symptoms.

**9. Hypokalemic periodic paralysis:** Proximal limb and respiratory muscles involved. The onset is sudden & postprandial with reduction or absence of deep tendon reflexes, but no sensory signs and symptoms. Page

**Critical illness polyneuropathy:** Flaccid limbs and respiratory weakness. The onset: acute, following systemic inflammatory response syndrome/sepsis Progression: hours to days with reduction or absence of deep tendon reflexes, but no sensory signs and symptoms.

*"The possibility of polio should be considered in any case of acute flaccid paralysis even in countries where polio has been eradicated".*

**Complications & Sequelae:**

Complications are the result of the acute and permanent effects of paralysis. Respiratory, pharyngeal, bladder, and bowel malfunction are most critical. Deaths are usually due to complications arising from respiratory dysfunction. Limbs injured near the time of infection, such as by intramuscular injections, excessive prior use, or trauma, tend to be most severely involved and have the worst prognosis for recovery (**provocation paralysis**). Postpolio muscular atrophy occurs in 30–40% of paralyzed limbs 20–30 years later, characterized by increasing weakness and fasciculations in previously affected, partially recovered limbs.

Treatment & Prognosis:

Therapy is **supportive**. Bed rest, fever and pain control (heat therapy is helpful), and careful attention to progression of weakness (particularly of respiratory muscles) are important. No intramuscular injections should be given during the acute phase. Intubation or tracheostomy for secretion control and catheter drainage of the bladder may be needed. Assisted ventilation and enteral feeding may also be needed. Specific therapy is not available for poliomyelitis. **Pleconaril**, which is active against most enteroviruses, has "no effect" on polioviruses. Postpolio paralysis is mild in about 30%, permanent in 15%, and results in death in 5–10%. Disease is worse in adults and pregnant women than in children.

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