**Explain the lab Diagnosis of poliomyelitis?**

Paralytic poliomyelitis may be clinically suspected in individuals experiencing acute onset of flaccid paralysis in one or more limbs with decreased or absent tendon reflexes in the affected limbs, that cannot be attributed to another apparent cause, and without sensory or cognitive loss. [38]

A laboratory diagnosis is usually made based on recovery of poliovirus from a stool sample or a swab of the pharynx. Antibodies to poliovirus can be diagnostic, and are generally detected in the blood of infected patients early in the course of infection. [4] Analysis of the patient's cerebrospinal fluid (CSF), which is collected by a lumbar puncture ("spinal tap"), reveals an increased number of white blood cells (primarily lymphocytes) and a mildly elevated protein level. Detection of virus in the CSF is diagnostic of paralytic polio, but rarely occurs. [4]

If poliovirus is isolated from a patient experiencing acute flaccid paralysis, it is further tested through oligonucleotide mapping (genetic fingerprinting), or more recently by PCR amplification, to determine whether it is "wild type" (that is, the virus encountered in nature) or "vaccine type" (derived from a strain of poliovirus used to produce polio vaccine). [39] It is important to determine the source of the virus because for each reported case of paralytic polio caused by wild poliovirus, it is estimated that another 200 to 3,000 contagious asymptomatic carriers exist. [40]

**Prognosis**

Patients with abortive polio infections recover completely. In those that develop only aseptic meningitis, the symptoms can be expected to persist for two to ten days, followed by complete recovery. [41] In cases of spinal polio, if the affected nerve cells are completely destroyed, paralysis will be permanent; cells that are not destroyed but lose function temporarily may recover within four to six weeks after onset. [41] Half the patients with spinal polio recover fully, one quarter recover with mild disability and the remaining quarter are left with severe disability. [42] The degree of both acute paralysis and residual paralysis is likely to be proportional to the degree of viremia, and inversely proportional to the degree of immunity. [30] Spinal polio is rarely fatal. [31]

1. Viral isolation

Poliovirus may be recovered from the stool or pharynx of a person with presumed poliomyelitis. Isolation of virus from the cerebrospinal fluid (CSF) is diagnostic, but is rarely accomplished.

If poliovirus is isolated from a person with acute flaccid paralysis, it must be tested further, using oligonucleotide mapping (fingerprinting) or genomic sequencing, to determine if the virus is “wild-like” or “vaccine-like.”

2. Serology

Neutralizing antibodies appear early and may be at high levels by the time the patient is hospitalized and, therefore, a 4-fold rise may not be demonstrated.
3. Cerebrospinal fluid (CSF)

The CSF in poliovirus infection usually contains an increased number of white blood cells (10 to 200 cells/mm³, primarily lymphocytes) and a mildly elevated protein from 40 to 50 mg/100 ml.

Without respiratory support, consequences of poliomyelitis with respiratory involvement include suffocation or pneumonia from aspiration of secretions.[43] Overall, 5–10% of patients with paralytic polio die due to the paralysis of muscles used for breathing. The mortality rate varies by age: 2–5% of children and up to 15–30% of adults die.[4] Bulbar polio often causes death if respiratory support is not provided;[37] with support, its mortality rate ranges from 25 to 75%, depending on the age of the patient.[4][44] When positive pressure ventilators are available, the mortality can be reduced to 15%.