POLIOMYELITIS
HISTORY

• First outbreak in Europe in early 1800’s
• First outbreak in U.S. reported in 1843
• Peaked in the U.S. in 1952
• Became known as the Heine-Medin disease due to the work of Dr. Jakob Heine and Dr. Karloskar Medin.
## EPIDEMIOLOGY

### Most recent Wild Polio Virus case by State

<table>
<thead>
<tr>
<th>State</th>
<th>Date of most recent polio case</th>
<th>Type</th>
</tr>
</thead>
<tbody>
<tr>
<td>West Bengal</td>
<td>13-Jan-11</td>
<td>P1</td>
</tr>
<tr>
<td>Jharkhand</td>
<td>22-Oct-10</td>
<td>P3</td>
</tr>
<tr>
<td>Maharashtra</td>
<td>16-Sep-10</td>
<td>P1</td>
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<tr>
<td>Bihar</td>
<td>01-Sep-10</td>
<td>P1</td>
</tr>
<tr>
<td>Uttar Pradesh</td>
<td>21-Apr-10</td>
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<tr>
<td>Jammu &amp; Kashmir</td>
<td>07-Feb-10</td>
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<tr>
<td>Haryana</td>
<td>13-Jan-10</td>
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<tr>
<td>Rajasthan</td>
<td>27-Nov-09</td>
<td>P3</td>
</tr>
<tr>
<td>Uttarakhand</td>
<td>06-Nov-09</td>
<td>P3</td>
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<tr>
<td>Himachal Pradesh</td>
<td>17-Oct-09</td>
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<td>13-Oct-09</td>
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<tr>
<td>Delhi</td>
<td>28-Jun-09</td>
<td>P3</td>
</tr>
<tr>
<td>Madhya Pradesh</td>
<td>04-Aug-08</td>
<td>P3</td>
</tr>
<tr>
<td>Odisha</td>
<td>22-Jul-08</td>
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<td>Andhra Pradesh</td>
<td>16-Jul-08</td>
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<td>Assam</td>
<td>09-Jun-08</td>
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<td>Karnataka</td>
<td>03-Nov-07</td>
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<td>Gujarat</td>
<td>16-Mar-07</td>
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<td>Daman &amp; Diu</td>
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<td>P1 &amp; P3</td>
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<td>Meghalaya</td>
<td>15-Oct-97</td>
<td>P1</td>
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<td>Tripura</td>
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<td>A&amp;N Islands</td>
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<td>Sikkim</td>
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</table>

### Wild Polio cases, India

**2010**

- **P1**
- **P3**

**2011**

- **P1**

**Legend:**
- P1: Type 1
- P3: Type 3

<table>
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<td>268</td>
<td>1600</td>
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</tbody>
</table>

*Note: The table represents the number of wild polio cases by month and year for the years 1998 to 2015.*
POLIO CASES IN INDIA

[Chart showing the number of polio cases in India from 1998 to 2011*]
India was declared polio free on 27th March 2014.
ETIOLOGY- POLIO VIRUS

• Genus: Enterovirus.
• Family: Picornaviridae.
• Single strand of RNA Virus.
• Three well defined serotypes: 1, 2 and 3
  - All types cause paralysis
  - Type 1 most frequently causes epidemic.
  - Type 2 most common cause for vaccine derived poliomyelitis
POLIO VIRUS
TRANSMISSION

• Feco-oral route predominates

• Highly communicable
  - One infected individual will infect all non-immune persons in a household.
INCUBATION PERIOD & EXCRETION OF VIRUS

• Short incubation period: usually 7-14 days (range 4-35 days)
• Virus intermittently excreted for 6-8 weeks after infection
• Majority of viral excretion
  - just prior to paralysis onset
  - up to first two weeks
  - dramatically tapers off after 4 weeks
CLINICAL FEATURES

- Asymptomatic Polio
- Non-paralytic
- Paralytic
  - Spinal
  - Bulbar
  - Bulbo-spinal
Paralysis is an infrequent manifestation of infection

- Paralytic poliomyelitis
- Asymptomatic infection
  - Clinical illness, no paralysis
    - Spinal
    - Bulbospinal
    - Bulbar
    - Encephalitic
ASYMPTOMATIC POLIO

• Accounts for approximately 95% of cases

• Virus stays in intestinal tract and does not attack the nerves

• Virus is shed in the stool so infected individual is still able to infect others
NON PARALYTIC POLIOMYELITIS

• Abortive poliomyelitis
• Does not lead to paralysis
• Mild symptoms seen, such as sore throat, fever, diarrhea, constipation
• Most recover in <1 week
• Non-paralytic aseptic meningitis
  – Occurs in 1-2% of polio infections
  – Symptoms are stiffness in the neck, back, and/or legs
  – Increased or abnormal sensations can occur
  – Complete recovery after 2-10 days of symptoms
PARALYTIC POLIOMYELITIS

• Most serious form
• About 0.1% of those infected develop this type
• Acute flaccid paralysis seen
  
  Paralysis is always LMN type
• Headache, neck/back stiffness, unusual sensations, increased sensitivity to touch
PARALYTIC POLIOMYELITIS

• 3 types-
  - Spinal form
  - Bulbar and bulbo-spinal form
  - Polio encephalitis

• 2 phases
  - **Minor phase**: similar to abortive illness
  - **Major phase**: occurs after several days - Muscle pain, fever returns, flaccid paralysis in 72 hr., diminished tendon reflexes, Asymmetrical, transitory urinary retention
PARALYTIC POLIOMYELITIS

Distinguishing features

• Asymmetric flaccid paralysis
  - proximal > distal
  - deep tendon reflexes diminished or absent

• fever at onset, muscle pain

• rapid progression to paralysis 2-3 days

• preservation of sensory nerve function

• residual paralysis after 60 days
SPINAL POLIO

• Most common form of paralytic poliomyelitis; (79% of all paralytic cases)
• Attacks motor neurons and causes paralysis of muscles innervated by spinal nerve
• Most commonly affected muscle groups are
  - Quadriceps, tibialis anterior, peroneal
  - Deltoid, biceps, triceps
  - Abdominal muscles, intercostal and diaphragm
• Respiratory muscle paralysis can cause life threatening impairment of ventilation.
• Those affected still retain sensation in extremities
BULBAR POLIO

• Accounts for <1% of paralytic polio
• Virus attacks motor neurons in brainstem particularly the medulla, hence the poliomyelitis with worst prognosis
• Commonly involved cranial nerves are:
  - III to VII nerve nuclei – good prognosis
  - IX to XII nerve nuclei – poor prognosis
• Impairment of respiratory center – may lead to respiratory failure
• Impairment of circulatory center (autonomic nervous system) – leading to hypertension and peripheral circulatory failure
BULBOSPINAL POLIO

• Accounts for 19% of paralytic cases
• Has mixed features; i.e. features of both spinal as well as bulbar forms
• Affects extremities and cranial nerves
• Leads to severe respiratory involvement
ENCEPHALITIC POLIO

• Very rare
• Causes inflammation of gray matter of brain
• Signs/symptoms include agitation, confusion, stupor, and coma
• Autonomic dysfunction is common and it has a high mortality
• Diagnosis is mainly based on clinical manifestations
• High index of suspicion is to be maintained in an unimmunized child with paralytic disease
• Clinical – A combination of fever, headache, neck and back pain, asymmetric flaccid paralysis without sensory loss and pleocytosis in CSF
• Laboratory diagnosis by isolation of poliovirus in stool and identification of strain
• CSF to rule out meningitis.
IDENTIFICATION OF POLIOVIRUS IN STOOL

- 2 stool specimens 24-48 hrs. apart
- High poliovirus concentration in first week of illness
- Minimum of 8 to 10g of stool sample
- Yield is 85 – 90% in acute phase of illness
# Differential diagnosis of Paralytic Poliomyelitis

<table>
<thead>
<tr>
<th>Signs and Symptoms</th>
<th>Poliomyelitis</th>
<th>GBS</th>
<th>Transverse Myelitis</th>
<th>Traumatic Neuritis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Progression of paralysis</td>
<td>&lt;4 days, max. 7 days</td>
<td>From hours to days</td>
<td>From hours to 4 days</td>
<td>From hours to 4 days</td>
</tr>
<tr>
<td>Flaccidity</td>
<td>Asymmetrical, proximal</td>
<td>Symmetrical, distal</td>
<td>Symmetrical, lower limbs</td>
<td>Symmetrical, one limb</td>
</tr>
<tr>
<td>Sensations</td>
<td>Preserved</td>
<td>Hyperesthesia</td>
<td>Anesthesia of lower limbs</td>
<td>Pain in gluteal region</td>
</tr>
<tr>
<td>Cranial nerve involvement</td>
<td>Only in bulbar &amp; bulbospinal</td>
<td>Often present</td>
<td>absent</td>
<td>Absent</td>
</tr>
<tr>
<td>Respiratory involvement</td>
<td>Only in bulbar &amp; bulbospinal</td>
<td>In severe cases</td>
<td>Absent</td>
<td>Absent</td>
</tr>
<tr>
<td>CSF- cells protein</td>
<td>High WBCs, N, or mildly raised</td>
<td>&lt; 10 WBCs</td>
<td>Normal Normal or slightly raised</td>
<td>Normal normal</td>
</tr>
<tr>
<td>Signs and Symptoms</td>
<td>Poliomyelitis</td>
<td>GBS</td>
<td>Transverse Myelitis</td>
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<tr>
<td>Bladder dysfunction</td>
<td>Transient retention</td>
<td>Sometimes</td>
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<td>EMG _ 3wks</td>
<td>Abnormal</td>
<td>Normal</td>
<td>Normal</td>
<td>Normal</td>
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<tr>
<td>sequelae</td>
<td>Severe, Asymmetrical atrophy</td>
<td>Absent or minimal</td>
<td>Moderate atrophy</td>
<td>Peroneal atrophy</td>
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</tbody>
</table>
INDICATIONS FOR HOSPITALIZATION

• Progression of paralysis
• Respiratory distress
• Bulbar involvement
• Paralysis of upper limbs of <3 days
• Marked drowsiness
TREATMENT

• Bed rest - Physical activity increases risk of paralysis.
• Pain relief – Analgesics, sister Kennedy's method, mild sedative in spinal form, not to be used in bulbar form, encephalitis.
• Neutral position of limbs.
• Physiotherapy prevents deformity.
TREATMENT CONT.

• Good nursing, feeding in prone position, suctioning of pharynx
• Respiratory insufficiency- mechanical ventilation
• Rehabilitation after acute phase - physical, emotional and psychological
• Intramuscular injections and massage avoided in acute stage
POLIO ERADICATION STRATEGIES

- Routine Immunization
- Immunization Days (NIDs)
- AFP Surveillance
- Mop-ups
POLIO VACCINES

Two types of polio vaccine

- Inactivated killed injectable polio vaccine (IPV) developed by Dr Jonas Salk

- Live attenuated Oral polio vaccine developed by Dr Albert Sabin
OPV

• Live attenuated
• Sabin strain
• Storage- freezer/ 2 – 8 degree C
• Dose – 2 drops oral
• Schedule – Birth, 6, 10, 14 weeks, 15-18 months, 5 years, NIDs, SNIDs
• Efficacy – 10-15% per dose
• Adverse effect – VAPP
• Contraindication – Immunodeficient persons
IPV

• Inactivated vaccine
• Salk strain
• Storage – 2 to 8 degree Celsius
• Schedule – 6, 10, 14 weeks, booster at 15 to 18 months
• Efficacy – 95 – 100 %
IAP SCHEDULE FOR IMMUNIZATION

• Birth - OPV-0
• 6 Wks. - OPV-1/ OPV-1 + IPV-1
• 10 Wks. - OPV-2/ OPV-2 + IPV-2
• 14 Wks. - OPV-3/ OPV-3 + IPV-3
• 15 - 18 Months - OPV-4 + IPV-B1
• 5 Yrs. - OPV-5
VACCINE ASSOCIATED PARALYTIC POLIOMYELITIS

• Rare but serious adverse effect of OPV
• Due to loss of attenuating mutations
• Stool samples show vaccine related poliovirus and no wild virus
• Occurs within 4 - 40 days of receiving OPV
• Can occur in vaccine recipient (Recipient VAPP) or contact of vaccine recipient (contact VAPP)
ACUTE FLACCID PARALYSIS SURVEILLANCE (AFP)
ACUTE FLACCID PARALYSIS (AFP)  
CASE DEFINITION

• Sudden onset of weakness and floppiness in any part of the body in a child < 15 years of age or paralysis in a person of any age in which polio is suspected.
COMPONENTS OF AFP SURVEILLANCE

- Immediate reporting
- Investigation of all AFP cases
- Classification of all AFP cases
- Weekly NIL reporting by reporting units
The process of AFP surveillance:

1. Onset of paralysis
2. Detection & notification
3. Case investigation & specimen collection
4. Specimens arrive at national lab
5. Virus isolation results reported
6. Follow-up exam
7. Classification of case
8. Isolates sent to regional lab for intra-typic differentiation
ACHIEVEMENTS OF ERADICATION PROGRAMME

• Last wild polio virus type 1 case: 13th Jan 2011, Howrah, WB.
• Last wild polio virus type 2 case: October 1999, Aligarh, UP.
• Last wild polio virus type 3 case: 22 October 2010, Pakur, Jharkhand.
• Last positive case from monthly environmental sewage sampling: November 2010, Mumbai.
POST ERADICATION ERA ISSUES

• curbing vaccine derived poliomyelitis and vaccine associated paralytic poliomyelitis.

• Shifting from oral polio vaccine to injectable polio vaccine.
WHY NOT CONTINUE ORAL POLIO VACCINE IN POST ERADICATION ERA?

• Continued reintroduction of the attenuated polioviruses of OPV into a polio-free world will result in polio cases due to vaccine-associated paralytic polio (VAPP), and polio outbreaks due to circulating vaccine-derived polioviruses (cVDPVs).
• The cVDPVs could re-seed the world with poliovirus, and thus negate the achievement of eradication.
• Thus, OPV cessation is the cornerstone to secure polio eradication.
THEN WHY DIDN’T WE USE IPV INSTEAD OF OPV?

- IPV confers very little immunity in the intestinal tract.
- When a person immunized with IPV is infected with wild poliovirus or a cVDPV, virus can still multiply inside the intestines and be shed in the stool, risking continued circulation.
- OPV is the vaccine of choice to stop polio outbreaks.
Thank you