Clinic of poliomyelitis: Challenges to diagnosis

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Conflict of interest

• I declare no conflict of interest

• Chair, National Verification Commission for the Elimination of Measles, Rubella, Congenital Rubella Syndrome 2013-Date

• Member, National Verification Commission for the Elimination of Polio 2013-Date
Clinical case

- Age: 23 months
- Male
- From: Anapoima, Cundinamarca
- Companion: Mother, 21 years old
- Date of entry FCI: 03/28/23

Perinatal: negative
Pathologicals: scabies, 1st year
Immunizations: EPI up to date
Psychomotor development: normal
Epidemiological: home with all services, no pets, no symptomatic contacts.

03/22-23/23 03/25/23 03/26/23 03/26-28/23 03/28/23

Fever 40°C
Dry cough
Vomiting
GE viral
SRO
Fever
Drowsiness
Weakness
Laboratories
Chest X-ray
Pneumonia vs.
Neuroinfection

Drowsy
No focus
Dehydrated
Clinical case

- Hemogram: Leu 9.170, N 5.530, L 2.820, Hb 12.4Gr%, Hcto 35.9%, Platelets 389.000
- Reactants: VSG 2 mm/h, PCR 0.42 mg/dL
- Electrolytes: Cl 97, P 5.6, Mg 2.2, K 4.7, Na 135
- Hepatic: ALT 18, AST 41, BT 0.3, BI 0.2, BI 0.1
- Uroanalysis: normal

Drowsy, normo-reactive isocoria, follows objects with gaze, follows simple commands, awakens with verbal stimulus, facial symmetry, central uvula, gag reflex present, generalized hypotonia, Brudzinski positive, Kernig negative.

Chest X-ray: bilateral parahilar interstitial infiltrates.

Simple cranial CT: normal

Lumbar puncture: normal

Acute flaccid paralysis

Comprehensive approach + management UCIP
Spinal cord

Anterior horn

Peripheral nerve

NM Union

Muscular

Encephalon

Transverse myelitis, compressive myelopathy, anterior spinal artery syndrome

ADEM, ANE, GBS with involvement of cranial nerves, stem encephalitis

Poliomyelitis, enteroviruses A71 and D68, Coxsackie, Echo virus, West Nile virus.

Guillain Barré Syndrome

GBS, toxic neuropathies, traumatic neuritis, acute intermittent porphyria.

Myasthenia gravis, botulism, snakebite, organophosphates

Polymyositis, trichinosis, hypokalemia, hypophosphatemia.

Differential Diagnosis
## Differential diagnostic evaluations

<table>
<thead>
<tr>
<th>Topography</th>
<th>Clinic</th>
<th>LCR</th>
<th>EMG, NC, EEG</th>
<th>RMN</th>
<th>Etiology</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Encephalon</strong></td>
<td>AEC, multifocal deficits, meningismus, pyramidal signs; ROT+++</td>
<td>Normal or pleocytosis</td>
<td>V-EEG: diffuse or focal slowing</td>
<td>Diffuse T2 hyperintensities, white s., poorly demarcated</td>
<td>ADEM, ANE</td>
</tr>
<tr>
<td><strong>Spinal cord</strong></td>
<td>Paraparesis, sphincteric involvement, sensory level ROT+++</td>
<td>Normal or pleocytosis, elevated IgG</td>
<td>Normal MCV</td>
<td>Central hyperintense T2 spinal cord lesion of more than 2 sec. thoracic</td>
<td>Transverse myelitis, Epidural abscess</td>
</tr>
<tr>
<td><strong>Anterior horn</strong></td>
<td>Fever, meningismus, asymmetrical weakness ROT-</td>
<td>Pleocytosis</td>
<td>Denervation Normal MCV</td>
<td>Anterior horn hyperintense on T2; owl's eye</td>
<td>Poliomyelitis, other non-poliovirus enteroviruses, etc.</td>
</tr>
<tr>
<td><strong>Root, peripheral nerves</strong></td>
<td>Ascending distal paralysis ROT -</td>
<td>AC dissociation, bands</td>
<td>&lt; VCM</td>
<td>Thickening and enhancement of anterior nerve roots, cauda and conus medullaris</td>
<td>GBS, enterovirus, WNV, toxics</td>
</tr>
<tr>
<td><strong>Neuromuscular junction</strong></td>
<td>Cranial paresis, bulbar Oculomotor ROT ++</td>
<td>-</td>
<td>Repetitive stimulus test</td>
<td>-</td>
<td>Botulism, Myasthenia, O/phosphates</td>
</tr>
<tr>
<td><strong>Muscle</strong></td>
<td>Proximal paresis, nuchal ROT +.</td>
<td>&gt; CPK</td>
<td>Normal MCV</td>
<td>High signal T2WI and STIR (edema), enhancement</td>
<td>Polymyositis, Dermatomyositis</td>
</tr>
</tbody>
</table>
Diagnostic evaluation in acute flaccid paralysis

Immediate reporting
- Stool sampling + others according to protocol**
- Start immediate investigation**

Evaluation by neurology & others
- Recording of clinical findings + additional studies

Diagnostic imaging
- Contrast MRI of the neuroaxis

Electro-diagnostic studies
- EMG neuroconduction
  - Repetitive stimuli

Diagnostic approach

Others
- *LCR
- *Blood
- *Other

** Recovery with corticosteroids

MENINGOENCEPHALITIS POST INFECTIONOUS
Objectives

Highlight and report on the types of presentation of poliomyelitis.

Illustrate the clinical features of acute forms of poliomyelitis & post poliomyelitis syndrome.

Emphasize the importance of the diagnostic approach & immediate reporting of flaccid paralysis.
> 3000 years ago
*Outbreaks of paralysis are documented in Egyptian papyri.

1887
*Polio epidemic shakes Stockholm, Sweden.
*Other epidemics arise in Europe and North America

1905: Ivar Wickham, MD discovers poliomyelitis

1908: Karl Landsteiner & Erwin Popper succeed in identifying & isolating the virus

1910-1960
*1910 vaccine idea initiated
*1951-1955 Salk develops vaccine, approved use of IPV
*1960s Approval of OPV vaccine

1968-2000's
*1968 Salk vaccine eliminated in US, OPV only
*1994 Polio eliminated from the Americas
*1997 New vaccine [IPV] introduced in the U.S.
*2014 begins transition to IPV
Poliomyelitis

A viral disease caused by one of the polioviruses, which is highly feared for its short- and long-term neurological effects.

Dramatic ↓ ↓ since the introduction of vaccination in the mid-1950s with elimination in the Americas in 1994.

Millions of people affected between 1940-1950 worldwide

Vaccine development brought hope!

Despite progress and elimination in many areas of the world:

- Millions of people still with sequelae [post polio syndrome].
- Some areas with wild poliovirus transmission.
- Cases of oral vaccine virus-associated poliovirus by ↓ of vaccination coverages.
**Poliovirus**

*RNA virus, enterovirus C, highly contagious.

* Virus with affinity for central nervous system.

*There are 3 types of wild-type viruses: 1 [> pre-vaccination cause], 2, 3

*Viruses related to oral vaccine have been identified.
Poliovirus

- The only reservoir is the human being
- It is transmitted by fecal-oral route due to poor hygienic conditions through pharyngeal secretions.
- It can also be transmitted by droplets [pharyngeal or respiratory secretions].
- Can be shed in saliva up to +/- 1 week
- Can be transmitted from people with or without symptoms
- Can remain in wastewater for long periods

Unvaccinated persons are at high risk for paralytic poliomyelitis if exposed to wild-type or oral vaccine-derived viruses
Duration of fecal excretion of wild poliovirus from infected persons.

- Pueden persistir en las heces por tiempo prolongado en inmunocomprometidos.

La excreción viral cae después de 14 días pero los virus del polio pueden ser detectados hasta 60 días desde el inicio.
Poliovirus transmission

- **Seasonality**
  - Seasonal climates: > summer transmission
  - Tropical zones: any time being > during the rainy season
- **High infectiousness**
  - 90-100% of non-immune household contacts will be infected
  - Sub-optimal sanitary conditions and overcrowding $\uparrow$ transmission.
Forms according to poliovirus acquisition

Routes of acquisition

Natural or wild virus
- Acute poliomyelitis

OPV vaccine-related virus
- Vaccine-derived polio
- Vaccine-associated paralytic poliomyelitis

Strain circulation in areas of low immunity to susceptible
- Reversion in the TGI of neurovirulence
*LB IDP
The global and regional epidemiological situation summary of poliovirus circulation is presented in this epidemiological update, as well as information on a confirmed vaccine-derived poliovirus case recently reported by Peru. The Pan American Health Organization / World Health Organization (PAHO/WHO) reiterates to Member States the importance of achieving and maintaining polio vaccination coverage greater than 95% in each district or municipality to minimize the risk of a poliomyelitis outbreak, strengthen epidemiological surveillance of acute flaccid paralysis (AFP) and update national poliovirus outbreak preparedness and response plans to detect and respond promptly and timely to an importation of wild poliovirus or vaccine-derived poliovirus (VDPV), or the emergence of a VDPV in any country of the Region.
Pathogenesis & clinical manifestations

Entered via oral or respiratory route [Incubation 7-14 days]

- Dissemination via lymphatic route
  - [90-95%] SUBCLINICAL Form
  - Fever, headache, discomfort, vomiting, rhinorrhea

- Dissemination via blood
  - [4-8%] ABORTIVE Form

  Central Nervous System
  - Virus in CSF + Symptoms of meningitis
  - < [1%] PARALYTIC form
    - Acute Flaccid Paralysis [Surveillance]
    - Bulbar involvement [High mortality +/-50%].
**Poliomyelitis**

**Cuadro Clínico**

- **Mild illness**
  - Fever, headache, odynophagia and fatigue.
  - Self-limited [2-5 days].

- **Serious illness**
  - Non Paralytic Poliomyelitis
    - IP [7-21 days].

- **Paralytic Poliomyelitis**
  - Absence of motor weakness
  - Symptoms typically include fever, headache, vomiting, and meningismus
  - Recovery [3-10 days].
Paralytic Poliomyelitis

ASYMMETRIC paralysis, ascending, proximal predominance, flaccidity or atony, intense myalgias, loss of reflexes, atrophy [1-5/200 cases]. Appears 1-2 weeks after nonspecific or mild symptoms.

CRANIAL NERVES III, VII, IX, X, XI
Paralysis of eye, face, pharynx [dysarthria], palate [dysphagia].

BULB: severe respiratory and circulatory damage [death].

Onset of pain and weakness may coincide with or follow the onset of a serious illness.
Paralytic Poliomyelitis

Flaccid paralysis without noticeable atrophy

Gluteal asymmetry
Síndrome Post Poliomielitis

Neurología condición afectando a los sobrevivientes del polio décadas después de su primer contagio [+/- 15 años], afectando hasta el 20-85%. Marca: participación motora

Nueva & persistente inicio de debilidad muscular progresiva, atrofia muscular, fatiga de los miembros, dolor muscular, artralgias, disfagia, fatiga generalizada.

A pesar de su alta prevalencia:
- Etiología es incierta
- Mecanismos de progresión son desconocidos
- Investigación limitada

Puede persistir durante décadas con impacto en la calidad de vida.
Neuropsicología aspectos son muy importantes, especialmente trastorno del sueño y polifarmacia.

Global Polio Eradication Initiative [GPEI]

- Routine childhood immunization
- Supplementary immunization campaigns
- Sweeping campaigns
- Flaccid paralysis surveillance **
Acute Flaccid Paralysis

Clinical picture of decreased or loss of strength and muscle tone of one or more limbs in children under 15 years of age, with acute or hyperacute onset in less than 5 days.

Surveillance for eradication of poliomyelitis.

- Guillain Barré Syndrome 40-60%
- Transverse myelitis 17%
- ADEM 15%
- Vaccine-associated polio/poliomyelitis
- Polio-like EV-D68 and EV-A71
- Traumatic neuritis
- Botulism
- Myasthenia gravis
- Toxins: organophosphorus heavy metals
- Neurotoxins: tick bites

*Neuroepidemiology. 2023;57:25-34; Rasul CH, Das PL et al. Med J Malaysia. 2002; 57(1): 61-5*
Approach + Management
Acute Flaccid Paralysis

Findings of spinal cord compression/sensory level on examination.

Immediate MRI of the spine
Non-compressive myelopathy
Compressive myelopathy
Neurosurgery + steroids
Acute transverse myelitis: steroids IV
Oculobulbar commitment?
CPK, K Urinary myoglobin
Polio, GBS, traumatic neuritis
Symmetrical?
Viral myositis
Periodic paralysis
Rhabdomyolysis
SGB
IGEV Neurology CSF
Diagnosis of polio

Confirmation of polio cannot be based on clinical manifestations.
* Other conditions must be excluded
* Not all cases of polio will present with paralysis

Definitive diagnosis includes:
* Stool samples collected < 2 weeks
* Samples > 2 weeks do not exclude diagnosis
* Clinical examination > 2 weeks to confirm if samples were not collected early

Residual paralysis suggests diagnosis
Each case of flaccid paralysis identified suggests possibility of poliovirus circulation: [Not the only one].
Diagnostic challenge

Immediate reporting of each case of flaccid paralysis is mandatory.

Early evaluation with appropriate sampling & differential diagnosis is imperative:
Rule out poliomyelitis
Gracias!