PNS Infections

→ PNS dysfunctions-
1. Sensory disturbance → numbness, ⊗ sensation tingling, burning.
2. Motor weakness → ↓ muscle mass painful cramps or fasciculations.
3. Autonomic disturbance.
4. All (Sensory, motor and autonomic)
↳ causes g- vascular, inflammatory or autoimmune causes or infection.

Rare, underrecognized but treatable

Viruses

HIV ② Herpes ③ Poliovirus

① HIV

↳ Retrovirus.
↳ Transmission g- sexual contact and contaminated food.
↳ Affects both CNS and PNS.
↳ forms of PNS dysfunctions-
1. Inflammatory demyelinating polyneuropathy (facial nerve paresis. Ascending weakness, generalized areflexia, Mild sensory involvement)
2. Mononeuropathy multiplex (Cranial nerve, median nerve, peroneal and ulnar nerve involvment, meralgia paresthetica.)
3. Polyradiculopathies (Radiating pain in cauda equina, flaccid paraparesis mild sensory loss, areflexia, sphincter dysfunction)
4. Distal symmetric polyneuropathy (DSP)

Early in the disease ← 1, 2, 3
Advanced infection ← 4

(Hyperesthesia, normal strength, Pain, ↓ ankle reflexes, ↓ response to pinprick and temperature, contact sensativity
⇒ Paresthesia of numbness in a stocking-glove distribution.

DSP → Most common PNS complaint (30-50%)
- Neurotoxicity of the virus.
- Neurotoxicity of cART (treatment).

② Herpes

↳ Larg, double-stranded, linear DNA genome.
↳ Latent. ↳ Recurring infection

α Herpes virusis:
- HSV1 → orofacial lesions
- HSV2 → Genital lesions
- Varicella Zoster virus (VZV) → chicken pox

Latency site is sensory nerve ganglia

Varicella ⇒ chicken pox
Zoster herpes ⇒ shingles. ← Recurs as shingles

↳ Peripheral sites (mucosal epithelial) → Termini of sensory neurons of the PNS → cell bodies ⇒ genomes are defosited in the nucleus (Latency)

↳ If reactivated, infections spreads toward
- the periphery
- The CNs (Trans-neuronally) → Lethal encephalitis.

↳ VZV g-
→ Primary infection occure in childhood (small and itchy skin rash that scab over).
→ Reactivation occurs in ① Elderly. ② immunosuppressed.
→ Treatment g- antiviral (acyclovir, valacyclovir, famciclovir) for 7 days. (Early treatment is recommended)
→ Most common PNS commplication ⇒ postherpetic neuralgia.

PHN g- chronic neuropathic pain condition (dermatomal distribution pain after shingles) that persist ≥ 3 months.

↳ Types of Pain ⇒ constant deep, aching of burning, paroxysm lancinating pain, hyperalgesia (more painful than expected) and allodynia.
↳ Treatment g- 1. Alpha-2 delta ligands (gabapentin and pregabalin) (Pain with non-painful stimuli)
2. Anticonvulsants (carbamazepine). 3. Tricyclic antidepressants (amitriptyline, nortriptyline, doxepin)
4. Topical analgesics (lidocaine patch, capsaicin). 5. Tramadol (opioids).

③ Poliovirus

↳ Enterovirus. ↳ Polio or infantile paralysis.
↳ 72% → Asymptomatic. 1% ⇒ flaccid paralysis.
↳ Diagnosis ⇒ 1. Viral recovery from the stool. 2. Antibody titer in blood.
↳ Rare (vaccines).

Bacteria

① Borrelia burgdorferi ② Clostridium tetani ③ Clostridum botulinum
④ Campylobacter jejuni ⑤ Mycobacterium leprae.

① Borrelia burgdorferi

↳ Lyme disease (multisystem). ↳ Tick-born.
↳ Peripheral nerve disorders g- 1. cranial neuropathneis.
2. Radiculopathies. 3. Diffuse polyneuropathies.
↳ Diagnose ⇒ 1. Clinical presentation. 2. History. 3. Serology.
↳ Doxycycline for lyme.
ceftriaxone

② Clostridium tetani

↳ Spore-forming, anaerobic, gram +ve rod.
↳ Produseces tetanospasmin ⇒ inactivates proteins regulat the inhibitory neurotransmitters (Glycine and GABA) ⇒ Excitatory synaptic activity ⇒ Spastic Paralysis.
↳ Rare (vaccines)

↳ trasmus or lockjaw (Masseter muscles) ⇒ muscle patient sardonic smile because of contraction of facial muscles is characteristic.

↳ Diagnosis: 1. Physical exam. 2. Immunization history. 3. Clinical presentation.

↳ ICU is recommended (quit room). Some patient require mechanical ventilation.

↳ Treatment: 1. wound debridement (the most important) and toxin mitigation
2. Tetanus immune globulin (3000 – 6000 unit) as soon as tetanus is suspected.
3. Metronidazole 4. Penicillin G ⇒ second-line therapy

↳ Death is due to involvment of respiratory muscles.

(3) Clostridum botulinum

↳ spore-forming, anaerobic, gram +ve rod. ↳ Tetanus.

↳ foodborn botulism ⇒ canned foods, over 1-3 days.

↳ Bilateral descending weakness (flaccid paralysis)

↳ Death because of respiratory paralysis.

↳ Infant botulism ⇒ ingestion of foods (honey, milk powder) contaminated botulinum spors ⇒ neurotoxin colonizes the GI tract ⇒ neuromuscular junction ⇒ botulinum endopeptedase inactivates the proteins regulate acetylcholine ⇒ ✗ neurotransmission at cholonergic synapses.

↳ Diagnosis: 1. Clinical symptomes. 2. Presence of toxin in the serum, stool or food.
3. Culturing from stool or wound.

↳ symptomes in adults: Diplopa, blurred vision, ptosis, slurred speech dysphagia, dry mouth and muscle weakness.

↳ Treatment: 1. supportive care. 2. Antitoxin ⇒ for food-borne, intestinal and wound exposure, not proven for inhaled botulinum.

(4) Campylobacter jejuni

↳ zoonotic infections. ↳ Bacterial gastroenteritis.

Shigella (most common) Salmonella Campylobacter

↳ Major triggering agent of Guillain-Barre syndrome (GBS)
→ Immune demyelinating polyneuropathy of PNS
→ Acute or subacute symmetrical ascending weakness, areflexia and mild to moderate sensory abnormalities.

↳ Molecular mimicry between sialylated lipooligosaccharide on the cell envelope and ganglioside on human nerves ⇒ Autoimmune driven nerve damage.

↳ Grow under selective conditions (microaerophilic).

↳ Microscopic morphology ⇒ Curved, gram-ve rods.

↳ Treatment: (Mostly self-limited).
1. Ventilator support. 2. Plasma exchange.
3. Intravenous immunoglobulin (IVIg).

↳ Corticosteroids are ineffective GBS.

(5) Mycobacterium leprae.

Hansen's disease

↳ Leprosy as one of the most common causes of nontraumatic peripheral neuropathy. ↳ Rare disease.

↳ Affects the 1. skin 2. Peripheral nerves. 3. upper respiratory tract. 4. Eyes.

⇓

symptomes: hypo or hyper pigmented skin macules with anesthesia (loss of sensation) infection of the peripheral nerves.

↳ it multiplies (unimpeded by organism-specific immunity) in Schwan cells ⇒ Destructs myeline, secondary inflamatory changes and destruction of the nerve.

↳ Morphology is same to M. tuberculosis.

↳ Diagnosis: 1. Biopsy (skin or nerve) 2. Acid Fast stain.

↳ Treatment: Multidrug therapy (MDT).

↳ The presence of the bacteria doesn't mean the presence of leprosy, it depends on the host immunity