Various Neuropathies

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Vasculitic Neuropathies

Peripheral nerves have ***low metabolic demands*** + ***extensive collateral circulation***:

* invulnerable to occlusion of *large peripheral arteries*;
* susceptible to *small blood vessel* diseases (focal circulation interruption in vasa nervorum - individual nerve fascicles) - many types of systemic vasculitis affect peripheral nerves!

Etiology

* 1. polyarteritis nodosa (nerves are most frequently damaged organs!)
  2. RA, SLE, Sjögren syndrome, systemic sclerosis
  3. vasculitides associated with infections (hepatitis B, Lyme disease, HIV).
  4. Churg-Strauss syndrome
  5. Wegener granulomatosis
  6. vasculitis restricted to PNS - special diagnostic challenge, because footprints of systemic inflammatory disease (e.g. ESR↑) are often absent.

Clinical Features

- reflect patchiness of underlying disease; characteristically – mononeuropathy multiplex:

* **asymmetry & length-independence**.
* evolves in stepwise fashion (e.g. wristdrop → contralateral footdrop → patchy areas of subjective numbness or sensory loss elsewhere on extremities).
* cranial nerve involvement, respiratory complications, and sphincter dysfunction are uncommon.

Diagnosis

In absence of diabetes mellitus, vasculitis becomes prime diagnostic consideration!

* screening to detect systemic vasculitis.
* vasculitis is histologic diagnosis - if no other organ involvement is identified → combined nerve and muscle **biopsy** (***axon loss***).
* **CSF** typically is normal (except with SLE).

Treatment

- treatment of underlying vasculitis.

* vasculitis restricted to PNS - **corticosteroids**, but most patients require **cytotoxic therapy** (as in polyarteritis).

Critical Illness Polyneuropathy

* occurs in critically ill patients (sepsis, multiple organ failure, etc).
* pathophysiology unknown (dietary deficiency is not considered candidate).
* severe sensorimotor neuropathy (***axon loss***).
* patients experience difficulty being weaned from ventilators.
* complete recovery may occur if underlying cause of multiple organ failure is successfully treated.

Toxic Neuropathies

* persons with *pre-existing nerve disease* are unusually susceptible to neurotoxins!
* most, although not all, neurotoxins produce **distal** ***axonal degeneration*** – distal sensory loss, loss of ankle tendon reflexes, distal weakness.
* **sensory** component suffers most;

toxins that produce predominantly **motor** neuropathy:

* 1. lead
  2. *n*-hexane (glue sniffer's neuropathy)
  3. tri-ortho-cresyl phosphate (”ginger jake”) - adulterant in illegal liquor (moonshine)
  4. dapsone (leprosy treatment)
* with continued exposure, symptoms may progress **proximally**.

***coasting*** - continuing progression even after offending agent is withdrawn.

* key to treatment - prompt recognition and withdrawal.

specific therapy for metal poisoning - D-penicillamine.

Bibliography for ch. “Peripheral Neuropathies” → follow this [link >>](http://www.neurosurgeryresident.net/PN.%20Peripheral%20Neuropathies\PN.%20Bibliography.pdf)

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