Plexopathies

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ETIOLOGY

1. Trauma
2. Neoplastic compression/infiltration (early prominent pain is characteristic!)
3. Radiation (painless and progressive weakness, usually bilateral but asymmetrical)
4. Immunologic attack (e.g., brachial neuritis, e. PARSOMAG-TURNER syndrome)
5. Diabetes mellitus
6. Neurofibromatosis

CLINICAL FEATURES

- etiology is complex (difficult to recognize and localize) - different patterns of motor and sensory loss - depending on which portion of plexus is affected.
- best clue is motor & sensory deficit that involves more than one spinal or peripheral nerve

- motor signs (weakness, tendon jerk loss, atrophy) are much more prominent than sensory changes (often patchy and incomplete).
- diffuse aching pain (sometimes quite severe) is often present!

- plexus avulsion pain is usually severe and immediate in onset: constant burning, crushing + intermittent shocklike pain

DIAGNOSIS

- imaging: N.B. high-resolution MRI is modality of choice!

1. empty enlarged nerve root sleeve, often with contrast extravasation through rent in dura
2. pseudo-meningocele (meninges pulsed through intervertebral foramina) at levels of root avulsion.
3. failure to visualize avulsed intradural roots in cervical? region when uninvolved roots are clearly visible.

- roots usually seem intact in lumbosacral plexus avulsion injuries
4. look for neurogenic at stumps of avulsed roots:

- pure root injuries leave dorsal root ganglion intact - distal nerve conduction velocities are intact.
- evoked potential (after median nerve stimulation) shows delay at Elb's point.
- N9 dorsal root ganglion evoked potential is preserved in pure root avulsion.

- CSF may contain blood.

TREATMENT

- acute transaction (lacerations with knife or glass) → rapid primary repair.
- closed stretch injuries with severe axonal degeneration 3-5 months after injury → surgical exploration and repair.
- missile wounds (usually leave nerve in continuity) - initial management is often conservative.
- avulsion of roots - unretractable injury: implantation of ventral roots into spinal cord may lead to recovery of motor function in animal and human studies.

- not improving obstetrical palsy → surgery at 3-9 months of age.

Pain management:

- plexus avulsion pain → DREZ lesioning.
- distal stretch injuries → spinal cord or deep brain stimulation.

PROGNOSIS

- because of long regeneration distances, intrinsic hand muscles and muscles below knee reinnervate poorly after axon loss lesions.

BRACHIALPLEXOPATHIES

Trauma is most common cause! other causes – see above >>

1. Compressive injuries
   - neoplastic: plexopathies are characteristically painful: >70% involve lower trunk and are due to axillary lymph node infiltration.

2. Open injuries
    - most often affect infrasacralic plexus; often associated with injuries to major vessels and lung.

3. Closed injuries
    - birth trauma – see p. Ped79 >>

   a) suprascapular - usually occur after high-speed motor vehicle accidents, often when rider is thrown from motorcycle, resulting in severe stretch injuries or avulsion of roots from cord. Hommer’s syndrome strongly suggests avulsion.
   b) infrasacralic - better prognosis (result of bony injuries in shoulder region; clavicular callus may compress plexus).

CLINICAL SYNDROMES

COMPLETE BRACHIAL PLEXUS lesion – flail, anesthetic upper extremity (except for medial strip along arm supplied by intercostobrachial branch of 2nd intercostal nerve).
DIACONI-ERMATONICplexus (C5-6 roots or upper trunk lesion)

Causes:
1. Most common cause - downward arm displacement: fall from horse or motorcycle, obstetrics (shoulder dystocia).
2. direct pressure by carrying heavy objects (knapsack,Palay), heavy backpacks; prolonged head misalignment, shoulder restraint in motor vehicles.
   - long necks, droopy shoulders, pendulous breasts may be contributing factors.
3. idiopathic brachial neuritis (s. PARSONAGE-TURNER syndrome, neuralgic amyotrophy).
4. radiation-inducedplexopathy.

Clinical features - mainly should & upper arm muscles (deltoide, biceps, brachialis anticus, brachioradialis, pectoralis major, supraspinatus, infraspinatus, subscapularis, teres major) - "waller's tip" position:
- upper arm hangs adducted (m. deltoideus - n. axillaris) and internally rotated (m. infraspinatus - n. suprascapularis).
- can't flex elbow; forearm is pronated (m. biceps brachii - n. musculocutaneous).
- can't reach with hand contralateral shoulder (clavicular head of m. pectoralis major - n. pectoralis lat).
- sensory loss is incomplete (hypesthesia on outer surface of shoulder, arm and forearm).

DIACONI-KLUMPKE'S plexus (C7-8 roots or lower trunk lesion)

Causes:
1. upward arm displacement: obstetrics (breech delivery), shoulder dislocation.
2. metastatic plexopathy (axillary lymph nodes), infiltrating tumor from lung apex (Paracost tumor).
3. true neurogenic thoracic outlet syndrome, cervical rib, scalenue syndrome.
4. coronary artery bypass surgery (associated with sental retraction).

Clinical features - mainly forearm & hand muscles:
- n. ulnaris n. medianus (flexor carpi ulnaris, flexor digitorum, interneus, thanar and hypothenar) - can't flex wrist, "claw hand", "s audition (flattened) hand".
- n. pectoralis med. - can't adduct upper arm.
- lesion to communicating branch to inferior cervical ganglion → Horner's syndrome.
- sensory - hypethesia on inner arm / forearm / hand.

Middle Radicular Syndrome (C5: root or middle trunk lesion) - paralysis of n. radialis muscles (except brachioradialis, which is spared entirely).
- sensory loss is incomplete (hypesthesia over dorsal forearm surface and external part of dorsal hand surface).

"BURNERS" / "STINGERS" - symptoms following sudden shoulder depression (in contact sports, usually football):
- burning dysesthesias going down ipsilateral upper extremity (often into thumb) ± hypersensitivity on inner arm / forearm / hand.
- symptoms resolve within few minutes (occasional cases last for weeks).

Root avulsions more commonly involve C5-8 roots, whereas extraforaminal ruptures more commonly affect C3-C4 roots.

DIAGNOSIS
- cervical myelography or MRI (2-4 weeks after injury) - traumatic pseudomeningoceles at site of avulsed nerve roots.

*root avulsion is generally not investigated radiologically in acute stage

TREATMENT
- flail or weak arm should be supported (immobilized across upper abdomen) against gravity to prevent additional damage!
- injury by sharp object (knife, glass, needles) → early surgical intervention.
- lost neural tissue during initial exploration (for repair of other injuries) → early grafting (after allowing local edema to resolve).
- blunt injuries → observation: situation depends on proximal or distal location of injury.
- gunshot wounds → observation for up to 3 months (to help establish degree of neural injury); if serial examinations demonstrate 4-5° lesions → surgical intervention.
- root avulsions (flail arm) → grafting of intercostal nerves to distal end of musculocutaneous nerve (gives useful elbow flexion when combined with distal limb prosthesis).

NEURALGIC AMYOTROPHY (s. brachial plexitis, PARSONAGE-TURNER syndrome, shoulder-girdle syndrome)

Similar disorder may affect LUMBOSACRALplexus:
- unknown cause (sporadic > familial) - viral or immunologic inflammatory processes?
- typically young men.
- often preceded by some antecedent event (e.g. upper respiratory infection, hospitalization, vaccination, non-specific trauma, intraovarian, genital, general anesthesia!!!).
- may be bilateral and asymmetric.
- upper trunk, suffers most (actually, multiple proximal mononeuropathies):
  - sudden onset of severe pain (usually about shoulder; often begins at night).
  - soon followed by weakness and wasting of various forearm muscles (esp. shoulder girdle);
  - weakness is maximal within few days then regresses.
- nerve conduction studies - axonal neuropathy (denervation) may play role in rare instances.
- CSF is normal.
- establish diagnosis - EMG
- corticosteroids have no proven benefit.
- clinical recovery takes 2-4 months or 3 years (so don’t rush to operate!!!) - good in 66%, fair in 20%, poor in 14%; if no improvement by 18-24 months, may recommend tendon transfer surgery.

LUMBOSACRAL PLEXOPATHIES

ETIOLOGY
1. most frequently - penetrating injuries.
   - NB: plexus is better protected in its retroperitoneal & pelvic location - injury is not as common as brachial plexus injury?
2. hip surgery, pelvic fractures, pelvic hematoma in possum muscle (e.g. due to anticoagulation).
3. labor & delivery (pressure by fetal head or forceps).
4. direct neoplastic infiltration.
5. radiation induced fibrosis (painless and progressive weakness, usually bilateral but asymmetrical)
6. idiopathic plexitis.
CLINICAL FEATURES

- most of motor output of lumbar plexus is in femoral nerve; of sacral plexus in sciatic nerve - it may be difficult to distinguish lumbosacral plexus lesions from lesions of their respective nerves.
  - weakness of thigh adduction or sensory loss in inguinal region or over genitalia are outside distribution of femoral nerve.
  - weakness of thigh abduction & internal rotation and of hip extension, or sensory loss on posterior thigh are lacking in sciatic nerve palsy.

BIBLIOGRAPHY for ch. “Peripheral Neuropathies” → follow this LINK >>