Reflex Sympathetic Dystrophy (Complex Regional Pain Syndrome)

Arthur Kitt

Risk

- Incidence of 5.5:100,000 person years at risk (50,000 new cases per y in USA).
- Prevalence of 21:100,000 person years at risk.
- Female:male ratio is 2–4:1.
- Mean peak age is 37–50 y.
- Incidence of CRPS I is 1–2% after fractures, 12% after brain lesions; and 5% after MI.
- Incidence of CRPS II is 1–5% after injury to a peripheral nerve.

Perioperative Risks

- Increased pain flare postop if procedure is on affected extremity
- Increased tolerance and/or opioid requirements if managed with chronic opioids
- Increased incidence of comorbid anxiety and depression

Worry About

- Exacerbation or recurrence of CRPS with manipulation of affected extremity.
- Careful positioning of affected extremity.
- Interactions and/or end-organ effects of chronic pain medications.
- IV access and/or tourniquet placement on affected extremity may be intolerable because of pain.

Overview

- Spontaneous intractable burning pain; allodynia; hyperalgesia
- Edema, autonomic (vasomotor/sudomotor) abn, trophic signs
- Significant decrease in normal function of affected limb
- Symptoms not limited to the region of a single nerve (other causes ruled out)
- Pain disproportionate to inciting event
- Dx largely based on pt's Hx and clinical criteria such as the Budapest criteria
- No diagnostic "gold standard" test
- CRPS I (reflex sympathetic dystrophy): No clear evidence of nerve damage
- CRPS II (causalgia): Clear evidence of nerve damage with inciting event

Etiology

- Pathogenesis of CRPS unknown
- Classically associated with antecedent trauma, surgery, MI, stroke
- Probable involvement of peripheral, autonomic, and central nervous systems; myofascial dysfunction; altered psychological states
- Many proposed mechanisms:
  - Abnormal sympathetic outflow and/or adrenergic receptor sensitivity
  - Abnormal spinal and/or central neuronal sensitization
  - Abnormal and/or exaggerated inflammatory process
  - Neurogenic inflammation
  - Psychological and/or psychogenic factors
  - Genetic predilection with HLA-DR/DQ polymorphisms

Usual Treatment

- Early Dx and multidisciplinary treatment associated with best outcomes
- Physical therapy using desensitization and ROM exercises most important component of rehabilitation to achieve optimal functional restoration
- Psychological intervention, cognitive-behavioral therapy
- Typical first-line oral medications:
  - Antidepressants (SNRIs, TCAs)
  - Antiepileptics (gabapentin, pregabalin)
  - NSAIDs
- Chronic opioid therapy controversial
- Oral corticosteroids employed if inflammatory component is prominent
- Other adjuvant and second-line therapies:
  - NMDA antagonists (ketamine, memantine)
  - GABA agonists (intrathecal baclofen)
  - Bisphosphonates
  - Free radical scavengers (DMSO, NAC)
  - Alpha-2 agonists (epidural clonidine)
- Interventional therapies:
  - Sympathetic ganglion blockade
  - Chemical/surgical sympatholysis
  - Regional IV infusion therapy (lidocaine, reserpine, guanethidine)
  - Neurostimulation (SCS, TENS, deep brain stimulation)

Assessment Points

System	Effect	Assessment by Hx	PE	Test
DERM	Skin/hair/nail changes	Changes in limb appearance Increased/decreased hair growth	Thickened/thin skin Glossy, waxy skin Increased/decreased hair growth Thickened/brittle nails	Serial physical exams Comparative exam photos
MS	Muscle mass/strength change Stiffened joints Bone changes/osteoporosis	Subjective weakness Decreased ROM	Muscle atrophy Objective weakness Decreased active/passive ROM	 Three-phase bone scintigraphy Radiographs
PNS	Spontaneous pain Allodynia/hyperalgesia Motor changes	Spontaneous pain Pain to nonnoxious stimuli Exaggerated pain to noxious stimuli	Allodynia Mechanical/thermal hyperalgesia Tremor/dystonia	Quantitative sensory testing (thermal/ thermal/pain/ vibratory)
ANS	Vasomotor/sudomotor abnormalities	Hyperhidrosis/hypohidrosis Temp changes Swelling	Moist, clammy, cool skin Edema, skin color changes Skin temperature asymmetry of limbs	QSART Infrared thermometry/thermography Thermoregulatory sweat test

Key References: Stengel M, Binder A, Baron R: Update on the diagnosis and management of complex regional pain syndromes, *Adv Pain Manag* 1:96–104, 2007; Bussa M, Guttilla D, Lucia M, et al.: Complex regional pain syndrome type I: a comprehensive review, *Acta Anaesthesiol Scand* 59(6):685–697, 2015.

Perioperative Implications

Preoperative Preparations

- Preop PE noting location of pain symptoms as well as neurologic and/or MS deficits.
- Careful planning of pt positioning.
- Consider combined regional/GA for periop and postop pain control.
- Make a detailed plan for postop pain-control strategies.
- Wait until acute phase of CRPS has resolved.

Monitoring

- Standard ASA monitors.
- Avoid BP cuff, pulse oximetry, or other monitors on the affected extremity.

Induction

- Possible increased dosage of induction agent (for pts on chronic opioids).
- Consider regional blockade and/or catheter infusion.

Maintenance

- Possible increased anesthetic and periop opioid requirements (for patients on chronic opioids)
- Diligent assessment of affected limb position and temperature

Adjuvants

- Continuation of all neuropathic pain medications if possible.
- NMDA antagonist bolus and infusion (ketamine) and/or other neuropathic meds can possibly avoid central sensitization (windup).

Postoperative Period

- Continue regional anesthesia and/or analgesia postop if feasible.
- Resume preop pharmacologic regimen (home medications).
- Facilitate early mobilization.
- Consider pain medicine consultation if pt is admitted postop.

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