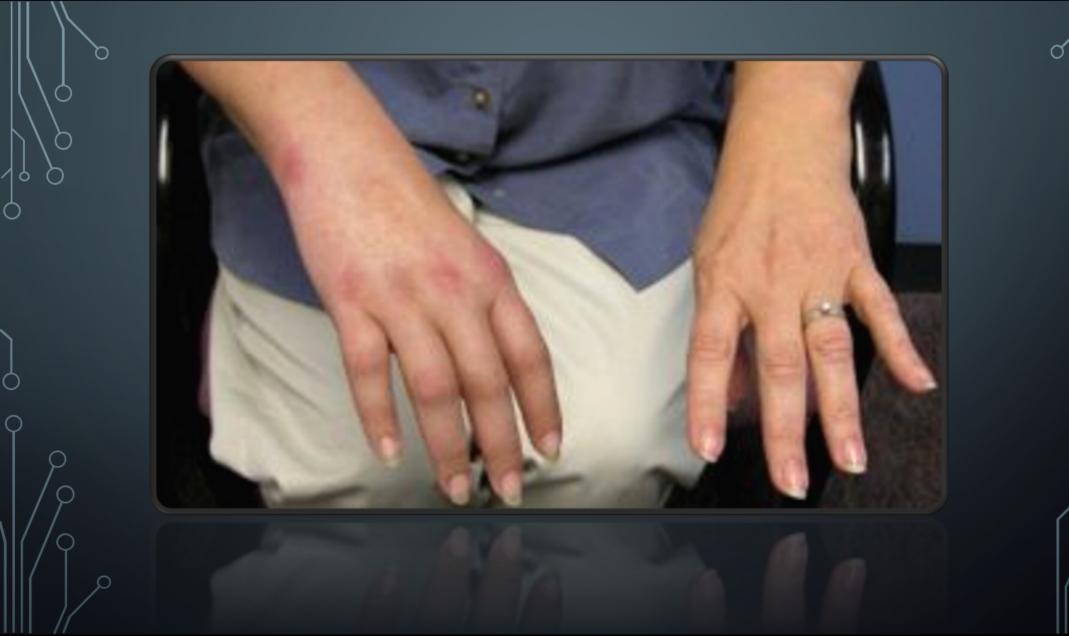
به نام خدا

دکتر محمد حسین عطایی فلوشیپ درد



DEFINITION

A neuropathic pain syndrome .lt is distinct from other pain syndrome by presence of autonomic dysfunction, persistence regional inflammatory changes and a lack of dermatomal distribution .



HISTORY

Ambrose Pare, a French surgeon made reference to a severe persistence pain after phlebotomy.

In medical literature, Weir Mitchell an AMERICAN civil war physician described a syndrome consist of burning pain ,hyperesthesia, trophic changes following nerve injury in the limbs of the solidiers. He named this pain syndrome Causalgia.

In 1946 Evan devised the term REFLEX SYMPATHETIC DYSTROPHY to describe a similar syndrome in patients with no obvious neve damage. In 1995, International Association for study of pain introduced the name CRPS.

CRPS 1=REFLEX SYMPATHETIC DYSTROPHY(without direct nerve injury)

CRPS 2=CAUSALGIA(with direct nerve injury)

PATHOPHYSIOLOGY

Numerous theories have been offered to explain the pathophysiology of CRPS, but the exact mechanism remain unclear.

-A Neuroinflammatory process

A trauma or injury leads to beginning inflammatory process, exaggreted response in nociceptors,

Interaction between nociceptors and sympathetic efferent fibers,central sensitization(F .MRI,PET,Magnetoencephalogram...)

Three phase observed:

Acute phase ;Usually Warm phase of 2-3 months.(hypoactive sympathetic)

Dystrophic phase: vasomotor instability 3-6 months after onset.

Atrophic phase :usually cold extremity with atrophic changes.



DIAGNOSIS

IASP criteria:

1-continuing pain

2-at least one symptom in three of the four following categories:

SENSORY-Hyperesthesia and/or allodynia

VASOMOTOR-temperature asymmetry and/or skin color asymmetry

SUDOMOTOR-edema and/orsweating change

MOTOR/TROPHIC-decrease rang of motion and/or motor dysfunction(weakness,tremor,dystonia)and/or Trophic changes(hair-nail-skin).

3-Must display at least one sign at time of evaluation in two or more of following categories:

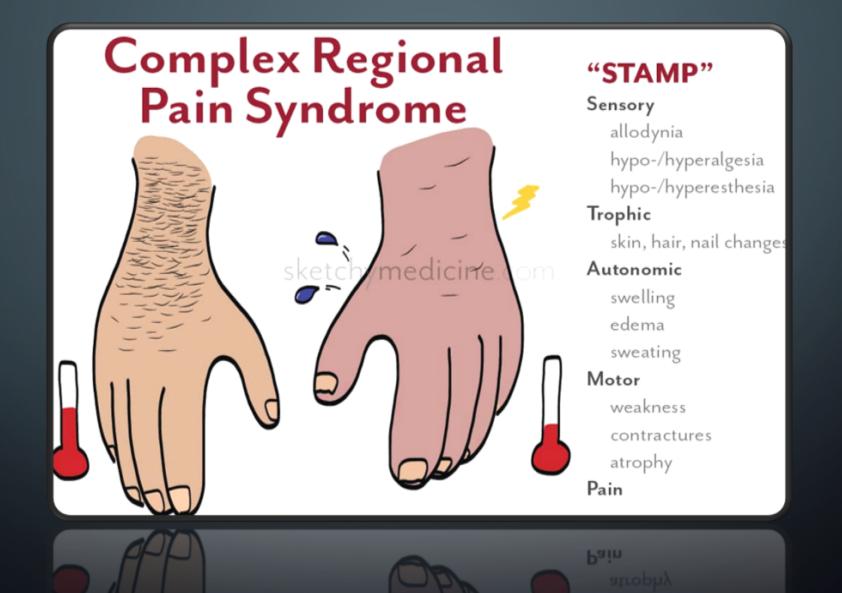
SENSORY;Evidence of hyperalgesia(to pinprick) and/or allodynia(to light touch and/or deep somatic pressure) and /or joint movement.

VASOMOTOR-evidence of temperature asymmetry and / orskin color asymmetry.

SUDOMOTOR-evidence of edema and/or sweatingchanges.

MTOR/TROPHIC-evidence of decrease range of motion and/or motor dysfunction and/ortrophic changes.

4-There is no other diagnosis that better explains the signs and symptoms.



EPIDEMIOLOGY

There is a 13-26 per 100/000 person incidence. Female predominance; more common in upper extremity, more common between 40-50 years old, more common in HLA-B62, HLA-A29, HLA-DQ8, more common in smokers, PTSD patients, anxious patients, In Antebrachial fractures in upper extremity, prolong general anesthesia for repair of Fractures, In R.A patients.

TREATMENT

NEED TO MULTIDISCIPLINARY APPROACH

REHABILITATION —BASED THERAPY

-PHYSICAL AND OCCUPATIONAL THERAPY AND PSYCHOLOGICAL THERAPY MUST BE STARTED AT TIME OF DIAGNOSIS

MEDICATIONS; NSAIDs, anticovulsants, tricyclic antidepressants, oral steroids, IV ketamine, Biphosphonates. Intranasal calcitonin, lidocaine patch,,,,

INTERVENTIONS

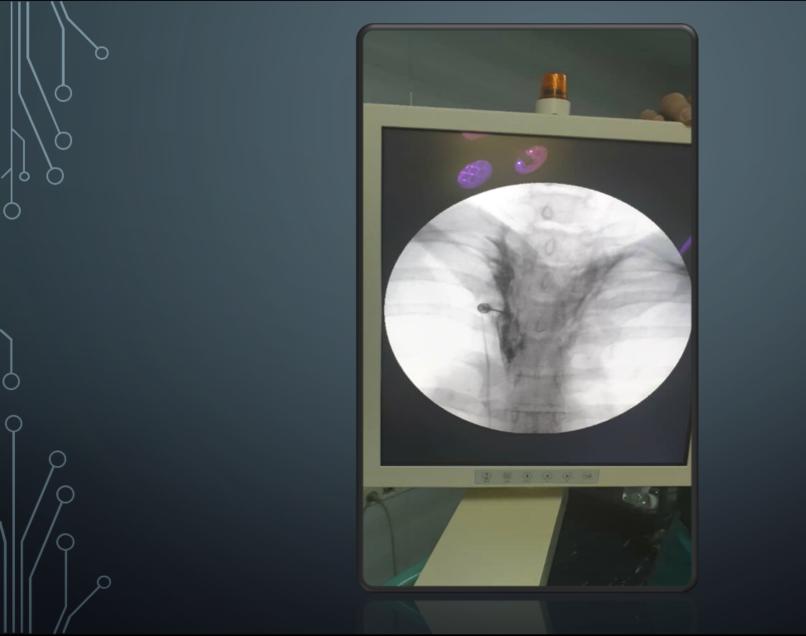
UPPER EXTREMITY SOMATIC BLOCKS(Interscalene, infraclavicula...)

(Cervical epidural)

SYMPHATETIC BLOCKs:

STELLATE GANGLION BLOCK

SECOND THORACIC GANGLION RADIOFREQUUNCY



SPINAL CORD STIMULATOR DORSAL ROOT GANGLION STIMULATORS

SURGERY

Surgical thoracic sympathectomy

Amputation

PREVENTION

VITAMINE C 500mg for 50 days

