# Complex regional pain syndrome or post traumatic pain syndrome

## Tribhuvan Narayan Singh Gaur<sup>1,\*</sup>, Veena Kachhawah<sup>2</sup>

<sup>1,2</sup>Associate Professor, Dept. of Orthopaedics, <sup>1</sup>Peoples College of Medical Sciences and Research Centre, Bhanpur, Bhopal, <sup>2</sup>Advanced college of Medical Sciences and Research Centre Kolar, Madhya Pradesh

#### \*Corresponding Author:

Email: tribhuwan\_dr@rediffmail.com

#### Abstract

Post traumatic pain syndrome or regional pain syndrome (CRPS) consists of abnormal pain, swelling, vasomotor and sudomotor dysfunction, contracture and osreoporosis. This entity described and literature with many synonyms like reflex sympathetic dystrophy, sudek's dystrophy, cusalgia.

Transient migratory osteoporosis, painful osteodystrophy or alegodystrophy etc. It may be primary or secondary, in primary it occurs directly to the damage tissue and in secondary it occurs in surrounding undamaged tissue. Orthopaedician must realize that these patient are not malingering and it is real perception of pain.

The patho-physiology is not clearly known. Treatment is multidisplinary involving medical, psychological and rehabilitation.

**Keywords:** Post traumatic, Pain syndrome, Multidisplinary treatment.

#### Introduction

Complex Regional Pain Syndrome combination of regional pain and sensory changes following trauma which exceed in magnitude and duration of anticipated healing period. The International Association for the Study of Pain defined it as "A variety of painful conditions following injury which appears regionally having a distal predominance of abnormal findings, exceeding in both magnitude and duration, the expected clinical course of the inciting event often resulting in significant impairment of motor function, and showing variable progression over time". There are two subtypes, CRPS I (Reflex Sympathetic Dystrophy) and CRPS II (Causa CRPS I (R S D): It is a syndrome of pain with hyperalgesia after an initiating, noxious event with symptoms disproportionate to inciting event and not limited to a single peripheral nerve. There is evidence of edema, skin blood flow abnormality or abnormal sudomotor activity. The diagnosis is by exclusion of condition that account for degree of pain and dysfunction. CRPSII (Causalgia)<sup>(1)</sup> is a syndrome of pain where a known nerve injury is responsible because not all pains are sympathetically maintained. The pain Sympathetically Maintained Pain (SMP) where the pain is dependent on the sympathetic activity in the affected area and is relieved by the blockade of the efferent sympathetic nervous system and Sympathetically Independent Pain (SIP) where pain is unresponsive to sympathetic blockade. Therefore, clinically a patient may present with either SMP or SIP or both i.e., part of chronic pain maybe SMP or part of chronic pain maybe SIP.

#### Incidence

CRPS can occur at any age, highest being among 50-70 years and upper limb being affected more

commonly in adults and the lower extremity in adolescents. Children below 7 years of age are more prone. It affects both men and women, although some studies show that it is more common in women (W:M = 4:1 ratio) and incidence in girls around 13 years is 80%. CRPS is seen with increasing frequency in injured workers, following surgery, myocardial infarction and stroke.

#### Etiology

CRPS may occur after any particular trauma while an identical stimulus in different limb does not cause it. The incidence is not changed by treatment method and open anatomic reduction and rigid internal fixation does not abolish it. It is unclear wheather injury severity or quality of reduction alter the incidence. There is however association with excessively tight plaster and may be a genetic predisposition. (2,3,4) The following etiology have been proposed.

- a. Psychological abnormality
- b. Abnormal (neuropathic) pain
- c. Sympathetic nervous system abnormality
- d. Abnormal inflammation
- e. Failure to use affected limb

**Symptoms:** CRPS presents with a wide range of symptoms and the hallmark of pain is that it is out of proportion to the inciting event2. It usually occurs in hands and feet which are rich in nerve innervations but can also present anywhere in the body. Pain can be spontaneous or stimulus evoked and does not progress sequentially. The disease progression is very variable and not time limited. Symptoms can be in the form of:

**Allodynia** – pain evoked by mechanical or thermal stimulus that usually does not cause pain, as light touch. **Hyperalgesia** – exaggerated response to a normally non-painful stimulus.

**Hyperpathia** – abnormal painful reaction to a stimulus.

### There are three stages of CRPS:

**Stage One:** 3 months from onset. There is severe, burning pain at the site of injury. Muscle spasm, joint stiffness, restricted mobility rapid hair and nail growth and vasospasm, affecting colour and temperature of skin.<sup>(5,6)</sup>

**Stage Two:** 3 - 12 months from onset.

This stage presents with more intense pain, swelling spreads, hair growth diminishes and nails become cracked, brittle, grooved and spotty. Osteoporosis becomes severe and diffuse, joints thicken and muscles atrophy.<sup>(6)</sup>

**Stage Three:** In this stage the pain is unyielding with irreversible changes in skin and bone, marked muscle atrophy, severely limited mobility of affected area and may involve the entire limb. Flexor tendon contractions with occasional limb displacement from normal position and marked bone softening and thinning are observed.<sup>(6)</sup>

Diagnosis: Diagnosis is mainly dependent on the clinical course. A detailed medical history of the initial trauma, history of sensory, autonomic, development, distribution disturbances, characteristic of pain and time course help in clinching the diagnosis. Detection of any swelling, sweating, trophic, temperature and motor abnormality in disturbed area are important. Muscle strength of somato-sensory abnormalities, affected limb, characteristic or distribution details, elicitation of pain on movement and pressure at joints must be tested. (2) The other diagnostic methods include:

**Bone Scintigraphy:** This is positive in Sub-acute period about (1 year) showing vascular bone changes. Radio-labelled technetium anti-TNF  $\alpha$  to image TNF  $\alpha$  scintigraphically and found uptake in clinically affected han.

**Radiography:** This is useful in chronic cases of CRPS only. It shows patchy osteoporosis due to disuse, two weeks after onset of CRPS. Bone scan and Bone densitometry are useful guide for follow up.

**QST** – **Quantitative Sensory Testing:** Testing of thermal and thermal pain threshold gives information about functioning of unmyelinated and small myelinated afferent fibres of spinothalamic tracts.

**Autonomic Functions:** Infrared thermometry, Laser Doppler flowmetry, Infrared thermography and quantitative sudomotor axon reflex test (QSART) are new methods.

**Thermography:** Skin temperature differences of 1.0 C between two symmetrical body parts are significant.

**Sudomotor testing:** QSRT tell of function of sudomotor reflex loops and resting sweat output test may be used.

All the above investigations are used to follow the progress and response to treatment.

**IASP Diagnostic Criteria for CRPS:** 

- 1. Presence of an initiating noxious event, or a cause of immobilization
- 2. Continuing pain, allodynia or hyperalgesia with pain disproportionate to inciting event
- 3. Edema, changes in skin blood flow, abnormal pseudomotor activity in the region of pain
- Diagnosis is excluded by the existence condition that would otherwise account for the degree of pain and dysfunction

# Research Diagnostic Criteria for CRPS: Budapest Criteria.

(Modified IASP Research Diagnostic Criteria for CRPS)

- Continuing pain disproportionate to any inciting event.
- 2. Report at least one symptom in each of the four following categories.
  - a. Sensory: Hyperesthesia an orallodynia.
  - Vasomotor: Temperature asymmetry and or skin colour changes and or skin colour asymmetry in the affected region.
  - c. Pseudomotor / edema: Edema and or sweating changes and or sweating symmetry in the affected region.
  - d. Motor /Trophic: Decreased range of motion and or motor dysfunction(Weakness, tremor, dystonia) and or trophic changes (hair, nail, skin) in the affected region Presence of 2 symptoms and 2 signs gives 80% accurate diagnosis and presence of 4 symptoms and 2 signs as diagnostic criteria for CRPS is most effective criteria to rule in and rule out CRPS across different population.<sup>(6,7)</sup>

In conclusion, the diagnostic value of TPBS as a confirmatory test for CRPS according to the Budapest research criteria is low. The current study supports that no specific test is available for CRPS, which is diagnosed primarily through absent of the symptoms and signs.

**Differential Diagnosis**: (8) The following are common diagnosis

- a. Soft tissue infection
- b. Mechanical problem
- c. Conscious exaggeration of symtoms
- d. Psychiatric disease
- e. Neuropathic pain
- f. Chronic pain state

#### **Management:**

Management of CRPS is multi-disciplinary. There are very few proper prospective randomized study. (9) Most suffers are sensible people, concerned about the development ox inexplicable pain.

Early treatment, begun before the contractures occur, give optimal results, so high index ox clinical suspicious must be maintained. It is not reprehensible to

have caused a case of CRPS through surgery or non operative management of injury.

Mordern CRPS terament emphasizes functional rehabilitation of the limb to break the vicious cycle of disuse. (10,11) Initial treatment from the clinician is with reassurance, excellent anaigesia and intensive care physical therapy avoiding exaberation of pain. Nonsteroidal anti-inflamatory drug may give better pain relief than opiates, and centrally acting analgesics such as ampitryptyline is often useful even at this early stage. Immobilization and spintage should generally be avoided but, if used, joint must be placed in a safe position and spintage is temporarily adjunct to mobilization. It seems sensible to give the patient vitamin C in view of early evidence of its efficacy.

Abnormality in pain sensation will often respond to desensitization. The patient is asked to stroke the area of allodynia. Where stroking is painful. They are reminded that simple strokeing cannot by definition be painful and they are instructed to stroke the affected part repetively while looking at it and repeatedly saying this does not hurt, it is merely gentle touch. Earlier this is begun, the more effective it is. A similar attitude can be take with early loss of joint mobility due to perceived pain rather than contracture.

The use of mirror virtual therapy is an exciting new concept. (12) If patient does not respond rapidly, a pain specialist should be involved and treatment continued on shared basis. Psychological or psychiatric input may be important. (13) Second line treatment is often uncessful and patient are left with pain and disability. Further treatment included cantraly acting analgesics medication such as amitriptyline, gabapentin or carbazepine, regional anesthesia. calcitonin, use of blockade and manipulation, desensitization of peirphral nerve receptors by capsicum or trans cutaneous nerve stimulation therapy may be necessary in children., where the knee affected epidural anesthesia and continue passive motion may be appropriate.

The role of surgery is limited and hazardous, Genraly surgery represents a painful stimulus that may exacerbate CRPS or precipitate new attack. This risk must be balanced carefully against the proposed benefit. Idealy for contracuture relese there should be one year gapsice the patient last experienced of pain and swelling.

Amputation of limb affected severe CRPS should be approached for recurrent infection or to improve

#### Conclusion

CRPS is responsible for significant acute disability and may causes long term problems. Diagnosis is purely based on clinical features and diagnostic tests are not validated. It is distressing to note that no single drug or combinations work for all. Management of CRPS is multidisciplinary involving medical, psychological, physical and rehabilitation interventions.12,19 The advent of newer drugs and spinal cord stimulation and

motor cortex stimulation and neuraxial route of analgesia are promising for these patients with intractable CRPS.

#### References

- Anthony S. Fauci, MD, Joseph b. MARTIN, MD, PHD, Eugen Braunwald, AB, MD Hrrison. Princples of internal medicine 14<sup>th</sup> edition 1998 volume2;pg;2375.
- Devor M, Raber P. Hretiability of symptoms in an experimental model of neuropathic pain. Pain1990;42:51-67.
- Kimura T, Komatsu T, Hosoda R, et al. Angiotensin converting enzyme gene polymorphisim in patient with neuropathic pain. In: Devor M, Rowbotham M. Wiesenfeld Hallim D, eds. Proceeding of the 9<sup>th</sup> world conference of pain. Seattle, WA:IASP Press2000:471-476.
- Knepper R. (Pthogenic evaluation of pain). Med Welt 1967;35:1994-1996.
- Steven D. Waldman MD, JD Pain Review, Saunders Elsevier;2009; pg: 328-329.
- Baron R. CRPS: McMohan S. Koltzenburg. M(ed): Wall and Melzack's Text Book of Pain, ed.5.Philadelphia, Churchill Livingston, 2006.
- Dr. Craig W. Martin, Senior Medical Advisor CRPS-Towards the development of Diagnostic criteria and treatment guidelines. (by evidence based practice group) Jan 2004.
- Robert W. Bucholz, James D. Hekman, et al Rockwoog and Green's Fractures in adult vol 1:2010 Wolters Kluwer610-611.
- 9. Kingery WS. Acritical review of controlled clinical trial for peripheral neuropathic pain and complex regional pain syndrome. Pain1977;73:123-139.
- Harden RN. The rationale for integerated functional restoration. In Wilson P, Stanton Hicks M, Harden RN, eds. CRPS: Current diagnosis and therapy. Seattle, WA: IASP Press2005:163-171.
- Harden RN, Swan M, King, et al. Treatment of complex regional pain syndrome: Functional restoration. Clin J Pain 2006;106:393-400.
- MaCabe CS, Haigh RC, Ring EF, ET AL. A control pilot study of the utility of mirror visual feedback in the treatment of complex regional pain syndrome (type1). Rheumatology (Oxford) 2003;42;97-101.
- 13. Bruehl S, Husfeldt B, Lubenow TR, et al. Psychological difference between reflex sympathetic dystrophy and non-RSD chronic pain patient of pain.Pain1999:81:147-154.