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SUMMARY OF COMPLEX REGIONAL PAIN SYNDROME (CRPS)

CRPS is formerly known as reflex sympathetic dystrophy (RSD) or Causalgia. This is a summary of information in the book called ***Chronic Pain*** subtitled reflex sympathetic dystrophy prevention and management, by neurologist Hooshang Hooshmand, M.D. published in 1993, and in the more recent portion of a publication by psychopharmacologist/psychiatrist Stephen M. Stahl, M.D., Ph.D. called ***Chronic Pain and Fibromyalgia*** published in 2009, reprinted in 2013. I have added some information from recent papers on pubmed.gov.

Chronic pain occurs in 30% of the population. A third of the cases of chronic pain is complex regional pain syndrome, which requires special recognition and treatment in order to alleviate the pain, and prevent it from progressing. With regard to pain, there is a component which is sensorimotor, and a component which is sympathetic, eg, has neurotransmitters of adrenalin and noradrenalin. The sympathetic division of the autonomic nervous system follows the blood vessels, and causes vasoconstriction/temperature change in them in regard to injury. Because of this pattern, the pain patterns in CRPS are related to sympathetic thermatomes rather than sensory dermatomes, and can become bilateral, and encompass parts of the body remote from the original injury according to Dr. Hooshmand. Sympathetic pain is burning or stabbing and diffuse, rather than localized.

CRPS involves an increase of the intensity and physical area of the pain called hyperpathia or hyperalgesia. Furthermore, this hyperpathia, also known as the wind-up phenomenon, occurs not only at the original site of the injury, but then also at the spinal cord level where the peripheral pain fibers(c fibers and alpha delta fibers) enter the dorsal root ganglion, and travel through the spinothalamic and reticulothalamic tracts to the limbic and cortical systems. These tracts synapse in the periaqueductal gray (PAG), the hypothalamus, thalamus, limbic forebrain (cingulate gyrus), and mesial frontal lobe. A third wind-up phenomenon occurs in the brain, consisting of anxiety, hypertension, insomnia, and depression.

Because of the thermal changes in the skin in CRPS, the best means of diagnosis is thermography of the body, comparing both sides of the body (Hooshmand). It can detect 0.5 to 1 degree C temperature changes in 6 mm thickness of skin, but the deeper structures reflexly cause the skin temperatures measured. In 400+ of Dr. Hooshmand's patients, use of thermography increased the diagnosis from 5.75 to 22% .

A second major means of diagnosis is bone scan. However, bone scanning may be better early in the course of the CRPS, and the thermography may be better later in the course. EMG, nerve conduction velocities, and sensory evoked potentials are biomarkers of the sensory motor activity of the pain fibers as compared to the sympathetic component.

There are 2 types of causes of CRPS. Type one is called disuse/immobilization, and Type 2 is called ephaptic, which is a short circuit of neurotransmission. Ephaptic refers to a scar from an injury, whether it be electrical or penetrating or blunt force. The disuse type 1 is found more

frequently than the ephaptic type 2. Predisposing risk factors have not been identified. However, there is a paper on epigenetic modification of the dorsal root ganglion as a risk factor and potential target for treatment (Wang et al 2014 Med Sci Monit).

There are 4 stages of CRPS.

Stage 1 is marked by burning pain, which follows thermotomes rather than dermatomes, and also has spasm and tendency for immobilization in the disuse type. The duration in the disuse type is 1-3 months, and the duration in the ephaptic type is 2-8 weeks.

Stage 2 encompasses 3-7 months in the disuse type and 2-4 months in the causalgia/ephaptic type. The Stage 2 features vasoconstriction, unilateral cold extremity, hair loss, and tendency for weakness, tremor and spasticity (flexed arm, extended leg).

Stage 3, which is atrophy, occurs over 7 months in the disuse type and over 4 months in the causalgia type. The disuse type is characterized as having smooth, glossy, edematous skin, pale or cyanotic skin, lymphedema, osteoporosis, atrophy of distal muscles, and spasm, dystonia and tremor, sweating.

Stage 4 lasts several months to years in the disuse type, and a few months in the causalgia type. In this stage, there may occur orthostatic hypotension, hypertension, neurodermatitis, depression, angiectasis, heart attack, loss of job and spouse and unnecessary surgery.

TREATMENT

Dr. Hooshmand suggests that the treatment of CRPS should be injection of trigger points, with possibly steroids, but preferably lidocaine, or just simply dry needling. The idea here is to decrease the repetitive input of hyperpathic pain in the original injury, and thus decrease the wind-up phenomenon that occurs at the site of the injury and at the place where the sympathetic portion of the pain feeds into the spinal cord, and to decrease the wind-up phenomenon in the spinal cord and in the spinothalamic tracts which lead to the thalamus, and to the cortex, and the cingulate gyrus, where the pain is emotionally perceived. Thus there are 3 places of wind-up phenomena, which occur if the pain is not addressed by trigger point injections and physical therapy, including deep massage, ultrasound and medications. Regular exercise is recommended.

The trigger points are described as being tender points under the skin, which are referred from the deeper portions of the injury. Thus, they are not trigger points in the sense that pressing on them causes a pain elsewhere, but in fact they are biomarkers of pain from deeper structures.

Dr. Stephen Stahl suggests that pharmacologic treatments of the CRPS include lidocaine patches or injections, nonsteroidal antiinflammatory drugs early in the treatment, tricyclic antidepressants, gabapentin and Lyrica, SNRIs, SSRIs, anticonvulsants, and novel therapeutics such as botulinum toxin, triptans, and capsaicin. The antidepressants also address the anxiety and depression component of CRPS from the cortex and from the cingulate gyrus.

Dr. Hooshmand declares that such phenomena as TMJ, migraine headache, and thoracic outlet syndrome may in fact be related to CRPS from neck injuries, and cervical nerve involvement of the sympathetic nerves. This provides a rationale for stellate ganglion block in some cases. Noninvasive block has been performed with laser or ultrasound ((Chu-De Laio et al in PLoS One 2016).

A 2016 paper by Russo and Santarelli in Pain Practice defines CRPS as microvascular dysfunction that would respond to alpha 2 adrenergic agonists, nitric oxide donors, and phosphatidic acid inhibitors. They treated 13 patients with a compounded analgesic cream of 10% ketamine, 6% pentoxyphillin, 0.2% clonidine, and DMSO (dimethyl sulfoxide) 6% to 10%, and had a response rate of 9 patients with total resolution in 2.

Dr. Hooshmand specifically inveighs against unnecessary surgery, which may worsen the CRPS. . He says that early application of nerve blocks in the hands of an experienced anesthesiologist can be of use in the early treatment of CRPS, but it should be combined with physiotherapy. He inveighs against tractotomy, rhizotomy, cingulotomy, or destructive procedures of the CNS. He does not endorse the use of spinal cord stimulators because they will fail after a few months. He says that the use of transcutaneous nerve stimulators are as good as the spinal cord stimulators, and have less complications.

Personally, I have episodically treated CRPS over more than 30 years of practice, both as a family doctor, and as a neuropsychiatrist. Invariably, the problem presented in Stage 2 or 3, with a limb that was alternately cold and blue or red and swollen, and a patient complaint of burning pain, that was increasing depression and anxiety. The recognition and characterization of CRPS has improved in the last 20+ years, but the great majority of doctors seeing it are unfamiliar with the diagnosis and treatment. I have used medications to reduce the sympathetic vasoconstriction, such as nitrol ointment to the area and catapres (clonidine) patch to the area. Oral gabapentin or Lyrica helps for neuropathic pain. If the x-ray shows osteoporosis, I have given Actonel (risedronate), a bisphosphonate, for 3 months or so. I have ordered physical therapy. As a family doctor, I did indeed do trigger point injections of procaine. These measures have stopped progression, and have alleviated or resolved the CRPS. I currently have 6 patients with CRPS, who also see pain management, neurologists, and rheumatologists.

There is a good summary of CRPS in wikipedia.org with a color picture of a foot with CRPS. Pain is a major component of many psychiatric disorders, and needs to be recognized and addressed by many more doctors than currently take responsibility.

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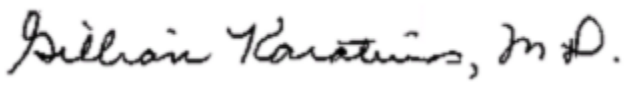

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