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Complex Regional Pain Syndrome

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Introduction

Complex regional pain syndrome (CRPS) is a chronic, centrally-mediated pain condition that is seen in many patients seeking pain management. The condition leaves patients in excruciating pain that is disproportionate to the injury. In addition, patients with this pain disorder experience a wide range of symptoms such as cold and heat allodynia, hyperalgiesia, edema, abnormal sudomotor activity and trophic changes (Caruso et al., 2015). CRPS disproportionately affects four times as many women as men (Alexander, Peterlin, Perreault, Greenough, & Schwartzman, 2012). There are two types of CRPS: type 1, often referred to as reflex sympathetic dystrophy (RSD2002), is not evident of nerve damage while type 2 does indicate nerve damage. Figure 1 provided by Marinus et al. (2011) depicts the appearance of CRPS in the acute state; figure 2 depicts the appearance of CRPS in the chronic state. The pathophysiology of CRPS remains unproven; however, many hypotheses exist due to this disorder’s multiple system dysfunction and the etiology is continuing to progress. As the pathophysiologic mechanisms of CRPS further advance, treatment modalities will continue to emerge in order for health care providers to improve the outcomes for patients suffering from CRPS.

Signs & Symptoms

CRPS may be caused spontaneously or by an injury that causes an abnormal response to tissue injury. The signs and symptoms of this multifactorial disorder include:

- severe pain
- Hyperalgiesia
- Hyperesthesia
- atypical sudomotor activity
- swelling of the affected limb
- changes in skin color
- cold and heat allodynia
- motor or tonic changes which may include weakness, tremor, dystonia, changes of the hair, skin and nails, wasting away of tissue, skin or muscle and bone thinning.

- Patients with CRPS also present with anxiety and depression when compared to healthy individuals.
- The pain is neuropathic in nature and is not limited to certain dermatomes (Alexander et al., 2014).
- The pain caused by CRPS spreads proximally over time and may even develop on the opposite limb.
- Patients with longstanding CRPS tend to perceive their affected limbs as larger than in reality and report feelings of hostility toward the limb, feeling as though the limb is a separate entity and causing the patient to desire amputation of the affected limb (Marinus et al. 2011).

Underlying Pathophysiology

Genetic association does appear to play a role in the pain-disorder. CRPS phenotypes are correlated with the human leukocyte antigen (HLA) system when HLA alleles are present at the loci (Watts & Kremer, 2013), associating CRPS with a genetic disposition. The pathophysiology of CRPS remains elusive, but with multiple hypotheses. CRPS involves interactions between the immune system and nervous system. CRPS also involves both the peripheral and central nervous systems. The immune system is crucial for the etiology of CRPS most likely begin with peripheral nociceptive overstimulation and can eventually create and sustain the central sensitization that is indicated by the sensory factors of the pain disorder (Marinus et al., 2011). The completed research on CRPS has identified three major pathophysiological pathways: abnormal inflammatory mechanisms, vasomotor dysfunction and maladaptive neuropsychophysiologic processes.

Abnormal Inflammatory Mechanisms

Alexander et al. (2012) identified significant changes in the plasma cytokines, chemokines and soluble receptors in individuals with CRPS that contribute to the inflammatory process. Cytokines most likely act not only in the affected limb, but also in the spinal cord. Neurologic inflammation is most likely the mechanism of post-junctional signaling caused by weak inactivation of neuropeptides and increased receptor availability (Marinus et al., 2011). The pro-inflammatory cytokines are liable for the initiation and sustained vasomotor and neuropathic pain and directly contribute to the extravasation of the limb, edema and increased cytokine expression in CRPS.

Vasomotor Dysfunction

Vasomotion changes and hypersensitivity have been associated with sympathetic dysfunction. In CRPS, the affected limb is initially warm due to vasoconstrictor neurons and further progresses to become cold and a blue-like color. The up-regulation of alpha-adrenoreceptors in cutaneous microvasculature has been identified as the mechanism of hypoperfusion (Watts & Kremer, 2011). Watts and Kremer (2011) report that the vasoconstrictor adrenergic receptors produce cold skin symptoms and damaged sympathetic fibers cause spontaneous pain. The result of this process is increased circulating catecholamines that vasoregulate sympathetic vasoconstriction. Neurotrophic factors of the sustained vasoconstriction may be caused in the endothelium that cause a decreased ability to release endothelial nitric oxide (Marinus et al., 2011).

Maladaptive Neuropsychophysiologic Processes

Maladaptive neuropsychophysiologic processes could be explained by evidence of structural brain changes. D. Lee et al. (2015) reported that CRPS patients revealed significantly decreased cortical thickness in the right dorsolateral prefrontal cortex, implicating CRPS is accompanied by cerebral atrophy that may contribute to the pathophysiology. In addition, Plage et al. (2014) found that CRPS patients have a decrease in gray matter density in the dorsal premotor cortex which is involved in controlling emotional correlates of pain. This evidence demonstrates that brain structure alterations in patients with CRPS are involved in regulating cognitive processes including emotional behavior and pain perception.

The central nervous system (CNS) is affected by CRPS. Although not completely understood, central sensitization may occur from lack of response to spinal cord and neuronal dysregulation of nociceptive or encephalopathic (Ding et al., 2011). Sensitization of the CNS can cause symptoms as chronic pain, hyperalgiesia, allodynia and spreading of pain to nearby non-affected areas.

Implications for Care

Treatment of CRPS involves a combination of modalities for both psychological and pain management. The condition leaves patients in excruciating pain that is disproportionate to the injury. In addition, patients with this pain disorder experience a wide range of symptoms such as cold and heat allodynia, hyperalgiesia, edema, abnormal sudomotor activity and trophic changes (Caruso et al., 2015). CRPS disproportionately affects four times as many women as men (Alexander, Peterlin, Perreault, Greenough, & Schwartzman, 2012). There are two types of CRPS: type 1, often referred to as reflex sympathetic dystrophy (RSD2002), is not evident of nerve damage while type 2 does indicate nerve damage. Figure 1 provided by Marinus et al. (2011) depicts the appearance of CRPS in the acute state; figure 2 depicts the appearance of CRPS in the chronic state. The pathophysiology of CRPS remains unproven; however, many hypotheses exist due to this disorder’s multiple system dysfunction and the etiology is continuing to progress. As the pathophysiologic mechanisms of CRPS further advance, treatment modalities will continue to emerge in order for health care providers to improve the outcomes for patients suffering from CRPS.

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Implications for Care

Treatment of CRPS involves a combination of modalities for both psychological and pain symptoms. Interferential therapies include peripheral nerve blocks, spinal cord stimulator, deep brain stimulation and chemical and surgical sympathectomy and deep brain stimulation. Interferential therapies consist of biphasic propodeal, phototheraphy, systemic, steroids, antidepressants, opioids, muscle relaxants and membrane stabilizers in order to treat the symptoms of this syndrome.