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Complex Regional Pain Syndrome

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Introduction

Complex regional pain syndrome (CRPS) is a chronic pain condition that is seen in many patients seeking pain management. The condition leaves patients in excruciating pain that is disproportionate to the inciting injury. In addition, patients with this pain disorder experience autonomic symptoms such as cold and heat allodynia, hyperesthesia, edema, abnormal sudomotor activity and trophic changes (Johannet et al., 2013). CRPS disproportionately affects four times as many women as men (Alexander, Pertuiset, Grethous, & Schwartzen, 2013). There are two types of CRPS: type 1, often referred to as reflex sympathetic dystrophy (RSD), 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 evidence is continuing to progress. As the pathophysiological 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 inciting injury that causes an abnormal response to tissue injury. The signs and symptoms of this multifactorial disorder include:

- Severe pain
- Hyperesthesia
- Edema
- Motor or trophic changes which may include weakness, tremor, distrophy, changes of the hair, skin and nails, wasting away of tissue, skin or muscle and bone thinning.

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 and HLA alleles are present at the loci (Watts & Kremer, 2011), 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 trauma usually associated in the etiology of CRPS most likely begin with peripheral nococeptive overstimulation and can eventually create and sustain the central sensitization that is indicated by the sensory factors of the pain disorder (Watts & Kremer, 2011). The completed research has identified three major pathophysiological pathways: abnormal inflammatory mechanisms, vasomotor dysfunction and maladaptive neuroplasticity.

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-junction 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 facilitation of inflammatory and neurogenic pain and directly contribute to the extracellular stress of the limb, edema and increased cytokine expression in CRPS.

Vasomotor Dysfunction

Vasomotor changes and hyperalgiesia 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 though the limb is a separate limb and causing the patient to desire amputation of the affected limb (Marinus et al., 2011).

Maladaptive Neuroplasticity

Maladaptive neuroplasticity 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, Plager et al. (2014) found that CRPS patients have an increase in gray matter density in the dorsolateral prefrontal cortex which is involved in cooling 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 retraining of spinal and trigeminal nociceptive circuits or an abnormal synaptic plasticity (Marinus et al., 2011). Sensitization of the CNS can cause symptoms such as chronic pain, hyperesthesia, allodynia and spreading of pain to non-traumatically injured areas.

Conclusion

CRPS is a painful disorder that affects more than 10,000 new cases every year in the United States (Watts & Kremer, 2011). The research continues to progress in the pathophysiology of CRPS continues to unfold. Current evidence of multiple mechanisms interconnected in the pathophysiology of CRPS should provide for additional discovery and more targeted therapeutic interventions for the future.

Implications for Care

Treatment of CRPS involves a combination of modalities for both psychologic and pain symptoms. Interventional therapies include peripheral nerve blocks, spinal cord stimulation, pump implantations, chemical and surgical sympathectomy and deep brain stimulation. Pharmacological therapies consist of bisphosphonate steroids, antidepressants, opioids, and muscle relaxants. In addition, physical therapy is used to maintain muscle strength and edema, abnormal sudomotor activity and atypical sudomotor activity. According to Watts and Kremer (2011), more than 50,000 new cases every year in the United States report experiencing CRPS syndrome. Treatment of CRPS involves a combination of modalities for both psychological and pain symptoms. Interventional therapies consist of bisphosphonate steroids, antidepressants, opioids, and muscle relaxants. In addition, physical therapy is used to maintain muscle strength and edema, abnormal sudomotor activity and atypical sudomotor activity.

Additional Sources


References


Figure 1 Acute CRPS (Marinus et al., 2011)

Figure 2 Chronic CRPS (Marinus et al., 2011)

Figure 3 Chronic CRPS (Marinus et al., 2011)

Figure 4 Acute CRPS (Marinus et al., 2011)