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Inflammation in CRPS: role of the sympathetic supply.

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Abstract

Acute Complex Regional Pain Syndrome (CRPS) is associated with signs of inflammation such as increased skin temperature, oedema, skin colour changes and pain. Pro-inflammatory cytokines (tumour necrosis factor-α (TNF-α), interleukin-2 (IL-2), IL-1beta, IL-6) are up-regulated, whereas anti-inflammatory cytokines (IL-4, IL-10) are diminished. Adaptive immunity seems to be involved in CRPS pathophysiology as many patients have autoantibodies directed against β2 adrenergic and muscarinic-2 receptors. In an animal tibial fracture model changes in the innate immune response such as up-regulation of keratinocytes are also found. Additionally, CRPS is accompanied by increased neurogenic inflammation which depends mainly on neuropeptides such as CGRP and Substance P. Besides inflammatory signs, sympathetic nervous system involvement in CRPS results in cool skin, increased sweating and sympathetically-maintained pain. The norepinephrine level is lower in the CRPS-affected than contralateral limb, but sympathetic sprouting and up-regulation of alpha-adrenoceptors may result in an adrenergic supersensitivity. The sympathetic nervous system and inflammation interact: norepinephrine influences the immune system and the production of cytokines. There is substantial evidence that this interaction contributes to the pathophysiology and clinical presentation of CRPS, but this interaction is not straightforward. How inflammation in CRPS might be exaggerated by sympathetic transmitters requires further elucidation.

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KEYWORDS: Complex regional pain syndrome; Cytokines; Inflammation; Sympathetic nervous system

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