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Clin J Pain. 2006 Mar-Apr;22(3):235-9.

Inflammatory mediators are altered in the acute phase of posttraumatic complex regional pain syndrome.

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Abstract

OBJECTIVES: Complex regional pain syndrome type 1 (CRPS 1) is a disorder that can affect an extremity after minor trauma or surgery. The pathogenesis of this syndrome is unclear. It has clinical signs of severe local inflammation as a result of an exaggerated inflammatory response, but neurogenic dysregulation also may contribute to it. **METHODS:** For further insights into the pathogenesis of CRPS 1, the authors investigated inflammatory and neurogenic mediators- C-reactive protein (CRP), interleukin-6 (IL-6), interleukin-8 (IL-8), soluble tumor necrosis factor receptor I/II (sTNFR I/II), sE-selectin, sL-selectin, sP-selectin, substance P, neuropeptide Y, and calcitonin gene-related peptide-in venous blood from both the healthy arm and the arm with acute CRPS I from 25 patients and from 30 healthy volunteers. **RESULTS:** Levels of IL-8 and sTNFR I/II were significantly elevated in patients, whereas all soluble forms of selectins were significantly suppressed. There was no significant difference in white blood cell count (WBC), CRP, and IL-6. Substance P was significantly elevated in patients. The other two neuropeptides were unchanged. None of the parameters studied showed any differences between the CRPS I-affected arm and the normal arm. **CONCLUSIONS:** Elevated IL-8 and sTNFR I/II levels indicate an association between CRPS I and an inflammatory process. Normal WBC, CRP, and IL-6 give evidence for localized inflammation. The hypothesis of neurogenic-induced inflammation mediated by neuropeptides is supported by elevated substance P levels.

PMID: 16514322 [PubMed - indexed for MEDLINE]

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