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INFLAMMATION AND PAIN IN TRIGEMINAL NEURALGIA: ROLE OF SOME PRO-INFLAMMATORY CYTOKINES

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ABSTRACT

The trigeminal ganglion transmits all the sensory information of the skull and face to the brain through afferent pathways. Afferent fibres are divided into nociceptive and non-nociceptive fibres. Primary afferent pathways express receptors for cold, heat, mechanical damage, and more. Inflammation in the head and neck can cause pain that can become neuropathic with nerve damage. An alteration of cytokine production, which may be activated by numerous potentially damaging stimuli, is involved in hyperalgesic states of several neurological diseases. In nerve injury, inflammation initiates pain, a process that is mediated by certain cytokines such as IL-1, TNF and IL-6, which cause neuropathic pain. TNF is mostly produced by mast cells and macrophages and is involved in arthritis, graft versus host disease, and other disorders, mediating inflammatory diseases and playing a key role in the neuropathic pain processes. TNF can activate other cytokines (such as IL-1) and after neurological injury it is expressed by macrophages, fibroblasts, neutrophils, and Schwann cells. In trigeminal neuralgia, TNF is linked to hyperalgesia and neuropathic pain. This shows that the inflammatory mechanisms are overlapping with the those of pain. In pain and inflammatory conditions, IL-1 is produced by many cell types, mediates hyperalgesia and neuropathy, and is involved in trigeminal neuralgia, and this effect is down-regulated by IL-1 inhibitors. IL-6 has also been reported to play a major role in the induction of neuropathic pain. In fact, in neuropathies, such as damaged nerves, IL-6 levels are increased, demonstrating that major pro-inflammatory cytokines participate in pain and inflammation. However, the pathophysiological mechanisms involved in pain and inflammation of the trigeminal nerve still need to be elucidated, as there is insufficient research on this topic. Here, in this article, we report the relationship between trigeminal neuralgia, inflammation, and pain that is mediated by pro-inflammatory cytokines.

KEYWORDS: *trigeminal neuralgia, inflammation, pain, cytokines, IL-1, IL-6, TNF, neuropathic pain*

INTRODUCTION

Trigeminal neuralgia (TN) (tic douloureux) is a specific orofacial neuropathic pain manifested by a sudden paroxysmal event on one side of the face affected by the trigeminal nerve and hyperesthesia, and most frequently affects the second and third divisions of the trigeminal nerve (1). TN is a neurovascular disease affecting the root of the trigeminal nerve, is the most common pain pathology due to compression of the cranial nerves, and is characterized by severe episodic, paroxysmal pain which is similar to an electric shock. The intensity of the pain that comes from a trigger area

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Simultaneous injection of 6-OHDA (60μg /per paw) with FCA resulted in increased paw swelling in the primary phase of arthritis, E_{Am} to noradrenaline was further depressed. A slight increase in the pD_2 value of noradrenaline was seen in inflammation, but this increase seems to be of no theoretical and practical significance.

Injection of 6-OHDA (60μg) into the paw of normal non-arthritic rats likewise resulted in a decrease of E_{Am} and an increase of pD_2 of noradrenaline in the isolated perfused hind legs up to day 5 after 6-OHDA injection. Then, E_{Am} slightly increased, but pD_2 decreased until day 12 after the 6-OHDA injection. It would be interesting whether E_{Am} will further increase due to sensitization by 6-OHDA evoked chemical denervation at later times after 6-OHDA injection. In rats injected with 6-OHDA into the same paw 5 days before FCA injection, E_{Am} increased in the isolated perfused hind leg from day 3 to day 5 of arthritis. No explanation can be given for this effect.

CONCLUSIONS

Here, we report that the injection of 6-OHDA increases paw swelling in a time-dependent manner and that adjuvant arthritis changed the vasoreactivity compared to normal non-arthritic rats (control).

Conflict of interest

The author declares that they have no conflict of interest.

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