ROLE OF CGRP-MEDIATED NEUROINFLAMMATION IN TRIGEMINAL NERVE INJURY

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Background. Calcitonin gene-related peptide (CGRP) is associated with various types of pain, with elevated levels noted in musculoskeletal pain. Trigeminal nerve injury (TNI), often resulting from facial fractures, can lead to chronic orofacial pain and neuroinflammation, contributing to atypical trigeminal neuralgia. **Aim**: CGRP is crucial in migraine and headache disorders, with elevated α-CGRP levels linked to trigeminal neuralgia, migraine, and cluster headaches. This study examined the role of CGRP in neuroinflammation following trigeminal nerve injury. **Method**: Four tissue samples from each oral quadrant (upper left, upper right, lower left, lower right) were collected from six patients post-TNI. Samples were fixed, cryosectioned, and immunohistochemically stained for CGRP. Analysis was performed using a Nikon epifluorescence microscope, recording CGRP levels and measuring fluorescence intensity in relative fluorescence units (RFU). Statistical analysis was conducted using ANOVA. **Results.** CGRP levels showed distinct patterns. In patient 1, values ranged from 67.7 RFU to 75.3 RFU in each oral quadrant. For patient 2, values ranged from 68.94 RFU to 72.45 RFU. Control values varied between 68.42 RFU and 72.7 RFU. These variations indicate differences in neuroinflammation and nerve function alterations, highlighting the differential impact of nerve injury on facial regions. Two-point discrimination tests showed thresholds of 3.1 mm for the inferior alveolar nerve and 2.8 mm for the infraorbital nerve, with statistical analysis confirming significance ($p < 0.05$). **Conclusion.** The results confirmed that CGRP plays a significant role in neuroinflammation with distinct patterns across different oral quadrants, indicating region-specific neuroinflammatory responses. These findings support the importance of targeted therapeutic strategies for managing neuropathic pain and neuroinflammation, emphasizing CGRP as a critical mediator in these processes. **Keywords**: Calcitonin gene-related peptide; Neuropathic pain; Migraine.

INFLUENCE OF SOCIAL MEDIA ON DISEASE PERCEPTION

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Background. Mass hysteria or mass psychogenic illness as termed by the rapid spread of medically unexplained symptoms within a social group. Symptoms such as fainting, headaches, and paralysis appear suddenly; this may be closely linked to the impact of social media, especially in the teenage population causing various problems such as tics, depression, etc. **Objectives:** To explore how social media influences the emergence and spread of sociogenic illness, particularly during health crises like the COVID-19 pandemic. **Methods:** Analysis of historical cases of sociogenic illness and review of recent studies examining social media's impact on public risk perception during COVID-19. **Results.** Social media plays a dual role in spreading accurate COVID-19 information and misinformation, complicating public risk perception. Real-time updates and personal stories on social media heighten emotional responses, increasing anxiety and fear. Health organizations are more trusted than individual accounts, but the overall credibility of information on social media varies widely. Social media significantly affects public behavior, including impact on health guidelines and vaccine acceptance. Instances of teenagers developing Tourette-like tics linked to TikTok videos show how social media impacts mental health. **Conclusion.** Social interactions and media consumption significantly influence mental health and behavior. Effective public health strategies must enhance social skills and ensure the credibility of information disseminated through media. Further research is needed to develop targeted interventions and communication strategies to mitigate the impact of sociogenic illness exacerbated by social media. **Keywords:** mass psychogenic illness, mental health, misinformation.