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Commentary

The Role of Neurological Diseases and Pain

Johanna Ernst*

Department of Neurology, Hannover Medical School, Hannover, Lower Saxony, Germany

DESCRIPTION

Twenty to forty percentage of patients with many basic neurological conditions experience chronic pain, which is a frequent component of many neurological disorders. These illnesses are caused by a variety of pathophysiologies, such as neurodegeneration, neuroinflammation, and traumatic injury to the central nervous system. Investigating the aetiology of pain in these illnesses offers a chance to learn new things about how pain is processed. Whether pain emanates from the central nervous system or the peripheral nervous system, it frequently becomes centralised due to maladaptive responses in the central nervous system that can significantly change brain systems and consequently behaviour (e.g. depression). Thus, chronic pain should be viewed as a brain condition in which changes to neural networks have an impact on various elements of brain chemistry, structure, and function. The lack of objective measurements for either the symptoms or the underlying mechanisms of chronic pain substantially complicates the study and management of this condition. It can be challenging to obtain even a subjective assessment of pain in cases of neurological disease-related pain, such as in individuals with end-stage Alzheimer's disease or those in a vegetative state [1]. Neurologists must participate more in the treatment and study of chronic pain (already significant in the fields of migraine and peripheral neuropathies). Greater efforts are required to improve neurologists' training in pain management and foster more interest in the field if this objective is to be met. This study explores the therapeutic promise of brain-targeted medicines, provides examples of pain in various neurological disorders, including basic neurological pain problems, and emphasizes the demand for objective assessments of pain [2].

Recent developments in basic and clinical neuroscience imply that the brain is crucial to the state of chronic pain. Neuroimaging studies have fueled recent developments in pain research, which have transformed our knowledge of how pain affects the brain. As a result, it is no longer believed that changes in the sensory systems are the primary cause of chronic pain. Rather, it is currently believed that chronic pain is a very complicated CNS condition where patterns of sensory system activation are improperly integrated with activity in other brain systems. such as the emotional, cognitive, and modulatory processes. Numerous brain regions with a variety of other functions are affected by obvious causes of pain, such as peripheral nerve injury-induced pain (neuropathic pain), including the anterior cingulate cortex, insular cortex, ventrolateral orbitofrontal area, amygdala, striatum, thalamus, hypothalamus, rostral ventromedial medulla, periaqueductal grey, pons (locus coeruleus), red nucle More recently, doctors and scientists have come to the opinion that chronic pain frequently results directly from neurological disorders or may even be regarded as a component of the underlying disease. Parkinson's disease, where 40–60% of patients describe chronic discomfort, may be the best illustration of this. The latter interaction is intricate; for instance, both pain and despair can trigger one another. Many chronic neurological illnesses have a significant rate of co-occurring depression [3].

A well-established cause of neuropathic pain is injury to the CNS or peripheral nervous system. It may be possible to understand how the brain processes pain in chronic disease by taking into account the changed patterns of brain activity in neurological diseases with pain. Some neurological illnesses are linked to reduced pain or no pain, in contrast to the many neurological diseases with accompanying pain sensations (i.e. congenital insensitivity to pain). Examining the underlying pathology and regional changes in brain systems that underlie some of these disorders may help to understand how changes to the central nervous system result in chronic pain. In this article, we define chronic pain as a brain disorder based on major abnormalities in architecture, chemistry, and function that accompany pathophysiological changes in pain pathways. These 'brain-wide' changes take place in regions of the brain associated with sensory, affective, and modulatory systems. These alterations are a direct result of the pain or result from comorbid alterations like sadness or anxiety [4-6].

The altered brain activity that underlies chronic pain may alter central pathways, causing pain to appear even when there is no peripheral cause. "Centralization" of pain, here defined as "the

Correspondence to: Johanna Ernst, Department of Neurology, Hannover Medical School, Hannover, Lower Saxony, Germany, E-mail: Johanna@ernst.de

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persistent static or dynamic brain functional state that contributes to or causes the behavioral responses to pain (e.g., depression increased sensitivity to stimuli, ongoing pain)," occurs as a result of altered brain dynamics in a variety of brain systems, including emotional, cognitive, and motor systems in addition to specific sensory systems. Regardless of whether the primary beginning mechanism is in the CNS as a result of primary brain illness, the peripheral nervous system (potentially including muscle), or subsequent to afferent input such a result of nerve or spinal cord damage, this altered state leads to a cognitive, sensory, and emotional experience of pain.

REFERENCES

1. David N, Ohgaki H, Otmar D, Webster K. Cavenee. The 2007 WHO classification of tumours of the central nervous system. Acta Neuropathol. 2007;114 (2): 97-109.
2. Kesari S, Ramakrishna N, Sauvageot C. Targeted molecular therapy of malignant gliomas. Curr Neurol Neurosci Rep. 2006; 5:186-97.
3. Huang, Paul H, Alexander M, Forest M. Oncogenic EGFR signaling networks in glioma. Sc signal. 2009; 87(2):re6.
4. Cameron W, Verhaak GW, McKenna A, Campos B, Noushmehr H, Salama SR, et al. The somatic genomic landscape of glioblastoma. Cell. 2013; 155(2):462-477.
5. Jenkins R, Blair H, Ballman K. A t(1;19)(q10;p10) mediates the combined deletions of 1p and 19q and predicts a better prognosis of patients with oligodendroglioma. Cancer Res. 2006; 66:9852-61.
6. Reifenberger J, Reifenberger G, Liu L. Molecular genetic analysis of oligodendroglial tumors shows preferential allelic deletions on 19q and 1p. Am J Pathol. 1994;145:1175-90.