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REVIEW ARTICLE

What is Emotional Pain? A Review of Pathophysiology and Treatment Options

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ABSTRACT

Pain is a dynamic process that involves multiple physiological systems for its perception and outcomes. The basic pain process involves a complex neurological process and biochemical changes. Emotional pain is an extended form of the already known pain wherein the stimuli are emotional in nature involving abstract feelings, for example, losing a loved one. Currently, there is growing evidence that like pain, emotional pain also involves inflammatory processes and that the behavioral approach is directly associated with them. This association can help us modify emotional pain by modulating the inflammatory processes that are already known. In addition to therapeutic and counseling interventions, mindfulness-, writing-, and exposure-based interventions can help in not only treating the condition but also exploring other avenues of the pain physiology. Emotional pain has always been a part of human history but one of the least discussed forms of pain. Future neuropsychiatric studies may help in further investigating the process of emotional pain.

INTRODUCTION

Pain is a perception formed by the cognitive part of the human brain. The brain computes the chemical changes in the body or receives a stimulus, which if harmful, is perceived as pain. Irrespective of whether the danger or fear is real and whether it is really harmful, it is perceived as damaging, and hence, we experience pain. To understand the mechanism of emotional pain, it is essential to define and understand pain as a whole. Pain is a multidimensional term used to label a range of situations we face, from physical trauma from an accident to the inevitable suffering of human life.

We acknowledge the different situations that cause us physical pain because there is evidence to support its origin; therefore, finding a cure to physical pain is easy. However, the scars of emotional pain are well hidden deep inside ourselves, and they are tougher to find and hence cure. The symptoms of emotional pain can be cured, but as long as the stimulus is present, the feeling does not go away; therefore, emotional pain needs much more assessment on the part of the physician and patient than physical pain. Although physical pain, e.g., pain in your leg, might stop you from walking around, the crippling pain of losing a loved one or not getting that job you worked really hard for might deprive you of the capacity to perform even the day-to-day activities to sustain life. The brain is not hardwired when it comes to pain perception; many systems, humoral and cognitive, come into play whenever we perceive pain (1). There is enough evidence to support the fact that psychological stress or emotional pain is connected to and predisposes us to persistent physical pain. The incidence of childhood adversities and early-life conflicts is associated with various pain conditions later in life, e.g., migraine headaches (2), interstitial cystitis or painful bladder, pelvic pain, irritable bowel syndrome, and fibromyalgia (3-7). Therefore, it can be safely considered that nerve-racking circumstances can not only exacerbate the pain but also trigger it.

In the following discussion, we review the biochemical and neurological aspects of pain perception. To make our understanding of emotional pain more comprehensive and to ensure better outcomes and prognosis of emotional pain, we have discussed it in comparison with physical pain.

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PATHOPHYSIOLOGY AND BIOCHEMISTRY PROFILE

Pain is a complex phenomenon comprising a combination of multiple, intricate neurological and biochemical changes in the brain. No specific cortical area in the brain is assigned to pain perception and processing as opposed to that for other basic human senses, such as hearing and vision. For more comprehensively understanding emotional pain, we have compared the biochemical changes that occur when there is a physical pain stimulus (e.g., physical blow and trauma) with those that occur when there is an emotional pain stimulus (e.g., bullying and losing a loved one).

For the purpose of this discussion, lets focus on the theory stating that pain, irrespective of its source or character, originates from inflammatory responses (8). All types of pain, whether they are sharp, dull, aching, stabbing, burning, or tingling in character, originate from inflammation and the inflammatory response. The biochemical mediators of inflammation include cytokines, neuropeptides, growth factors, and neurotransmitters.

In the event of physical pain/trauma, numerous pro-inflammatory and anti-inflammatory mediators that are released have been identified. In some studies, higher levels of C-reactive protein, tumor necrosis factor-alpha (TNF-α), interleukin (IL)-2, IL-6, IL-8, IL-10, and CD40 ligand were found to be associated with greater pain (9,10).

There is growing evidence to support the theory that inflammatory mediators of pain play a crucial role in the pathophysiology of depression and suicidal behavior, thereby supporting the initial hypothesis that postulates an overlap between physical and emotional pain.

There is substantial evidence supporting the association between suicidal behavior and disruption in the levels of IL-2, IL-6, IL-8, IL-10, C-reactive protein (CRP), and TNF-α. During stress or inflammation, substance P, IL-1, and IL-6 levels increase. IL-6, in turn, induces the release of corticotrophin-releasing factor, which results in elevated systemic levels of corticosteroids. Furthermore, chronic psychological stress states have been associated with increased levels of IL-10 and suppression of immune system by the activation of lymphocyte apoptosis in mice, leading to increase susceptibility to infections and opening up more channels of emotional suffering (11-15).

Irrespective of the origin or characteristic of pain, the biochemical changes occurring after the pain stimulus is similar in both emotional and physical pain. This association can help us understand the nature of emotional pain and methods to overcome it.

NEUROPHYSIOLOGY OF PAIN

As specified previously, there is no specific area of brain responsible for pain perception. Imaging studies have shown extensive networks of neurons connecting various parts of the brain during pain perception, including activation of the contralateral anterior cingulate and primary and secondary somatosensory cortices along with areas of the forebrain primarily responsible for controlling emotions (16). The same stimulus also activates various pathways related to memory, cognition, and pain response in the brain, thus involving more areas in the cerebral cortex than previously imagined (17).

Several studies have established numerous classes of pain or nociceptive fibers, such as type Aδ and C fibers, which arise from all over the body. Aδ fibers respond to fluctuations in temperature and to mechanical stimuli; they are further classified into type I or IIA fibers. Type C fibers are slow mechano-heat receptors (18). When nociceptors are activated, the impulses travel along these nociceptive fibers into the dorsal horn of the spinal cord. Further processing of this stimulus is diverse and complex, involving the spinohypothalamic, contralateral spinothalamic, spinoreticular, and spinomesencephalic tracts, leading to the medulla, hypothalamus, and brainstem depending on the type, position, and frequency of stimulation of the respective nociceptor (19,20). These fibers then ultimately activate the primary and secondary somatosensory cortices, cingulate gyrus, and various parts of the forebrain.

Studies have shown association between expectations of impending pain with pain perception (21). The discovery of pre-emptive activity in the medial orbitofrontal cortex (OFC), prefrontal and frontal cortices, and ventral striatum has established the importance of the OFC in the modulation of pain perception. In one study, analysis of the data obtained using fMRI indicated considerable activity in the periaqueductal gray and OFC during pain anticipation; however, during the actual stimulation, the anterior insula, primary and secondary somatosensory cortices, dorsolateral and ventrolateral prefrontal cortices, and OFC showed activity. The involvement of amygdala through parabrachio-amygdaloid pathway was also revealed (22,23).

The above discussion indicates that the prefrontal cortex plays a major role in pain perception and modulation. The prefrontal cortex has been shown to be associated with emotional pain and stress (24). Disturbance in the dorsal and ventral parts of the prefrontal cortex is associated with depression and emotional traumatic states. Acute and recurrent trauma/stress has also been shown to result in functional and structural changes in the prefrontal cortex and amygdala (25). Chronic restraint stress and chronic immobilization causes dendritic shortening in the medial prefrontal cortex (26,30). Other studies have shown that stress leads to the dendritic development of neurons in the amygdala and OFC (31,32). Furthermore, social rejection and alienation leads to the activation of the anterior cingulate gyrus, which is also activated in the event of physical pain inflicted on the subject (33).

Thus, from the above review of pain pathways, it can be concluded that emotional pain activates similar areas in the brain as those activated by nociceptors (34). Although pain and emotions have the ability to modulate and influence each other through various pathways in the brain, there is evidence of a neurocognitive overlap between emotional distress and physical pain.

TREATMENT

It is known that both emotional stability and physical well-being are essential to lead a content and fulfilling life; the

struggle to reach that point is something we are all familiar with. The biggest hurdle in this path leading to happiness seems to be our oblivion to the emotional side of pain. Although an X-ray will provide a definite diagnosis in case of a broken bone, there is no such diagnostic test in the matters of a broken heart. Our ignorance or lack of awareness about our thoughts, sensations, and emotions make the matters worse internally. Although we reassure ourselves that there is nothing wrong with us, a huge part of us is in distress due to our unawareness and insensibility.

Various pharmacological agents have been used for treating physical pain. Inability to effectively treat pain has been shown to increase disability, depression, loss of income, unemployment, and mortality (35-38). Various guidelines have been established to counter the physical pain experienced by people; however, guidelines to counter emotional pain remain uncertain (39-41).

One study has reported that psychological effects play a huge role in pain perception and management (42). Psychological assessment results in better management of patients, thereby improving the quality of life of the patients, improving compliance to the treatment, decreasing stay at the medical facility, and improving prognosis. This emphasizes the importance of emotional pain management (43).

Various interventions have been tested for combating emotional pain, including mindfulness and acceptance therapies, emotional disclosure, and emotional exposure-based interventions.

Mindfulness

Mindfulness techniques have been used to enhance psychological wellbeing and equilibrium and increase pain tolerance (44,45). Mindfulness is often practiced in the mindfulness-based stress reduction (MBSR) program, which has shown mixed results. Another method called the loving-kindness meditation program, derived from the Buddhist tradition of loving and transforming anger, has shown good prognosis in the management of back pain, further supporting the overlap between emotional and physical pain (46). Acceptance and commitment therapy, derived from the mindfulness therapy, has also shown to have promising effects on pain management and improvements in the quality of life (47,48).

Writing Therapy

Reportedly, writing out or talking about our feelings and emotions regarding any significant event and visualizing it for a series of sessions with a clinician helps in understanding the pain and consequently reaching a state of acceptance (49-51). This acceptance helps in achieving emotional stability.

Exposure-based Intervention

This therapy exposes the patients with histories of psychosocial trauma to emotional exposure and processing techniques with help from a clinician. This therapy identifies the stimuli and practices that each patient avoids or is traumatized by and uses exposure-based techniques (e.g., written disclosure, imaginal desensitization, empty chair techniques, assertiveness training, and in vivo exposure) to help patients confront and process the avoided emotional experiences. This therapy has been shown to be helpful in patients who have post-traumatic stress disorder or have received childhood abuse or sexual abuse (52).

Other Therapies

We all have various ways to cope with stress and emotional pain, which are less complex than the ones stated above.

Take a deep breath! This is what we hear most of the times when we express our distress to anyone. Breathing techniques have been shown to normalize stress response, emotions, and autonomic and neuroendocrine system function (53).

Do Yoga! Yoga has shown considerable effects on the stress mediators in the brain. It improves spiritual wellbeing and general wellbeing of the patient in stress (54,55).

Work it out! The anti-depressive effect of exercise has long been established (56-58). Physical activity of various types have been shown to uplift the mood and help overcome emotional pain.

CONCLUSION

Emotional pain is a type of pain that has existed from the advent of human race. The knowledge of its association with inflammatory processes can help improve its diagnosis and treatment. An intensive neuropsychiatric interventional study may help to understand the emotional pain process and to develop methods for managing it. Many simple treatment methods, in addition to the traditional therapeutic options, can be used to help manage emotional pain.

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AUTHOR CONTRIBUTIONS

All authors contributed in this study equally.

CONFLICT OF INTERESTS

None.

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