

Commentary

Is There Hope for Chronic Pain Patients?

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Abstract

Many websites are dedicated to educating the public about chronic pain and its management. The majority of these websites say there is no cure for chronic pain, but encourage patients to try exercise, diet, stress reduction and other techniques to decrease the severity of chronic pain. Patients are also taught to use oral or injected drugs to treat their chronic pain. This can be dangerous and leads to adverse events and death in some patients. The majority of these websites teach patients that chronic pain comes from the brain and brain stem. This teaching is incorrect, since chronic pain is generated in the skin and can best be treated and cured with topical medicines.

Keywords

Chronic pain; skin; chemokines



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1. Introduction

Education of patients and their families is an essential aspect of healthcare. Websites are developed with the purpose of public education about healthcare issues, including chronic pain [1-8]. These websites are developed by the Cleveland Clinic, Johns Hopkins University, the National Institutes of Health and other healthcare institutions. How useful are these websites? It is not known if chronic pain patients refer to these websites. Should patients trust these websites? The opinions expressed represent a consensus opinion. The result of this consensus opinion is that patients may be dissuaded from trying safe alternatives.

Chronic pain should be separated from long term pain clearly associated with a disease process such as arthritis, cancer, gout, diabetic neuropathy and other diseases. Treatment of these diseases is the important issue and can decrease or even cure long term pain, such as cancer pain. There are currently, three types of pain: nociceptive, neuropathic, and nociplastic pain [9]. Nociceptive pain is caused by acute tissue damage and results in bradykinin release that activates transient receptor potential cation channels in the skin. Nociplastic pain is caused by nerve sensitization due to ongoing tissue damage and inflammation, such as diabetic neuropathy. The ongoing disease process results in skin ischemia that damages sensory neurons. This activates transient receptor potential cation channels and other receptors. Neuropathic pain is caused by nerve damage [9]. One of the characteristics of damaged sensory neurons is that they regrow. As the new growth reattaches into the skin, paresthetic impulses are generated which may involve transient receptor potential cation channel activation.

In the current paper, chronic pain is defined as pain that has no obvious cause, such as nociplastic and neuropathic pain. It may have resulted from an automobile accident, a traumatic event, physical abuse, a severe injury, infection or other causes, but should have disappeared when the injury healed. Instead, the pain lingers and can last for years or even the remainder of the patient's life. This pain may also be called psychogenic or psychosomatic pain.

2. Pain Mechanisms

Pain is due to nociceptors found in the skin, such as transient receptor potential cation channels, that can be activated by bradykinin released from damaged keratinocytes [10]. The sensation of pain in the skin generates a signal that is relayed to the brain stem and processed in the brain. The brain itself, has no nociceptors and cannot sense pain. Of course, opioid receptors are found in the brain, but are not nociceptors.

Chronic pain is generated in the skin by the pain chemokine cycle [11]. A lingering activation of transient receptor potential cation channels in the skin causes the release of chemokines in the skin that attract macrophages and neutrophils [12, 13]. These cells release prostaglandins and leukotrienes that increase and prolong pain. IL-17 is released by skin T cells in response to prostaglandins [14]. Sensory neurons release more chemokines due to IL-17 stimulation [14]. This results in a vicious cycle of more pain and more chemokines that is the source of chronic pain [11, 15, 16]. Activated macrophages and neutrophils penetrate into the brain, increase chemokine production in the brain, and may be involved in central sensitization [15, 16]. Chronic pain can be treated with topical preparations in order to stop the pain chemokine cycle.

3. Pain Treatments

A number of oral and injected medicines are used against chronic pain, can decrease chronic pain, but do not cure chronic pain [17]. Long term use of opioids or nonsteroidal anti-inflammatory drugs results in the induction of chemokines that make chronic pain worse [17]. These dangerous drugs kill about 150,000 people yearly due to respiratory depression, seizures, ulcers, heart attacks and strokes [17].

Alternative therapies can decrease chronic pain. Heat therapy and cold therapy can be helpful since they inhibit transient receptor potential cation channel activation [10]. Acupuncture, osteopathic manipulation and other manipulations can decrease but not cure chronic pain [18]. Stress reduction techniques or anxiolytic agents can decrease but not cure chronic pain [18]. Benzodiazepines are anxiolytic drugs that must be used carefully since they are addictive. Several topical medicines are available for pain and chronic pain, that contain methylsalicylate, menthol and camphor. These topical preparations are useful in the treatment of pain, but have not been reported to cure chronic pain [17, 18].

Moderate, daily exercise for 20-60 minutes can be effective against chronic pain, including fibromyalgia [19, 20]. Walking, running, bicycling, swimming and other exercises can decrease chemokine production and either cure or diminish chronic pain [19, 21]. Blood endorphin levels are increased in chronic pain [22], and can be increased even more with exercise. Endorphins may be involved in modulating chronic pain. Exercise is also an effective and safe treatment for depression, a cause of chronic pain [23]. Running does not cause arthritis, a long term painful condition, but actually decreases the progression of arthritis [24]. Exercise also results in the release of endocannabinoids that can decrease pain [25]. Endocannabinoids inhibit transient receptor potential cation channels in the skin [10]. Endorphins activate opioid receptors in the skin and other areas to decrease pain [10].

An alternative approach that is reported to cure chronic pain is called retraining the brain [3]. Central sensitization may respond to emotions and thought processes such that retraining the brain may decrease central sensitization. One very effective way to retrain the brain is by physical exercise.

California Indians teach us how to cure chronic pain [26]. A liniment made from *Artemisia californica* or a foot soak made from *Salvia mellifera* are very effective against pain and cure chronic pain [26]. These preparations contain many monoterpenoids that inhibit transient receptor potential cation channels. Diterpenoids are present that inhibit IL-17 and chemokine production. These preparations are topical which allows the medicines to be applied directly to where they are needed. The medicinal compounds penetrate into the skin, then evaporate from the skin resulting in pain relief without systemic toxicity. The author has used these preparations to help many patients cure themselves of chronic pain including fibromyalgia, whiplash, chronic back pain, bursitis and chronic pains that were misdiagnosed as ankylosing spondylitis, systemic lupus erythematosus, polymyalgia rheumatica and other conditions [26, 27]. The liniment is preferred by patients instead of a placebo [28].

The liniment is very effective against headaches and migraine headaches [26, 28]. Headaches are not generated in the brain. They actually are generated in the skin of the head [29]. This may involve calcitonin gene related peptide receptors on sensory neurons surrounding arterioles of the skin.

The combined use of exercise and topical plant medicines may be the best treatment and cure for chronic pain. Clinical trials should be helpful in finding the best approach to curing chronic pain. So far, these clinical trials have not been approved [30].

Author Contributions

The author did all the research work of this study.

Competing Interests

The author has declared that no competing interests exist.

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