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The mechanisms of joint and muscle pain

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CLINICAL PROBLEM

A 34-year-old woman complained of experiencing persistent pain in the left side of her jaw over the previous two years. The pain was constant, and the patient rated its severity as a 7 on a pain rating scale 0 of 10. The pain was located around the temporomandibular joint (TMJ), masseter and temporalis muscles, and angle of the mandible. The patient reported that this pain was exacerbated when she moved or used her jaw. Whenever the skin around the jaw angle was touched gently, a strong, burning pain was elicited that lasted for minutes. The patient also experienced significant pain in her shoulders and neck, and she revealed that she had been feeling tired and depressed. Over-the-counter pain medication and oral splints did not relieve the pain. One year previous, she had undergone TMJ surgery on the left side of her jaw that involved the placement of a total TMJ prosthesis. We wanted to determine which pain mechanisms were involved in this patient's case.

EXPLANATION

Woolf and colleagues[1] described four basic types of pain mechanisms (Table). Nociceptive, or transient, pain may be the easiest type to understand, though even the simple processing of pain is complex. The nociceptor is the essential receptor on primary afferent nerve fibers that innervate the orofacial tissues, including the TMJ and jaw muscles.[2] A range of different transducing receptors and ion channels are located on the primary afferent nerve endings, which detect and respond to specific high-intensity stimuli (for example, thermal heat, cold, mechanical and chemical stimuli) that may be associated with tissue damage (activation). These sophisticated sensors in the TMJ and jaw muscles can be activated during high-intensity stimulation and strenuous tasks (for example, excessive jaw-opening or hard clenching) and, thereby, serve as a warning system against tissue damage. The pain, however, disappears when the stimulus stops. Owing to the persistent nature of the pain in the patient whose case we report, nociceptive pain did not seem to be the only pain mechanism involved.

Tissue damage due to trauma, infections or surgical procedures commonly is associated with inflammatory pain.[2] Advances have been made in terms of understanding the neurobiological changes in the nociceptive system that occur in response to inflammatory conditions. For example, the nociceptors can begin to trigger spontaneous activity that can lead to pain without the presence of a peripheral stimulus. Another feature is sensitization that has lowered nociceptor activation thresholds and increased neuronal responses (modulation).[3] In

addition, silent nociceptors can be awakened and contribute further to pain. There also is evidence that functional shifts occur in the number and activity of receptors and ion channels on the nociceptor, activating receptors for nerve growth factor, bradykinin and prostaglandins, thereby increasing membrane excitability. Second-order neurons in the trigeminal sensory nucleus complex react to the increased trafficking of action potentials from the nociceptor, and sensitization of the neurons in the central nervous system occurs.[4] Although the phenomena of peripheral and central sensitization can develop within minutes, these processes usually are reversible in inflammatory pain conditions, which respond well to nonsteroidal anti-inflammatory drugs. Gross inflammatory changes rarely are present in jaw muscles, whereas osteoarthritis is associated with inflammatory processes in the TMJ.[5] In the case we report in which the patient had received a TMJ prosthesis, we attempted to rule out inflammatory reactions around the implant by using imaging techniques such as computed tomography and magnetic resonance imaging.

Neuropathic pain may follow damage to peripheral nerve fibers during surgery (for example, implantation of a TMJ prosthesis) or be caused by disease such as postherpetic neuralgia or diabetic neuropathy. Neuropathic pain also may develop after the central somatosensory system is injured (for example, by stroke, multiple sclerosis and spinal cord injuries). The consequences of the development of these nerve lesions include hypersensitivity to painful stimuli and to normally nonpainful stimuli (allodynia),[2] as well as to spontaneous pain in which the primary afferent nerve fiber can initiate electrical discharges due to ectopic neural activity near the peripheral nerve lesion. Phenotypic changes and alterations in the expression and distribution of ion channels can occur, which contribute to an increase in membrane excitability (modification). The central nervous system often plays a significant role in these situations. In the case we report, we observed changes in somatosensory function (that is, burning pain outlasting the duration of the touch stimulus), which indicated that neuropathic pain mechanisms could be involved.

TABLE

Putative pain mechanisms.*

PAIN MECHANISM	BASIC MECHANISMS	CLINICAL MANIFESTATIONS
Nociceptive	Activation (nociceptor specialization)	Pain linked to stimulus
Inflammatory	Activation Modulation (peripheral and central sensitization) Modification (structural changes)	Localized pain hypersensitivity (for example, heat hyperalgesia)
Neuropathic	Modification (structural changes) Activation Modulation (central sensitization)	Localized pain hypersensitivity (for example, allodynia often combined with aftersensations, paroxysms and sensory loss)
Functional	Central amplification	Generalized hypervigilance

* Source: Woolf and colleagues.[1]

Functional pain is poorly understood. No peripheral tissue pathology can be detected, but it is believed that an abnormal amplification and processing of peripheral stimuli occur in the central parts of the somatosensory system (hypervigilance). The exact mechanisms are not known, but they may involve genetic and environmental interactions.[6] Fibromyalgia, chronic neck and shoulder pain, irritable bowel syndrome and tension-type headaches are examples of this type of disorder. In contrast to inflammatory and neuropathic types of pain in which hypersensitivity to painful stimuli occurs within a specific nerve innervation territory, functional types of pain are characterized by widespread or generalized hypersensitivity. Clinicians should conduct a comprehensive pain analysis, including careful elucidation of psychosocial aspects, to assess the somatosensory function in the painful and nonpainful parts of the body.[2,5] Simple tests of somatosensory function can be performed easily in the dental office, and more elaborate tests (for example, quantitative sensory tests) often can be performed at university clinics. In the case we report, the concomitant pain conditions and depressive symptoms alerted us to consider the involvement of more functional pain mechanisms.

CLINICAL IMPLICATIONS

For the patient in the case we report, we needed to take a complete history, perform a careful clinical examination, obtain TMJ images and conduct a neurosensory examination.[2] We found that a prominent part of the patient's complaint of pain

ABBREVIATION KEY. TMJ: Temporomandibular joint.

in the TMJ could have been due to neuropathic pain after the TMJ surgery, and functional pain mechanisms could have contributed to the muscle pain. In cases such as this one, invasive procedures should be avoided, and palliative management including pharmacotherapy and cognitive-behavioral therapy should be initiated.[5]

CONCLUSION

Clinicians should try to identify the underlying mechanisms of pain in the orofacial region for all of their patients. Pain in the jaw joint and muscles is not always due to simple nociceptive pain mechanisms but may involve inflammatory, neuropathic and functional components. While it may be more common to see nociceptive and inflammatory types of pain in the TMJ region, clinicians should consider the possibility of other types of pain in this region, because it will influence the management strategy as well as the eventual outcome. ■

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