CASE REPORT

Dental restoration induced orofacial pain and its management

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ABSTRACT

Dental procedure induced pain may develop into a chronic condition that accompanied with functional or neuropathy changes in the nerve system. In this case, severe persistent pain gradually developed after repeatedly placing a subgingival amalgam restoration in the right second molar. Hyperalgesia and allodynia were present at the affected region. A provisional diagnosis of chronic orofacial pain with peripheral and central sensitization was considered. After re-contouring, local debridement and occlusal adjustment the pain disappeared. The underlying mechanism in this case is neuronal sensitization and peripheral Aβ-fiber mechanoreceptor activation. Its diagnosis and management depend on identification and treatment of the cause for pain generation and sensitization.

Key words
Allodynia, central sensitization, dental restoration, neuropathic pain, orofacial pain

INTRODUCTION

Pain is a common symptom of patients receiving dental procedure. Some of them may develop into chronic orofacial pain that does not respond to customary care. Due to its complexity and unclear pathogenic mechanisms,[1,2] chronic orofacial pain is often difficult for management.[3] The key to successful treatment is to identify the specific mechanism involved in generating the pain.[4,5] However, in most cases, multiple mechanisms are involved in the development and maintenance of chronic pain.[6]

One mechanism for the development of chronic pain is peripheral deafferentation. Partial or total loss of afferent nerves supplying a particular area may induce the pathogenesis of neuropathic pain.[7,8] The sensitization theory proposes that neuronal plasticity occurs in the sensory nerve system that enhances the sensitivity to noxious stimuli or even change the innocuous slight touching to pain.[9] It is believed that the pain signaling itself and the accompanied releasing of proinflammatory cytokines, neuronal transmitters and modulators participate in these plastic changes in sensory neuronal circuits.[10] Therefore, any diagnostic or treatment procedure, which might cause a neuropathic lesion and possible increase in pain, should be carefully evaluated and prevented.[11] If a dental procedure is absolutely necessary, administration of a local anesthetic, even under general anesthesia, should be routinely applied. Preemptive analgesia should also include the use of preoperative and postoperative analgesics in addition to local anesthetic.[12]

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CASE REPORT

A 48-year-old Caucasian female presented with orofacial pain at the Division of General Dentistry, Eastman Institute for Oral Health at the University of Rochester Medical Center. The chief complaint was severe pain in the right upper jaw that has lasted for 1.5 years. The patient described the pain as persistent with brief, episodic sharp attacks evoked by touching and/or eating. The pain quality, according to the patient, was constant, achy and deep in her right maxillary posterior jaw. This pain radiated to her head, neck and shoulder. The pain was severe and sensitive to touch. The patient reported that she could not chew on the affected side and sometimes she had to stop eating or not eat at all.

The pain developed gradually after placement of a lingual amalgam restoration on the right upper second molar, which was the abutment for a 3-unit bridge (#2-4). The filling came out twice and was replaced. Each placement

caused excruciating pain. The patient sought care and each time she was told the restoration looks fine, and her dentist could not provide an explanation for the pain. OTC analgesics and tramadol had minimal effect on the pain.

Intra-oral examination was unremarkable except the presence of a slight swollen and dark-redness area of the marginal gingiva on the lingual of #2. Periodontal examination revealed the presence of 4-5 mm pockets with no bleeding on probing. A rough subgingival restoration surface and local gingivitis (periodontitis) on the lingual of #2 was present. Extra-oral examination was negative except palpation pain at the right middle face. There was asymmetry to the lower face, and the chin was slightly shifted to the left. The patient denied the presence of TMD. Panoramic and periapical (PA) radiographs demonstrated no obvious abnormalities and pathological changes in the TMJ, teeth, periodontium and bone. There was no sensitivity to hot or cold and no sinus related problems. There was no tooth clenching and/or grinding.

Even though there was no direct evidence for the diagnosis of neuropathic pain, somatosensory neuronal sensitization was considered. To support this hypothesis, a sensory functional test was performed. Cranial nerve examination revealed the presence of hyperalgesia and allodynia in the area of #2.[13,14] A local infiltration analgesia (lidocaine 2.5% with 1:100K epinephrine) significantly reduced the background pain, hyperalgesia and allodynia. These results suggest the presence of the jagged restoration, local inflammation (gingivitis/periodontitis) as the cause of the pain. The underlying mechanism might be related to peripheral lesion (deafferentation) and neuronal sensitization induced by repeated invasive dental procedures and local inflammation.[15] There maybe dysfunctional synaptic and neural circuit plasticity changes in the central nervous system (central sensitization).[9] However, nerve lesions and primary neuropathy in the peripheral and central nervous system also needs to be considered as a differential diagnosis.[16]

CLINICAL MANAGEMENT AND RESULT

At the first visit, initial examination and sensory functional test were performed. Periapical (PA) and panoramic X-ray were ordered. Ultimately, a simple local irrigation with normal saline was performed. The patient reported no significant improvement in her pain. The provisional diagnosis of chronic orofacial pain with neuronal sensitization was established. Figure 1 shows the palatal gingiva of the right posterior maxilla. At the second visit, under local anesthesia (lidocaine, 2.5%, epinephrine 1:100K), the amalgam restoration in #2 was re-contoured, the local periodontium was debrided with a dental scaler, a through irrigation with chlorhexidine and normal saline was performed. Post-operative analgesic (ibuprofen) was also prescribed.

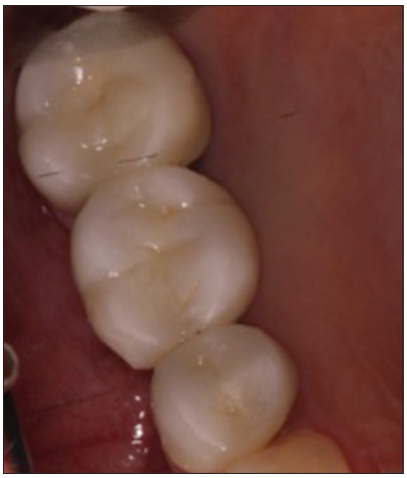

Figure 1: Intra-oral picture shows the view of #2 and the palatal gingiva

The patient noted that she had not experienced a severe pain the next day. She experienced some discomfort chewing harder foods on her right side, but the intense pain was gone. At the third visit, occlusal adjustment was performed in #2 and the patient reported that she could chew all foods on her right side, something she hadn't been able to do for quite a long time. Figure 2 shows the X-ray before and after re-contouring of the amalgam restoration.

Four weeks later, the patient reported mild tenderness in the affected tooth with a score of 2-3 (0-10 visional analogue scale). The patient had slight pain when flossing. The gingival appearance appeared largely normal, and the pocket depth remained unchanged. Further examination revealed that the existence of slight rough site at the mesial portion of the restoration. Thus, the decision to perform a second debridement was made.

At the fifth visit, additional re-contouring at the mesial site of the restoration, local gingiva debridement and irrigation were performed. Five days later, she reported the presence of biting pain. "It seems when I bite on certain food in a certain way there is pain. Also, flossing the gum feels slightly sore, but not the same as before." She was advised to avoid eating on the affected side.

At the follow-up sixth visit, No pain was evoked with biting. Further examination showed no sign of tooth fracture. Slight occlusal adjustment was performed before dismissing the patient. One week later, the patient reported no further pain from the gingiva, and biting problem was improved. Twelve months after the treatments, the patient reported no further problem and pain.

DISCUSSION

In this case, the pain developed after repeated placement of a dental restoration. A jagged amalgam restoration and the local infection/inflammation may have further contributed to the development and persistence of

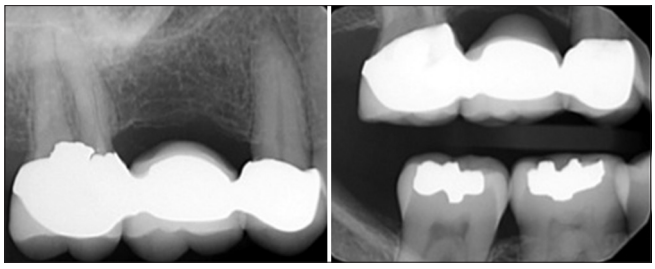

Figure 2: Periapical and bitewing X-ray shows the jagged amalgam restoration before and after recountering

the long-term severe pain. Pitcher and Henry have demonstrated that the nociceptive signal reaching the second-order neurons can induce central sensitization.[17]

In orofacial trigeminal neuropathies, such as atypical odontalgia, patients, usually, display normal tooth and tissues. Some patients have undergone unnecessary dental procedures, such as root canal treatment, apicoectomy, and tooth extraction.[18] After these procedures, pain may still exist or even worsen. Therefore, a careful examination and illness history are important for the correct diagnosis and efficient management of orofacial pain. Successful treatment also depends on a carefully designed treatment plan. It is recommended that any unnecessary invasive procedure should be avoided. In this case, the pain was managed effectively by approaches targeting its etiological factors (jagged filling) with local anesthesia and postoperative analgesics.

The underlying mechanism for neuropathic pain still remains elusive. One hypothesis proposed for the development of neuropathic pain is peripheral deafferentation. It is believed that partial or total loss of afferent nerves supplying a particular area is responsible for the pathogenesis of neuropathic pain. In these patients, damaged nerve terminals may not regenerate or recovery properly, which will induce persistent pain.[7,8] The sensitization theory proposes that neuronal plasticity occurs in the nociceptive pathway will significantly increase the sensitivity to noxious or innocuous stimulus.[9] These plastic changes include mechanisms involving suppression of inhibition, potentiation of presynaptic release and postsynaptic excitability.[19-21] The activated microglia and astrocytes also contribute to the maintenance of central sensitization.[22,23] The pain signaling itself and the accompanied releasing of proinflammatory cytokines (IL-1 β, tumor necrosis factor-α, IL-6, NO, PGs), neuronal transmitters and modulators induce these plastic changes in neuronal circuits.[10]

Since the pain that persisted in this patient was not responsive to NSAID, it suggests that local inflammation was no longer a major player in the chronic pain condition. Interestingly, as the jagged restoration was removed, significant pain relief was noted. This strongly suggests the mechanical stimulation contributed to the pain generation and maintenance. It has been suggested that the spinal dorsal horn nociceptive sensory (NS) neurons undergo a phenotypic switch by acquiring characteristics of wide dynamic range neurons and become responsive to innocuous stimuli such as a slight touch.[9,24-26] Recently, it has been observed that the spinal nociceptive network contains a silent circuit between low threshold Aβ primary afferents and NS projection neurons.[9] This circuit, which contains excitatory interneurons expressing PKC-γ is normally inactive because of receiving input from inhibitory glycine interneurons, but is activated under sensitization and as such, "turns touch into pain."[27,28] Therefore, the mechanical stimulation produced by jagged restoration may activate the Aβ-fibers and then induce the severe pain.

The management of this case demonstrates that reducing the Aβ-fiber mechanical stimulation and preventing PKC-γ neuron activation would provide efficient therapy for peripheral stimulus-dependent neuropathic pain. Recognition and identification of the peripheral stimulus-dependent orofacial pain will help to improve the paradigm for its diagnosis and management in dental clinic.

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