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<td>Author(s)</td>
<td>Handa, T; Fukuda, K; Ichinohe, T</td>
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<td>Journal</td>
<td>Bulletin of Tokyo Dental College, 54(3): 171-175</td>
</tr>
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<td>URL</td>
<td><a href="http://hdl.handle.net/10130/3735">http://hdl.handle.net/10130/3735</a></td>
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Effect of Combination of Trigger Point Injection and Stellate Ganglion Block on Non-odontogenic Mandibular Molar Pain Referred from Masseter Muscle: A Case Report

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Received 20 February, 2013/Accepted for publication 15 April, 2013

Abstract

We report a case of myofascial pain syndrome (MPS), manifested as nonodontogenic mandibular molar pain referred from the masseter muscle, relieved by a combination of trigger point injection (TPI) and stellate ganglion block (SGB). The patient was a 32-year-old woman who had experienced cold hypersensitivity in the right third mandibular molar 2 months prior to visiting our department. Subsequently, she had visited a family dentist and undergone pulpectomy under local anesthesia. She eventually visited our clinic because there was no marked change in her symptoms. On the first visit, no tooth abnormality was found and the patient was neither anxious nor depressive. Tender points were found in the right masseter and temporal muscles during muscle palpation. Referred pain radiating to the right mandibular molars was observed when pressure was applied to the central portion of the right masseter muscle. As a result, we diagnosed MPS based on evidence of nonodontogenic tooth pain caused by referred pain from the masseter muscle. We performed TPI with 2% lidocaine hydrochloride to the tender point in the masseter muscle. Although the visual analog scale (VAS) pain score dropped from 97 to 36, complete pain relief was not achieved. The TPI was effective for approximately 7 hrs, after which severe throbbing pain returned. The sustained nature of the tooth pain suggested that it was sympathetic nerve-dependent. Subsequently, we performed SGB, resulting in a reduction in the VAS pain score from 90 to 32. Therefore, we performed another TPI and the VAS pain score dropped to 0. We continued SGB and TPI for the next 3 days and the symptoms disappeared. Thus, a combination of TPI and SGB controlled MPS manifested as masseter muscle-mediated nonodontogenic tooth pain.

Key words: Myofascial pain syndrome — Referred pain — Trigger point injection — Stellate ganglion block
Introduction

Myofascial pain syndrome (MPS) occurring in the head and neck region often manifests as referred pain in the teeth, and is sometimes too severe to be alleviated. Although MPS is believed to be related to sympathetic nerve activation, to our knowledge, no studies to date have investigated the effectiveness of stellate ganglion block (SGB) for MPS-related tooth pain. Here, we report a case of severe mandibular molar pain caused by referred pain from the masseter muscle in MPS that was relieved using a combination of trigger point injection (TPI) and SGB.

Case

This case report was approved by the Tokyo Dental College Human Research Ethics Board and informed consent to publish obtained from the patient. The patient was a 32-year-old woman. She had visited another hospital complaining of dull pain and cold hypersensitivity in the right third mandibular molar 2 months prior to presenting at our department. Subsequently, she had undergone pulpectomy under local anesthesia. Although the cold hypersensitivity was resolved, the dull pain persisted around the right third mandibular molar. Although the patient subsequently underwent repetitive root canal treatment, these symptoms persisted, even after administration of oral loxoprofen sodium hydrate. One week prior to visiting our clinic, the dull pain became severe and the patient was unable to sleep. The patient also experienced hypoesthesia, mainly in the right corner of the oral cavity. Although the patient’s family doctor advised a wait-and-see approach, the patient decided to visit our clinic as there was no change in her symptoms.

On her first visit to our clinic, X-ray imaging revealed no specific findings. Percussion examination also revealed no abnormal findings in the first, second, or third mandibular molars. Mouth opening distance was >30 mm, and there was no clicking sound when the mouth was opened and closed. The patient gave the following answers to the McGill Pain Questionnaire: throbbing, pricking, pressing, tingling, pulling, taut, numb, splitting, pounding, fearful, and unbearable. The visual analog scale (VAS) pain score was 90; the pain started upon rising in the morning and was most severe at approximately 8 pm. The pain disturbed the patient when she was going to sleep, and she woke repeatedly during sleep. The State-Trait Anxiety Inventory (STAI) and Hospital Anxiety and Depression (HAD) scales were used to assess psychological factors. On STAI, the score was 30 for trait anxiety (level II: moderately low) and 38 for state anxiety (level III: average). In contrast, on HAD, the score was 2 for anxiety and 1 for depression. This indicated that the patient was neither anxious nor depressive. The Semmes-Weinstein test around the corner of the oral cavity yielded a score of 1.65–3.65, indicating mild hypoesthesia. Palpation of the masticatory muscles showed markedly tender points at the right masseter and temporal muscles accompanied by a taut band at these locations. The patient exhibited the jump sign when pressure was applied to the central portion of the right masseter muscle, and there was a myofascial trigger point (TP) that caused referred pain, radiating to the right second and third mandibular molars. Although we attempted a regional block using 2% lidocaine hydrochloride at the second and third mandibular molars, there was no change in the severity of the pain. We suspected that the tooth pain was caused by referred pain due to MPS and therefore performed TPI with 2% lidocaine hydrochloride at a dose of 1 ml into the masseter TP. The VAS score for the tooth pain dropped from 97 to 36. Complete pain relief, however, was not achieved. The next day, the patient visited our clinic again, still complaining of severe throbbing pain. The TPI administered the previous day had been effective for approximately 7 hrs. We speculated that the patient might have hyperalgesia due to sympathetic nerve hyperactivity because the tooth pain...
Effect of Combination of TPI and SGB

had lasted for >1 week. Therefore, we injected SGB at the transverse process of the 6th left cervical vertebra using 2% lidocaine hydrochloride at a dose of 5 ml. The VAS score for tooth pain subsequently dropped from 90 to 32. We then performed another TPI at the masseter tender point, and the VAS score dropped from 32 to 0. Because a combination of masseter TPI and SGB achieved complete pain relief, we diagnosed MPS manifest as referred pain from the masseter muscle, complicated by sympathetic nerve-dependent pain. We continued masseter TPI and SGB over the following 3 days and the symptoms of both pain and hypoesthesia disappeared.

Discussion

It has been reported that the etiology of tooth pain in 3% of all patients is nonodontogenic and that in 9% of all patients it is mixed in nature. Tooth pain of myofascial origin is the most common among patients with nonodontogenic pain. Another study reported that 11% of patients with MPS experienced nonodontogenic tooth pain. Studies suggest that the main masticatory muscles involved are the masseter and temporal muscles. Observation of pain relief after TPI is useful for diagnostic examination. Therefore, in patients showing tooth

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Fig. 1 Trigger point injection for the masseter muscle

Fig. 2 Stellate ganglion block
pain with no abnormalities in the tooth or periodontal tissues, detailed examination of the head and neck muscles is key in achieving a diagnosis.

In tooth pain of myofascial origin, referred pain arises from TPs formed in fatigued muscles, as described in the classic theory of myofascial pain by Travell and Simmons\(^9\). Although Mascia et al. reported that this type of tooth pain disappeared when TPI was administered\(^5\), only a few articles have provided scientific evidence for this method\(^10\). In routine clinical practice, however, TPI is undoubtedly a useful method for the diagnosis and treatment of myofascial pain.

Cord-like indurations and localized tenderness, a localized twitch response, and a jump sign were observed in the present case. These symptoms were partly relieved by TPI, and the addition of SGB resulted in complete pain relief. Travell and Simmons\(^9\) reported that the origin of TPs might lie in the sympathetic nervous system. Although the mechanism remains to be elucidated, it may be related to a failure of peripheral blood flow and pain-producing substances such as nor-epinephrine. Hubbard and Berkoff\(^2\) recorded spontaneous electrical activity in myofascial TPs in patients with temporomandibular pain and observed muscle spindles in biopsies from these sites. This was attributable to intrafusal muscle fibers because the electrical activity was eliminated using the sympathetic blocker phentolamine. Furthermore, the involvement of the sympathetic nerves is important in the development of MPS. Fibrous C polymodal nociceptors distributed densely in the muscle can form a tender point with ischemia, leading to the accumulation of endogenous pain-producing substances and stimulation of the sympathetic nervous system, thereby establishing a vicious cycle of reflexes\(^4\). The present results suggest that the sympathetic nerves are always involved where there is myofascial pain. This case demonstrates the effectiveness of SGB for pain that cannot be completely resolved with TPI.

Perry et al.\(^6\) reported that MPS was caused not only by excessive movement, but also by sympathetic nerve hyperactivity due to emotional stress such as anxiety or nervousness. In this case, the patient did not complain of daily or social activities that caused emotional stress, and the results of the HAD and STAI psychological tests provided no evidence supporting the involvement of emotional factors. Therefore, it seems unlikely that emotional factors were involved in the pain reported by this patient.

Referred pain from the masticatory muscles in MPS is one of the most common presentations in a clinical setting. However, referred pain is often overlooked, leading to pointless repetition of cycles of dental treatment. In the present case, referred pain from the masseter muscle caused sustained mandibular molar pain, resulting in sympathetic nerve hyperactivity and severe pain. There are various theories on the mechanism underlying referred pain. This report reconfirms that in cases of sustained pain, sympathetic nerve activation could amplify symptoms, and that SGB in combination with TPI can be effective in resolving such symptoms.

References


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