Case Report

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Postherpetic Neuralgia Aggravated by Upper Complete Denture

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Postherpetic neuralgia (PHN) is a chronic and refractory pain disease. It requires long-term treatment and follow-up. Comorbid diseases can change or aggravate the pain condition and responsiveness of patients to PHN treatment. In such cases, the cause of pain should be identified through proper testing, and appropriate treatment should be administered. Herein, we report the case of a 67-year-old man with PHN in the maxillary nerve. As the pain was being controlled with medication and infraorbital nerve block, the patient experienced deterioration of pain caused by a newly worn upper complete denture. The patient's pain was relieved following correction of the upper complete denture. (Ewha Med J 2018;41(4):82-85)

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Key Words

Denture, complete; Neuralgia, postherpetic; Trigeminal nerve

Introduction

Postherpetic neuralgia (PHN) is a chronic and refractory pain disease characterized by neuropathic pain that persists even after successful herpes zoster (HZ) treatment. Treatments for PHN include pharmacological modalities, including anticonvulsants and antidepressants, as well as non-pharmacological modalities such as block of the infiltrated nerve, nerve stimulation, spinal cord simulation, and intrathecal drug administration. However, PHN currently has no definitive cure; instead, the goal of treatment is to alleviate symptoms.

Patients with PHN may exhibit pain as a symptom of nerve irritation or damage to the infiltrated area. They may also experience allodynia due to mechanical stimulation or temperature change, as well as hypoesthesia and paresthesia. However, the same symptoms can appear as a result of other diseases that cause neuropathic pain. Therefore, when PHN does not respond to treatment, when the response to treatment changes, or when

the pain pattern changes worsens, various tests must be performed, including medical history taking and physical examination, to exclude secondary causes other than PHN.

In the present report, we describe a case of PHN that had been kept under control using pharmacological therapy and nerve block, but deteriorated after the patient began wearing an upper complete denture. The symptoms were relieved following correction of the upper complete denture. We report the details of this case, along with a literature review.

Case

A 67-year-old man was admitted for a chief complaint of pain on the right upper lip. Medical history taking revealed that, 2 months earlier, he had received treatment at another hospital for HZ, which had been diagnosed on the basis of vesicular lesions and a piercing pain in the area near the maxillary nerve which branch of trigeminal nerve. However, despite treatment,

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formication on the upper lip and tingling pain persisted. Thus, the patient was transferred to the pain clinic at our hospital.

His medical history indicated that he was taking medication for hypertension. He also had hearing and speech impairment, so he was accompanied by a sign language interpreter when he came to our hospital.

Examination of the patient showed a scar on his upper lip that had been caused by the HZ, and he indicated his pain level to be about 4-5/10 on a numerical rating scale (NRS). He was diagnosed with PHN and treated with gabapentin (800 mg/ day) and tramadol (100 mg/day). In addition, an infraorbital nerve block and local infiltration were performed on the site of pain using 3 mL of 0.1% ropivacaine and 2 mg of dexamethasone. During a follow-up visit 7 days later, his pain score had improved to 1-2/10 on the NRS. Infraorbital nerve block was then repeated every week three more times, and the same dose of gabapentin and tramadol were administered each time. Two months after the start of the treatment, his pain and paresthesia had dissipated almost completely. Accordingly, we planned to taper the dose of medication and subsequently discontinue it completely. The dose of gabapentin was decreased to 200 mg/ day over 2 weeks. However, the patient experienced a sudden aggravation of pain characterized by throbbing and tingling corresponding to 5-6/10 on the NRS. We suggested that the pain aggravation had been caused by the decreased drug dose, so we increased the dose again and performed an infraorbital nerve block. However, even though the dose of gabapentin had been increased to 800 mg/day, the pain did not subside, in contrast to the initial treatment response. Thus, the gabapentin was replaced with pregabalin, and the patient was treated with infraorbital nerve block and local infiltration using local anesthesia at the site of pain. The dose of pregabalin was subsequently increased to 300 mg/day, but the pain could not be controlled.

Medical history taking and physical examination were performed again in response to this sudden aggravation of pain. It transpired that the pain showed a pattern, becoming aggravated in the morning and subsiding when going to bed. Therefore, an association with the patient's upper complete denture was identified—the pain had become aggravated when the patient had begun wearing the denture. When the patient wore the denture, the pain in his upper lip area became aggravated; when he took off the denture at bedtime, the pain was alleviated. When the upper complete denture was removed in the examination room,

his pain level decreased immediately to 1–2/10 on the NRS. Accordingly, we determined that the patient's pain was being caused by the denture, and a correction of the right upper portion of the denture was performed in the department of dentistry. Subsequently, the patient reported no more pain associated with wearing the denture. At the time this report was written, the patient's medications for PHN were being maintained at a decreased dose, and his pain was controlled at 1–2/10 on the NRS.

Discussion

PHN is a chronic and refractory pain disease accompanied by paresthesia and neuropathic pain, including deep burning pain, spasmodic shooting pain, and allodynia along the skin segments of infiltrated nerves. This pain persists even after the skin lesions caused by HZ have completely healed. Although the pathogenesis of pain in PHN is still uncertain, it involves a combination of various mechanisms [1]. Treatment for PHN may involve pharmacological modalities and various interventions, including nerve block and nerve stimulation. However, many patients show poor treatment response or adverse effects that make it difficult to continue treatment, so complete pain relief is difficult to achieve. For this reason, the treatment goal is to alleviate pain and reduce other symptoms, such as depression, insomnia, and functional impairment. In this regard, clinicians must work together with the patient to establish an appropriate treatment level.

HZ is difficult to diagnose until skin lesions appear. In some cases, back pain and radiating leg pain caused by early-stage HZ are mistaken for a herniated disc or spinal stenosis, which can lead to delays in antiviral therapy or inappropriate surgery [2,3]. Similarly, PHN must be differentiated from heart disease, pleuritic chest pain, acute and subacute abdominal disease, herniated disc, neuropathic pain (intercostal neuralgia and trigeminal neuralgia), and cervical myalgia. In this regard, differential diagnosis is based on the site of pain that precedes the skin lesions

Conversely, PHN can be more easily diagnosed based on pain that persists even after HZ has been successfully treated. However, since PHN requires long-term treatment with the goal of alleviating symptoms, comorbidities that can affect the PHN site may be neglected during the treatment. Several studies have

reported inappropriate treatment or delays in proper treatment in patients with spinal cord tumor, vertebral compression fracture, or scarring from HZ who were misdiagnosed as having PHN-related pain only [4-6]. When pain does not dissipate with PHN treatment, when there is a sudden aggravation of pain that was under control, or when the site or pattern of pain changes, alternate causes of pain should be considered.

In the present case, the patient was admitted for a chief complaint of persistent pain and paresthesia despite treatment for HZ. Considering the site and pattern of pain, and because the pain had persisted for over 2 months, pharmacological treatment with anticonvulsants and antidepressants was initiated alongside infraorbital nerve block. The symptoms of pain and paresthesia subsequently dissipated. Thus, the patient's dose of the medication was reduced. However, the symptoms became exacerbated again during follow-up. The aggravated pain showed a pattern similar to the existing PHN and was believed to be due to the dose reduction. However, symptoms failed to improve, even after the drug dose was increased. Through thorough inquiry and physical examination, an association between the pain aggravation and the patient's newly worn upper complete denture was discovered.

The site of PHN in this patient was the maxillary nerve, which is the second branch of the trigeminal nerve and is a sensory nerve that passes through the foramen rotundum and exits from the pterygopalatine fossa. Some branches pass through the pterygopalatine ganglion and are distributed in the oral cavity, nasal cavity, and pharynx, while others pass through the inferior orbital fissure to become the infraorbital nerve. These then exit into the face from the infraorbital foramen to be distributed throughout the upper lip, lower eyelid, and external nose. The middle superior alveolar nerve and anterior superior alveolar nerve, which are responsible for sensation in the upper gum, branch as they pass through the infraorbital canal.

Upper complete dentures are affixed to the palatal surface and gum, generating pressure on the palatal surface and thus potentially affecting the nasopalatine nerve and greater and lesser palatine nerves. Meanwhile, contact with the gum can compress the superior alveolar nerve. The main complications associated with complete dentures are foreign body sensation and damage or inflammation of the gum; the denture may also cause symptoms of intraoral vascular or nerve compression [7]. In particular, nerve compression caused by a denture can lead

to nerve entrapment syndrome, as well as burning mouth syndrome, which is characterized by a burning sensation or tingling pain in the oral mucosa [8]. In the present case, we believe that the patient's new upper complete denture caused aggravation of pain by compressing the superior alveolar nerve, which is the peripheral branch of the maxillary nerve. The patient's symptoms improved after correction of the lateral incisors and canines in denture. It follows that compression of the superior alveolar nerve was the main cause of pain. Moreover, through inquiry, we identified that the patient's pain became aggravated when he was fitted with the upper complete denture, and that his symptoms changed when the denture was removed. Thus, we could have found the cause of aggravation easily and administered proper treatment through correction of the denture. However, the pain was instead attributed to the dose reduction of PHN medication, resulting in unnecessary medication and nerve block.

PHN that requires long-term treatment, and exacerbation can be caused by secondary comorbidities, as well as by nerve irritation or compression by structures like dentures. Therefore, when patients show no or a reduced response to PHN treatment, or when existing pain patterns change or become aggravated, detailed inquiry and physical examination must be carried out to check for secondary causes, such as PHN exacerbation, or secondary comorbidities. Such an approach will ensure proper treatment.

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