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Case Report

Intrathecal Morphine Infusion for Trigeminal Deafferentation Pain Following Percutaneous Intervention for Unexplained Facial Pain: A Case Report

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ABSTRACT

Chronic pain in painful post-traumatic trigeminal neuropathy, formerly called trigeminal deafferentation pain (TDP) or anesthesia dolorosa, is virtually incurable neuropathic pain. In severe cases, no effective method has yet been established. A 58-year-old woman presented with chronic dysesthetic pain in the right side of her face that had persisted for 8 years. It was caused by percutaneous balloon compression for an unexplained, persistent right gingival pain. The TDP did not respond to any medications or radiosurgery. Considering the typical occipital neuralgia that occurred later, the incomprehensible gum pain was interpreted as referred trigeminal pain from occipital neuralgia. Decompression of the greater occipital nerve improved occipital neuralgia; however, TDP did not respond to internal neurolysis or invasive brain stimulation. The last attempt was made to administer an intrathecal opioid because of pain sufficiently severe to cause suicidal ideation. Trial administration of intrathecal opioids had some effect on pain relief. Although incomplete, the effects of intrathecal morphine infusion were maintained up to 1 year later. Invasive neurosurgical interventions should be cautiously performed for continuous pain in persistent idiopathic facial pain and referred facial pain cases that do not show typical neuralgic pain in primary trigeminal neuralgia because of the risk of TDP.

Keywords: Greater occipital nerve; Morphine; Neuropathic pain; Occipital neuralgia; Persistent idiopathic facial pain; Trigeminal nerve

INTRODUCTION

Deafferentation pain of the trigeminal nerve is one of the most difficult to treat neuropathic pain. In contrast to the idiopathic nature of primary trigeminal neuralgia, there is an individual subset with persistent facial pain with an apparent history of trigeminal system injury.[3] Because there was no exact classification for this trigeminal neuropathic pain other than the term anesthesia dorolosa, Burchiel[3] divided chronic neuropathic pain caused by trauma to the trigeminal nerve into trigeminal neuropathic pain (TNP) and trigeminal deafferentation pain (TDP), according to presence of intentional damage. TDP refers the neuropathic pain resulting from injury to the nerve by trigeminal nerve ablation, gangliolysis,

or rhizotomy in an intentional attempt to treat either trigeminal neuralgia or other facial pain.[3] However, TDP is currently classified as 13.1.2.3 painful post-traumatic trigeminal neuropathy in the third edition of the International Classification of Headache Disorders.[8] Despite better understanding of the underlying mechanisms and the development of proven analgesic therapies, an unfortunate minority of patients are still refractory to all available therapies.

A case of chronic medically refractory pain of painful post-traumatic trigeminal neuropathy responding to intrathecal morphine infusion is presented. Her hemifacial pain developed following percutaneous balloon compression (PBC) for treatment of chronic upper molar pain that is not typical of idiopathic trigeminal neuralgia. Continuous dysesthetic pain in her right hemiface did not respond to invasive pain surgeries including, radiosurgery, internal neurolysis, and trial of thalamic deep brain and motor cortex stimulations. The last attempt intrathecal morphine infusion reduced her pain by about half. TDP is the most feared complication of neuroablative treatment. Neuroablative treatment for trigeminal neuralgia should only be performed under an accurate diagnosis of idiopathic trigeminal neuralgia. Its application to continuous trigeminal pain should be very cautious.

CASE REPORT

A 58-year-old female patient presented with chronic persistent pain in the right face that had lasted for 8 years. Eight years ago, dull aching pain occurred suddenly in the lower and upper gums. It was located around the first molar and lasted all day (**FIGURE 1A**). It was not induced by eating, brushing teeth, or washing face and was not controlled by medication. Her dentist tried to extract the upper first molar tooth, but the pain did not go away. Due to uncontrolled gum pain, she was referred to a department of Oral Medicine at a dental university hospital. Even after a year of continuous drug treatment, her pain did not subside. She said that the exact diagnosis for her pain was not specified at the dental clinic. Two years after the onset of gum pain, she was diagnosed with trigeminal neuralgia at a hospital and underwent percutaneous balloon compression to control her symptoms.

Severe dysesthetic pain developed immediately after PBC. Hypesthesia occurred in the right V2 and V3 regions, and in the mouth and tongue (**FIGURE 1B**). Pain like a lump and tightness continued to occur in the affected area. There was also pain in the right eye and periorbital area, and tears continued to flow when the pain was severe. This new pain was not controlled by anticonvulsants such as carbamazepine, gabapentin, or pregabalin, and persisted at an intensity of 7–8/10 with Numeric Rating Scale (NRS)-11. The right masticatory function was also weakened. About 3 months after PBC, a momentary stabbing pain occurred gradually and repeatedly in the right occipital region, which was accompanied by right facial pain and continued along with the right facial pain (**FIGURE 1C**). Since there were no other treatment options, she continued to take the medication, waiting for the pain to improve over time. Anesthetic blocks for the trigeminal nerve branch were performed several times, but there was no effect.

Two years after the onset of post-PBC dysesthetic pain, she visited another university hospital and was recommended a radiosurgery. Radiosurgery had no effect on her pain. Fortunately, no further exacerbations occurred after radiosurgery. Due to years of anticonvulsant medications, she suffered from several digestive side effects. If her pain occasionally worsened intolerably, she had to be hospitalized several times for painkillers injections.

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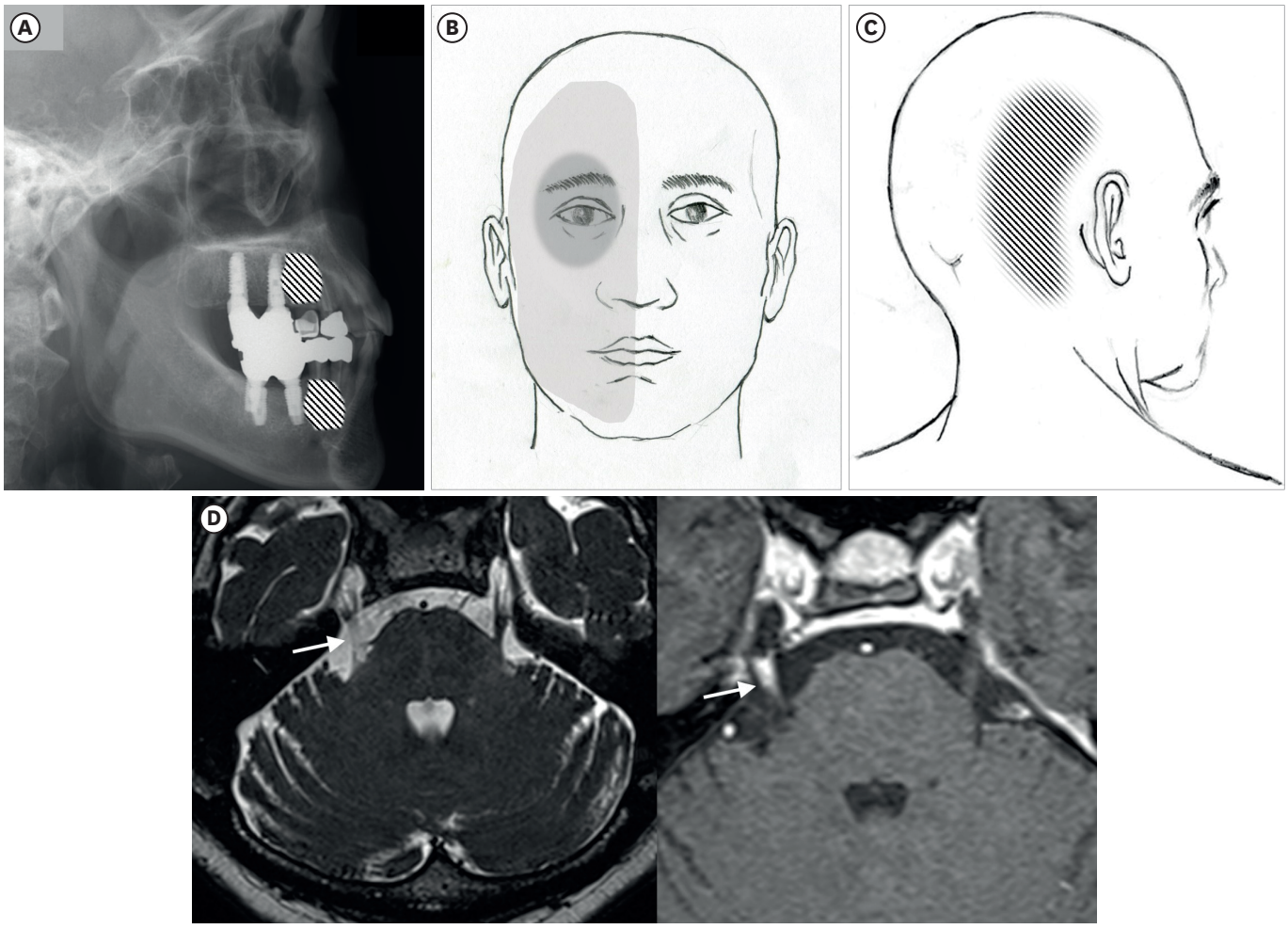

FIGURE 1. Diagrams demonstrating the distribution of right facial pain.
(A) Location of right gum pain (hatched area, shown on X-ray). (B) Distribution of dysesthetic, deafferentation pain in the right hemiface (gray area), especially severe in the periorbital area, after percutaneous balloon compression. (C) Occurrence of right occipital pain (obliquely hatched area) over the right suboccipital and occipital area. (D) An axial T2-weighted MRI image (left) of the right trigeminal nerve (arrow) showing severe atrophy compared to the left. The right trigeminal nerve on axial T1 MRI image showing contrast enhancement.
MRI: magnetic resonance imaging.

Five years after dysesthetic pain, she visited the author's outpatient clinic due to unbearable facial pain. She complained of recurrent and persistent unpleasant heaviness and tightness in the face and mouth. At the same time, it was reported that there was repeated aching and tightening in her right occipital region. On physical examination, hypesthesia to light touch and coldness was found in her right V1, V2, and V3 dermatomes. No allodynia was present. The right masticatory strength was normal. Mild tenderness was observed in the right suboccipital trapezial canal area. Chronic dysesthetic pain that occurred after PBC 6 years ago was still present in the V1, V2, and V3 dermatomes, the oral mucosa of the right cheek, and the tongue. Severity of her pain and anguish was rated on an NRS-11 score of 7–8/10, and she said she couldn't stand it any longer. A sophisticated magnetic resonance imaging (MRI) scan showed marked atrophy of the right trigeminal nerve with mild gadolinium enhancement (**FIGURE 1D**).

Because her pain was TDP that occurred after an invasive intervention for unexplained trigeminal gingival pain, she was diagnosed with painful post-traumatic neuropathic pain of trigeminal neuropathy (formerly anesthesia). Her occipital pain was found to be

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temporarily ameliorated for 6 hours by occipital nerve block (2 mL of 1% lidocaine), which was interpreted as chronic entrapment of the greater occipital nerve (GON). Although there is no evidence-based treatment for anesthesia dorolosa, trigeminal rhizotomy/or internal neurolysis and GON decompression were recommended first, considering the severity and chronicity of pain. Informed consent was obtained after explaining the possibility of surgical failure and explaining that failure may require additional neuromodulatory treatment.

Internal neurolysis of the trigeminal nerve and GON decompression were performed in the lateral position using two separate incisions using a conventional lateral suboccipital approach (**FIGURE 2A**). After dural and arachnoidal opening, the trigeminal nerve and its root exit zone (REZ) were addressed. As expected, the right trigeminal nerve showed significant atrophy and had a pale yellowish green color (**FIGURE 2B**). Internal neurolysis was performed through fascicular dissection without rhizotomy of the trigeminal nerve root. After closure of the lateral suboccipital wound, an oblique paramedian approach to the trapezial canal for GON decompression was performed. Severe entrapment of the GON by the aponeurotic edge of the trapezius muscle was confirmed and careful decompression of the GON was performed under microscopic vision (**FIGURE 2C**). Postoperative course was uneventful. After internal neurolysis, the pain in the right forehead decreased slightly, but the pain in the right eye, cheek, jaw, and mouth did not change. The aching, tightening pain in the right occipital area disappeared with GON decompression. A mild paresthesia in the right occipital region lasted for a month and then subsided. Although the pain in the back of the head improved,

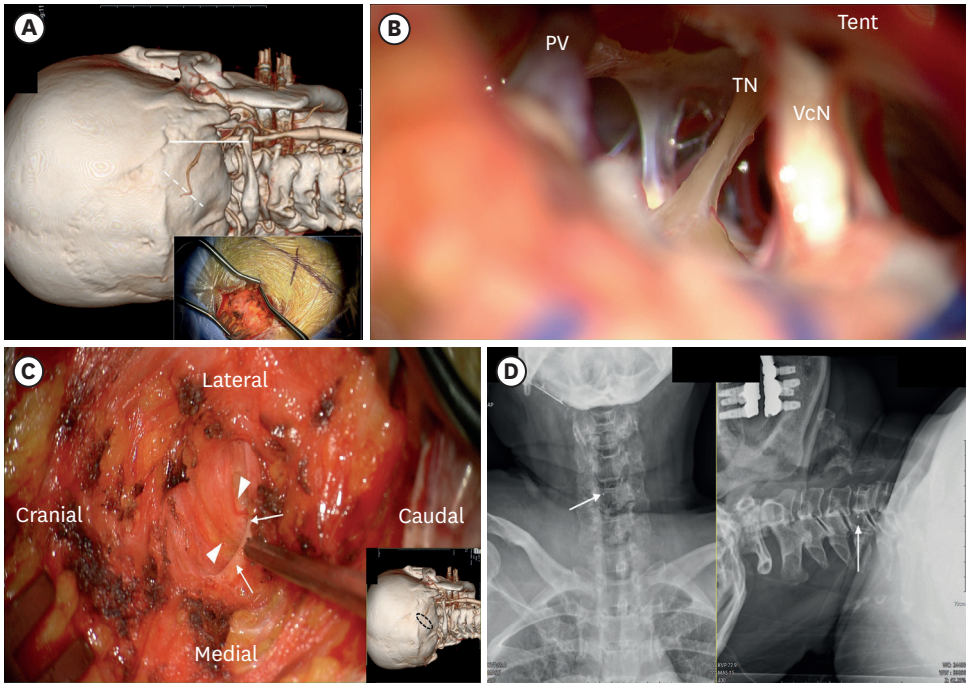

FIGURE 2. Intraoperative photographs during decompression of the right GON and internal neurolysis of the trigeminal nerve.
(A) Diagram showing the location of the oblique paramedian incision for GON decompression and the suboccipital incision for internal neurolysis. The lower right inset is an intraoperative photography showing the location of the incisions during the actual operation. (B) An intraoperative photograph showing the atrophic trigeminal nerve. The right trigeminal nerve was severely thinned and discolored yellow. (C) An intraoperative photography showing severe entrapment of the GON (white arrowheads) by the fibrous aponeurotic edge of the trapezius (white arrows). The lower right inset shows the location of incision for the GON decompression. (D) The anterior-posterior (left) and lateral (right) X-rays showing the position of the catheter tip (arrows) at C5 vertebral body level.
GON: greater occipital nerve, PV: petrosal vein, Tent: tentorium, TN: trigeminal nerve, VcN: vestibulocochlear nerve.

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the pain in the right face did not improve, so her pain and suffering were the same as before surgery despite large amount of opioid analgesics and neuropathic pain medications.

After waiting for delayed pain relief for 6 months, it was confirmed that there was no improvement. Trial stimulation electrodes for the motor cortex stimulation (MCS) and deep-thalamic brain stimulation (DBS) were implanted with pre-planned neuromodulation options. The mechanical effect of thalamic DBS electrode implantation resulted in slight pain relief, but thalamic stimulation did not induce paresthesia in her face, and the pain was exacerbated by a sharp electric sensation at high stimulation intensity. The pain relief effect of motor cortex stimulation could not be obtained. Because there was no significant pain relief during the one-week test stimulation period, the extension cable to the scalp was cut and she was discharged for follow-up.

Three months after discharge, she complained that she could no longer endure the chronic excruciating pain and anguish. The pain forced her to sleep on medication and was unable to carry out her daily activities. When she could not sleep at night, she had to go to several hospital emergency rooms for painkillers injections. Six months later, she complained that the pain was so severe that she had frequent suicidal thoughts. As another option, intraventricular or intrathecal morphine injection was explained to the patient and family and informed consent was obtained.

A 0.25 mg of morphine sulfate was injected via lumbar puncture. A significant reduction in her chronic dysesthetic pain was observed for about 6 hours. No side effects were observed after intrathecal morphine injection. Confirming a positive response to the morphine injection, and 0.5 mg of morphine sulfate was re-injected the next day via lumbar puncture. The pain relief effect of about 60% by the patient's evaluation lasted for 12 hours, and the pain was reduced until the next morning. She reported that the tears that flowed spontaneously when the pain was severe were no longer sustained. For the first time in 8 years, she started to smile. The analgesic effect of intrathecal morphine was confirmed, and implantation of intrathecal drug delivery system was performed. The tip of the catheter was positioned at the level of the C5 vertebral body in consideration of orofacial pain (**FIGURE 2D**). A drug pump (Synchromed II®; Medtronic, Minneapolis, MN, USA) was implanted on the left abdomen. No side effects were observed due to drug pump implantation and chronic infusion.

One year after chronic morphine infusion, she rated her pain as 5–6/10 on NRS-11. She reported that although she was still suffering from pain, she was able to live with and endure simple daily activities without suicidal thoughts. The dosage of morphine sulfate was initially 0.5 mg per day, but adjustment was required every 3 months. In the first year of surgery, 0.6 mg infusion per day and 0.25 mg booster infusion 4 times a day were required.

DISCUSSION

Etiology of unexplained persistent idiopathic facial pain (PIFP)

The cause of persistent gum pain in the current case, which first occurred 8 years ago, was not clearly identified even after 2 years of treatment at several dentists and oral clinics. Tooth extraction did not affect pain at all and there was no abnormality in temporomandibular joint and intraoral structures. Therefore, it can be classified as 13.12 PIFP according to the ICHD-3.[8] PIFP is defined as persistent facial and/or oral pain, with varying presentations but

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recurring daily for more than two hours/day over more than three months, in the absence of clinical neurologic deficit.[8] A dental cause has been excluded by appropriate investigations.[8] A previously used term for PIFP was atypical facial pain, and atypical odontalgia was also used.

The term "atypical odontalgia" has been applied to a continuous pain in one or more teeth or in a tooth socket after extraction, in the absence of any usual dental cause.[8] This is thought to be a subtype of 13.12 PIFP although it is more localized, the mean age at onset is younger and genders are more balanced. If there is an obvious history of trauma to the dental nerve, atypical odontalgia may also be a subform of 13.1.2.3 painful post-traumatic trigeminal neuropathy.[8] Despite of continued discussion and reports, these subtypes/forms, if they exist, have not been sufficiently studied to propose diagnostic criteria.[8] As shown in the present case, PIFP has a chronic course and is difficult to treat.

Referred trigeminal pain from chronic entrapment of the greater occipital nerve

The authors noted the possibility that pain in the right gum and occipital region in the present case may be a symptom of trigeminal neuralgia associated with occipital neuralgia. Referred trigeminal pain from occipital neuralgia (GON entrapment) is an important cause of occipital and facial pain that is not well known.[12,13,16,17] This referring of facial and occipital pain is caused by the convergence of nociceptive inputs of the trigeminal and occipital regions in the trigeminocervical complex (TCC).[1]

The GON originates from the medial branch of the dorsal rami of the second cervical nerve root.[19] It ascends through the semispinalis muscle and runs rostrolaterally before emerging into the scalp by piercing the aponeurotic fibrous sling between the trapezius and the sternocleidomastoideus near their occipital attachment to the superior nuchal line.[19] This aperture (trapezial tunnel) is a common site of entrapment of the GON.[5,12,13,16,17,19] After piercing the aponeurotic sling of the trapezial tunnel, the GON widens toward its course to the periphery.[5] This finding, as widening of the nerve makes it susceptible to entrapment especially in the firm trapezial aponeurosis.[5] The GON constitutes the main sensory afferents through the C2 root and this afferent input is transmitted directly to the C2 dorsal horn. The chronic, continuous, and noxious afferent input of GON entrapment appears to be associated with sensitization and hypersensitivity of the second-order neurons in the TCC.[1,11,16,17]

The TCC is a population of neurons in the C2 dorsal horn that receive convergent input from trigeminal nerve and occipito-cervical structures innervated by the high cervical C1-C3 roots.[1] Convergence of nociceptive afferents and sensitization of TCC neurons have clinical correlates, including hypersensitivity and the spread and referral of pain frequently seen in patients with primary headache such as migraines.[1] Patients with migraine headache often report pain not only involving the frontal head innervated by the trigeminal nerve but also involving the occipital region innervated by the GON.[1] As such, occurrence of referred facial pain by occipital nociceptive input has already been proven.[12,13,16,17] Pain of occipital neuralgia can reach the frontoorbital (V1) area through sensitization of TCC.[1] Referral to the facial trigeminal distribution occurred not only in the V1 region but also in the V2 and V3 regions and even caused hemifacial sensory changes.[12,13,16,17] In addition, referred trigeminal pain often occurred much earlier than the onset of occipital neuralgia.[16] Clinical manifestations of referred trigeminal pain caused by the sensitization of the TCC by chronic entrapment of the GON can be diverse.[16]

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The first right gingival pain in this case could be classified as PIFP because the cause could not be found. However, considering the late occurrence of ipsilateral occipital neuralgia, we considered this referred trigeminal pain from GON entrapment.[12,13,16,17] This is because, as described above, the referred pain caused by GON entrapment can occur before appearance of occipital symptoms. Indeed, we have experienced some cases in which the referred facial pain caused by GON entrapment coincides with or precedes occipital pain.[12,13,16,17] Therefore, we performed GON decompression together with internal neurolysis of the trigeminal nerve. The symptoms of occipital neuralgia improved, but the dysesthesia of TDP caused by trauma to the trigeminal nerve did not.

Painful post-traumatic trigeminal neuropathy (formerly-called anesthesia dolorosa)

Chronic PIFP of this patient took on the typical characteristics and symptoms of TDP due to nerve damage caused by the invasive treatment (PBC) for trigeminal neuralgia. Severe atrophy and discoloration of the trigeminal nerve found during trigeminal nerve exploration is thought to be the result of traumatic intraneural hemorrhage that occurred during the PBC procedure (**FIGURE 2B**). Thus, we were able to confirm this patient's pain as neuropathic pain of traumatic trigeminal neuropathy.[8] It is neuropathic pain due to obvious trigeminal nerve damage, known as intolerable dysesthetic sensory disturbance in the area of deafferentation. The effectiveness of neuroablative treatment for the stabbing pain typical of primary trigeminal neuralgia has already been demonstrated.[14] However, its effectiveness for PIFP (formerly called atypical facial pain), which is characterized by continuous dull and nagging pain, has nerve been reported. Invasive treatment is not recommended for PIFP.[14] Moreover, it can cause sensory disturbances that are much more serious and difficult to treat than PIFP, as in this case.

Painful anesthesia, formerly called as anesthesia dolorosa, is a well-known complication following neuroablative treatment for trigeminal neuralgia.[2,3,20] It can occur with any destructive procedure for the trigeminal nerve, including alcohol rhizotomy, radiofrequency gangliolysis, balloon compression, extradural and intradural rhizotomy, radiosurgery, craniofacial and oral trauma, even minor dental internventions.[2,3,20] The incidence of this troublesome dysesthetic sensory disturbance is highly variable, ranging from 4% to 10%, depending on the reports.[2] It is characteristically consistent for hours or for all waking hours. Burning, aching, stinging, pressure "like a flat hot iron pressed against the cheek," crawling on or under the skin "like glass being ground around," itching, a swollen sensation, numbness, and a wooden feeling worsening before the weather changes are some of the words and phrases which may be used to describe whit is felt.[20] In many of the patients a major part of the discomfort is in, around or in back of the eye regardless of the site of the original paroxysms of pain.[20] Early diagnosis and treatment are essential, because once chronic pain is established the condition is hard to treat.[2]

This post-traumatic trigeminal deafferentation pain, is a chronic pain that is difficult to treat like PIFP. When the pain of TDP patients who are refractory to medical treatment is chronic and severe, several invasive methods have been tried inevitably, although the evidence is weak. Anecdotal reports suggested that central procedures may be useful for recalcitrant TDP and PIFP cases.[11] However, it was not carried out due to our limited experience with DREZ (dorsal root entry zone) lesions in the caudate nucleus and concerns about neurological sequelae.[2,11] Therefore, trigeminal sensory rhizotomy or internal neurolysis was chosen as a less invasive alternative method.[10] In the trigeminal nerve exploration, the author's intention was to perform sensory rhizotomy if the nerve atrophy is not severe, and to perform

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less invasive internal neurolysis if the atrophy is too severe. Also, if the lateral suboccipital approach for the trigeminal nerve exploration was implemented, decompression of the GON could be performed simultaneously in the same operative field.

Neuromodulatory treatment

Trials of motor cortex and thalamic deep brain stimulation have been attempted as a last resort for refractory TDP.[15,18] Unfortunately, brain stimulation showed no analgesic effect other than a mild mechanical lesion effect. In the 1990s and early 2000s, motor cortex and thalamic stimulations were considered as alternatives for severe neuropathic pain that was not controlled by all other methods.[15,18] However, they are currently not recommended as treatment option in most cases due to insufficient long-term efficacy and medical evidence.[4] Nevertheless, although this treatment is invasive and the evidence is weak, it was attempted with the potential for control of chronic TDP that was too severe.[4]

Intrathecal drug administration holds substantial promise for pain management, particularly in the area of cancer-related pain and neuropathic pain.[6,9] Since demonstration of analgesic efficacy of intrathecal opioid in rats, intrathecal delivery of opioid has been used as an important pain treatment algorithm for cancer and noncancer-related chronic pain.[4,9] Intrathecal use of opioids allows the use of highly potent dose with minimal systemic side effects.[6] Opiates (morphine, hydromorphone, fentanyl, sulfentanil) are generally recommended for nociceptive pain, whereas nonopiates such as clonidine and ziconotide would be preferable for neuropathic pain.[6] The most common indication use of intrathecal opioids in humans remains nociceptive pain due to cancer and nociceptive pain due to mechanical spinal disorders.[6,9] Despite its long-term use for decades, the evidence showing the effectiveness of intrathecal opioid therapy on neuropathic pain is still weak.[4] The indications for intrathecal drug delivery vary with the agent infused.[4] Morphine and ziconotide are approved by the U.S. Food and Drug Administration (FDA) for intrathecal therapy and are recommended as first-line therapy for neuropathic pain.[6]

Ziconotide, which has been recommended as a first-line intrathecal drug, can be used instead of morphine, which has many side effects, and has been reported to be effective in neuropathic pain.[6] However, it has not been introduced in our country at present, so morphine has been tried as an intrathecal agent. Morphine is a proven effective agent for intrathecal pain control.[6,7,9] However, there are side effects of intrathecal morphine infusion such as constipation, nausea, lethargy, pruritis, diaphoresis, mental status change, urinary retention, and peripheral edema.[6,7,9] In addition, hardware problems of the intrathecal drug pump system such as migration and breakage of the catheter, catheter tip granuloma, twiddling of the pump are not rare.[6] Despite the complexity and limitations of intrathecal morphine, a trial of intrathecal morphine was unavoidable in this desperate case.

Although there is no firm consensus on where the catheter tip should be placed, placement of the catheter close to the spinal cord is recommended.[7] The authors installed the catheter tip at the level of the C5 vertebra for drug delivery as close as possible to the TCC located on the C2 cord. Distribution of morphine within the cerebrospinal fluid was reported to be very limited, and there were significant spinal cord drug concentration gradients in ambulatory animal study.[7] Morphine concentration decreased exponentially as a function of distance from the catheter tip, resulting in a 5- to 10-fold decrease over a distance of only 5 to 10 cm.[7]

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The long-term effect of intrathecal morphine on chronic neuropathic pain remains controversial.[4] However, the analgesic effect of intrathecal morphine exceeded our expectations in the current case. According to the patient's subjective assessment, intrathecal morphine halved her chronic pain and suffering. Not all opioids could be withdrawn from prescription, but prescriptions of transdermal fentanyl and gabepentin could be removed. In fact, impressed by the effect of intrathecal morphine in the current case, a trial of intrathecal morphine injection was conducted on two other patients with refractory anesthesia dorolosa. Unfortunately, trial morphine infusion failed in these patients due to pruritis and insufficient analgesic effect. Based on these results, it was difficult to conclude that intrathecal morphine therapy is universally effective for TDP. Nevertheless, if the chronic pain and distress of TDP is too severe, intrathecal morphine therapy is considered worth trying.

CONCLUSION

The application of neuroablative treatment for PIFP, which is continuous pain rather than typically stabbing pain of primary trigeminal neuralgia is dangerous. The unexplained facial pain of the present case is considered to be a referred trigeminal pain from chronic GON entrapment, and this entity needs attention. Traumatic manipulation of the innocent trigeminal nerve can result in the development of TDP, called anesthesia dolorosa. The severe pain of TDP is currently not curable. Fortunately, the present case has been relieved to some extent with intrathecal opioid therapy. In the experience of the authors, intrathecal morphine was not always successful in severe TDP, but in severe cases like the present case, it was worth a try.

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