An effective case of glycerol injection into the trigeminal cistern against trigeminal neuralgia resulting from pontine infarction

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Abstract
We experienced an unique case of trigeminal neuralgia resulting from pontine infarction. The patient was a 68-year-old woman who had suffered from the hemifacial numbness in the mandible on the right side since 1985. The numbness gradually recovered in six months, but she suddenly felt spontaneous toothache-like pain on that region in 1988. She was finally diagnosed as trigeminal neuralgia in 1993. She administered carbamazepine and had several mandibular nerve blocks with local anesthetics since 1993. When she was introduced our hospital in 2002, she had been suffering from stabbing pain in the mandible on the right side. Head MRI revealed only a wedge-shaped shadow on the right middle cerebellar peduncle. The patient was treated by an anhydrous glycerol injection of 0.25 ml into the right trigeminal cistern. Three days after the injection, she was discharged with complete pain relief.

The mechanism of pain relief produced by the glycerol injection is still unknown. One possible explanation is that the glycerol injection into the trigeminal cistern could suppress the abnormal excitation of partly demyelinated nerve fibers and may be effective for secondary trigeminal neuralgia, such as trigeminal neuralgia resulting from pontine infarction.

Key words: Trigeminal neuralgia; Pontine infarction; Glycerol injection; Trigeminal cistern

Idiopathic trigeminal neuralgia mainly results from the compression of the trigeminal nerve root by ectatic and twisting blood vessels. Other causes such as multiple sclerosis plaque and tumors can develop facial pain similar to trigeminal neuralgia. Here, we report an unique case of trigeminal neuralgia resulting from pontine infarction.

CASE REPORT
The patient was a 68-year-old woman. She had been suffering from the hemifacial numbness on the right side with general fatigue and nausea since 1985. Her family doctor suspected of menopausal disorder. The numbness gradually recovered without any sequela in six months.
In 1988, she suddenly felt spontaneous tooth-ache-like pain in the mandible on the right side. Although she had tooth extraction on that region, pain radiated occasionally to her right maxilla. She was diagnosed as trigeminal neuralgia and followed periodic administration of carbamazepine in 1993. Her pain continued for 4 to 5 days and then decreased for 2 to 3 months. In 1996, she was suffering from hypertension and started to take an antihypertensive medicine.

In 1998, she abruptly experienced spontaneous pain in the right side of the mandible so severe that she could not speak. She had mandibular nerve block with local anesthetics four times, which led her a partial pain relief. Then, the dose of carbamazepine was gradually increased to 600 mg per day. In 2002, she consulted another clinic for electric shock-like pain, which was evoked by speaking and chewing. She was administered right mental nerve block with mepivacaine. Since she still felt a stabbing pain in the gingiva on the right side of the mandible, she was introduced to our hospital.

After the administration of carbamazepine, her pain became less severe in remission, but did not completely disappear. She showed no abnormality of cranial nerves, such as the sensory disorders on her face. Although head MRI revealed no compression of the trigeminal nerve by blood vessels, it showed a wedge-shaped shadow on the right middle cerebellar peduncle (Fig. 1). For differential diagnosis, we considered pontine infarction, multiple sclerosis, and brain tumors. Head MR tomoangiography did not show any tumors. On account of the history of preceding facial numbness and no other neurological symptoms of multiple sclerosis, we diagnosed her case as trigeminal neuralgia resulting from pontine infarction.

The patient was treated in the operating room with C-arm units (also known as radiographic/fluoroscopic units). A 22-gauge needle of 10 cm was inserted into the trigeminal cistern by the anterior route under the C-arm navigation. Before the glycerol injection, we used a water-soluble contrast medium, iohexol (Omnipaque™), to visualize the trigeminal cistern (Fig. 2). Then, sterilized anhydrous glycerol of 0.25 ml was injected into the right trigeminal cistern through the needle. Three days after the injection, she was discharged with complete pain relief. She has been followed for 28 months and was free of pain without carbamazepine after the glycerol injection.
**Table 1** Case reports on trigeminal neuralgia resulting from pontine infarction

<table>
<thead>
<tr>
<th>Author</th>
<th>Nakamura</th>
<th>Kim</th>
<th>Golby</th>
<th>Delitala</th>
<th>Kohjiro</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nature of Pain</td>
<td>Not detailed</td>
<td>Electric shock-like, Lancinating</td>
<td>Lancinating, Explosive</td>
<td>Typical trigeminal neuralgia-like</td>
<td>Toothache-like, Electric shock-like</td>
</tr>
<tr>
<td>Trigger</td>
<td>?</td>
<td>Chewing, Tooth brushing, Spontaneous</td>
<td>Chewing, Touching</td>
<td>?</td>
<td>Speech, Eating, Spontaneous</td>
</tr>
<tr>
<td>Neurological Sign</td>
<td>Orofacial sensory deficit</td>
<td>Numbness of left tongue and upper gum</td>
<td>Hypesthesia</td>
<td>?</td>
<td>None</td>
</tr>
<tr>
<td>Preceding Sign</td>
<td>?</td>
<td>Lancinating pain over left forehead</td>
<td>Left hemifacial numbness</td>
<td>?</td>
<td>Right hemifacial numbness</td>
</tr>
<tr>
<td>Complication</td>
<td>?</td>
<td>Right vertebral artery stenosis</td>
<td>Myocardial infarction</td>
<td>?</td>
<td>Hyper lipidemia</td>
</tr>
<tr>
<td>Therapy</td>
<td>?</td>
<td>Carbamazepine</td>
<td>Percutaneous glycerol rhizotomy</td>
<td>Partial sensory rhizotomy</td>
<td>Percutaneous glycerol rhizotomy</td>
</tr>
<tr>
<td>Prognosis</td>
<td>Good</td>
<td>Good</td>
<td>Good</td>
<td>Good</td>
<td>Good</td>
</tr>
</tbody>
</table>

**DISCUSSION**

Causes of the trigeminal neuralgia became clear with the development of visually diagnosing equipments in past decades. They revealed ectatic blood vessels, arteriovenous malformation, tumors, deformed bone in the posterior fossa, multiple sclerosis plaque, and pontine infarction. Several investigators had reported the case of the trigeminal neuralgia resulting from the pontine infarction. They reported similar symptoms as follows: preceding neurological signs on the distribution of trigeminal nerve, paroxysm and remission same as idiopathic trigeminal neuralgia, and good outcome from therapeutic procedure. In our case, there was preceding hemifacial numbness, but no neurological deficit was found on consultation. Head MRI showed no vascular compression to the trigeminal nerve. On the other hand, a wedge-shaped shadow in the middle cerebellar peduncle was found, which was similar to other reports on trigeminal neuralgia resulting from pontine infarction. We assumed that hypertension, which was the most common and important risk factor for infarction, was a possible cause for pontine infarction in this case. Lacunar occlusions or basilar atheromatous branch occlusions were likely to be the pathogenetic mechanisms of pontine infarction.

The etiology and clinical features of trigeminal neuralgia are various. Sites of pain from multiple sclerosis sometimes occur bilaterally. The remission is not obvious and the course of pain is progressive. Characteristics of pain caused by infarction and multiple sclerosis are widely ranged. The onset age of facial pain with multiple sclerosis is younger than that of facial pain with infarction and idiopathic trigeminal neuralgia. The typical image of head MRI shows a wedge-shaped shadow in the pons in the case of infarction, an oval-shaped plaque in that of multiple sclerosis, or compression of the trigeminal nerve by vessels in that of idiopathic trigeminal neuralgia.

After the glycerol injection into the trigeminal cistern was performed at the distal site of the lesion in the pontine, her facial pain was well relieved. In the case of the trigeminal neuralgia in the multiple sclerosis with plaque in the pons,
there are many case reports where glycerol injection into the trigeminal cistern produced pain relief. One possible explanation is that the glycerol injection into the trigeminal cistern could suppress the abnormal excitation of partly demyelinated fibers which are involved in the trigger mechanism.

Secondary trigeminal neuralgia caused by infarction in the pons has aspects somehow similar to those of idiopathic trigeminal neuralgia. Therefore, it is worth trying the administration of carbamazepine and the glycerol injection into the trigeminal cistern to the secondary trigeminal neuralgia.

References
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