Management of traumatic neuralgia in a patient with the extracted teeth and alveoloplasty: a case report

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A majority of patients who sustain injuries to the peripheral sensory nerves of the face and jaws experience a slow but gradual return of sensation that is functional and tolerable, if not the same as before the injuries. However, long-term effects of such injuries are aggravating for many patients, and a few patients experience significant suffering. In some of these patients, posttraumatic symptoms become pathological and are painful. The predominant painful components are (1) numbing anesthesia dolorosa pain, (2) triggered neuralgiaform pain, (3) burning and aching causalgiaform pain, and (4) phantom pain. This is a case report of conservative management of traumatic neuralgia and neuritis as part of posttraumatic pain syndromes in geriatric patients who have undergone the teeth extraction and alveoloplasty.

Key Words: Alveoloplasty; Neuritis; Posttraumatic pain syndrome; Tooth extraction; Traumatic neuralgia.

Pain and sensory disorder resulting from injury to peripheral nerves of the face and jaws are a major source of patient dissatisfaction and suffering. Although there has been a reduction in nerve injury from traffic accidents, the greater availability of oral and maxillofacial operations has, ironically, increased the risk of concomitant iatrogenic nerve injury. Often, attempts by oral and maxillofacial surgeons to help nerve injury patients have been hampered by medicolegal and disability considerations that surround this problem. A better understanding of the nature and effects of sensory nerve injuries and the recent development of microsurgical techniques offer new possibilities for improved treatment of neuropathies.

The majority of patients who sustain injuries to the peripheral sensory nerves of the face and jaws experience a slow but orderly return of sensation that is functional and tolerable in quality, if not normal (i.e., return of pre-injury levels of function and sensation). For some patients, however, the posttraumatic symptoms become pathological and painful. The predominant pain components are (1) numbing anesthesia dolorosa pain, (2) triggered neuralgiaform pain, (3) burning and aching causalgiaform pain, and (4) phantom pain [1,2].

Of these components, the most serious clinical problem is a triggered pain in the form of trigeminal neuralgia, similar to convulsions [3]. This study discusses the case of a geriatric patient with cerebral infarction who had undergone dental extraction and alveoloplasty in a broad range, and his postoperative pain in the form of posttraumatic trigeminal neuralgia was treated with intravenous (I.V.) infusion and oral administration of carbamazepine, resulting in effective analgesia and desirable prognosis.

CASE REPORT

A 74-year-old man was admitted to Wonju Severance...
Christian Hospital for extraction of his remaining maxillary teeth and mounting of a removable full denture after alveoloplasty. He had suffered a stroke due to a cerebral infarction three months ago and was on medication while hospitalized and under the care of the Neurology Department. Although he was undergoing treatment at the rehabilitation department, a combined examination revealed that dental surgery likely would be uneventful.

On the basis of an oral examination and radiography results showing progressive periodontitis, it was deemed necessary to extract the remaining maxillary teeth (#15, #17, and #25) for mounting of a removable full denture; alveoloplasty of teeth #12–#15 and #27 and the surrounding regions was required where sharp margins of the remaining alveolar bones were present.

The three teeth extractions and the alveoloplasty procedures were performed under local anesthesia by use of 2% lidocaine HCl with 1:100,000 epinephrine on August 17, 2015. Wound closure was uneventful, and sutures were removed at postoperative day 7. On day 10 postoperatively, he was urgently admitted to our Department of Dentistry for paroxysmal throbbing pain in regions 12, 13, 14, 15, and 17, comprising the sites of tooth extraction and alveoloplasty. His vital signs were normal, and there were no abnormal findings on oral examination and radiography (Fig. 1).

Neuritis owing to systemic weakening was diagnosed; posttraumatic pain syndrome was caused by tooth extraction and alveoloplasty. The authors determined the pain to be trigeminal neuralgia pain, and clinical tests were performed accordingly (Table 1). As therapy for acute neuroinflammation, an I.V. infusion (normal saline 1,000 cc I.V., clindamycin 600 mg I.V., diazepam [half ampule mixed with 10 cc distilled water] was administered. As a result, the pain was decreased but did not disappear completely. Therefore, a cephalosporin oral antibiotic, anti-inflammatory drugs (streptokinase, acetaminophen), an anti-seizure drug (carbamazepine 300 mg/day), and a digestive drug (simethicone) were administered orally three times a day for one week.

The pain was decreased further and improved to a

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**Table 1. Initial major laboratory data and normal ranges**

<table>
<thead>
<tr>
<th>Test</th>
<th>Value</th>
<th>Normal Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>WBC</td>
<td>6.23</td>
<td>E 9/L</td>
</tr>
<tr>
<td>RBC</td>
<td>3.11</td>
<td>E 12/L</td>
</tr>
<tr>
<td>Hemoglobin</td>
<td>10.9</td>
<td>g/dL</td>
</tr>
<tr>
<td>Hematocrit</td>
<td>33.9</td>
<td>%</td>
</tr>
<tr>
<td>Platelet</td>
<td>350</td>
<td>E 9/L</td>
</tr>
<tr>
<td>BUN</td>
<td>6.3</td>
<td>mg/dL</td>
</tr>
<tr>
<td>Creatinine</td>
<td>0.80</td>
<td>mg/dL</td>
</tr>
<tr>
<td>Glucose</td>
<td>103</td>
<td>mg/dL</td>
</tr>
<tr>
<td>ESR</td>
<td>54</td>
<td>mm/h</td>
</tr>
<tr>
<td>Albumin</td>
<td>3.3</td>
<td>g/dL</td>
</tr>
<tr>
<td>Globulin</td>
<td>2.7</td>
<td>g/dL</td>
</tr>
<tr>
<td>SGOT</td>
<td>19</td>
<td>U/L</td>
</tr>
<tr>
<td>SGPT</td>
<td>18</td>
<td>U/L</td>
</tr>
<tr>
<td>Total protein</td>
<td>6.0</td>
<td>g/dL</td>
</tr>
<tr>
<td>CRP</td>
<td>1.06</td>
<td>mg/dL</td>
</tr>
</tbody>
</table>

WBC, white blood cell count; RBC, red blood cell count; BUN, blood urea nitrogen; ESR, erythrocyte sedimentation rate; SGOT, serum glutamic oxaloacetic transaminase; SGPT, serum glutamic-pyruvic transaminase; CRP, C-reactive protein.
tolerable level on August 31, 2015, and the medication dosing regimen was reduced to twice daily. Thereafter, the patient recovered completely on September 14, 2015, after two weeks of treatment, and he was transferred to the prosthetic dentistry department for mounting of a removable full denture.

**DISCUSSION**

When diagnosing maxillofacial neurological problems, the examination of the neurological history should begin with a question on the systemic diseases and environmental conditions that have neurological effects on the maxillofacial region. Patients should then be questioned about past history of major neurological and psychiatric disorders. Once the systemic, environmental, primary neurological, and psychiatric disease factors have been taken into consideration, the patient should be questioned on the nature and incidence of lesions in the maxillofacial tissues themselves [1,6]. On the basis of the neurological history, it may be possible to make a tentative pathological diagnosis, such as an infectious, degenerative, arterial or demyelinating condition, or a tentative etiologic diagnosis such as a posttraumatic, odontogenic, psychogenic, or diabetic condition. Because the patient in this case also had metabolic disorders including anemia, among other systemic environmental factors, and the major neurological disorder was neuralgia, which occurred after tooth extraction during dental surgery and alveoloplasty, he was diagnosed with traumatic neuralgia.

In addition, clinicians should examine specific and local sources of acute neuritis in detail using routine dental diagnostic skills and aids such as inspection, palpitation, radiography, and electrodiagnosis. In summary, a differential diagnosis is made after reviewing the neurological history and examining a patient with maxillofacial neurological disorders. The four disease elements (symptoms, pathology, location, and etiology) should be ascertained in each case. In particular, it is necessary to differentiate posttraumatic neuropathy after oral maxillofacial surgery from the existing masticatory myofascial pain, trigeminal neuralgia, and systemic neuropathy.

In clinical dentistry, neuralgia is defined as paroxysmal, intense, and intermittent pain that usually is confined to specific nerve branches of the head and neck. Currently, studies have shown that paroxysmal spurts of maxillofacial pain may share a common histopathology, which is caused by disruption of the insulating mechanism between axons without destroying them [2,9]. This primary condition may occur in peripheral nerve branches, sensory ganglion tissues, or posterior roots. Various study results have shown that these peripheral lesions could cause pain by creating afferent imbalances and setting up abnormal pools of secondary central neurons in the trigeminal descending tract nuclei, possibly the epileptogenic foci type [8,9].

An injured nerve undergoes segmental dymyelination, Wallerian degeneration, and degeneration processes such as dying-back neuropathy, neurotrophic effect, and normal or abnormal regeneration; among these, the biggest clinical problem is posttraumatic pain syndromes due to abnormal regeneration from injury during surgery [2,6]. Unfortunately, many other factors may delay the return of proper functioning in regenerated peripheral nerves, resulting in abnormal nerve regeneration. Poorly myelinated tubular zones, called neuromas in continuity, may occur in the regenerated nerve.

The nerve tissues of the peripheral neumomas rarely mature and myelinate, and thus their stimulation may result in bursts of intermittent pain and bizarre paresthesias (e.g., “pins and needles” sensations). This phenomenon may be explained by setting up artificial synapses, in which impulses in one demyelinated fiber may excite neighboring demyelinated fibers, resulting in an abnormal chain reaction to the original stimulus. The concept of an artificial synapse occurring in pathological peripheral nerve zones may be a common explanation for many trigeminal neuralgias, including multiple sclerosis lesions and paroxysmal shocking pain of the tic douloureux. A similar explanation can be applied for the deep burning...
pain of posttraumatic causalgia, which may be caused by
the excitation of demyelinated sensory nerve segments
by adjacent unmyelinated sympathetic fibers.

In addition to neuroma formation, other potential rege-
nerations can occur. Relocation of the growth cone fibers
in the distal Schwann cell passages reportedly is largely
a nonspecific selection process, and the identical match-
ing of new regenerating fibers with their former tissue
receptors may not occur. Fibers may innervate the wrong
tissues, potentially disrupting motor controls in reinerv-
ated skeletal muscle and glands; because regenerated
fibers rarely attain their original diameters, distances
between the nodes of Ranier may decrease in regenerated
nerves. These two factors can lead to disproportionate
reduction of nerve conduction velocities [10]. According
to gate control theories of pain and sensory modulation,
imbalance in afferent fiber diameters could lead to
sensory abnormalities such as hyperpathia.

In addition to the imbalance induced by histopatho-
logical conditions in the peripheral nerve fibers, serious
imbalances also can be caused by selective effects on the
nerve cell bodies themselves. Moreover, various studies
have suggested that trigeminal ganglion cell bodies can
be lost selectively as a result of various life and disease
processes. Trauma, metabolic disease, and viral infection
can result in neuronal necrosis. Peripheral nerves sub-
sequently may undergo Wallerian degeneration, and
central nerves may disintegrate. This can damage the
functional synaptic connection with secondary transmi-
sion, reflex, and integration centers in the central nervous
system.

Trigeminal deafferentation (i.e., loss of peripheral
fibers and synaptic contacts that normally reach the
primary synaptic regions) can induce both morphological
and physiological changes in the nuclei of the descending
trigeminal tract [4,11]. Deafferentation conduct patterns
in brain stem regions are similar to that of initial epilepsy
response, showing electrical characteristics called epilep-
togenic foci. It has been postulated that these epilepto-
genic firing patterns may represent the physiological
change responsible for paroxysmal and atypical neuralgia
states. Deafferentation effects eventually may explain the
cause of poorly understood conditions such as trigeminal
neuralgia, postherpetic neuralgia, and phantom pain. Bell
et al. grouped deafferentation pain states as a syndrome
classified by (1) posttraumatic pain, (2) traumatic neu-
roma, (3) reflex sympathetic dystrophy, (4) neuritic neu-
ralgia, and (5) phantom pain [4,6].

As in the present case, a stabbing, flashing pain may
be experienced within the first several weeks following
a nerve injury, and thus clinicians should always consider
the possibility of a secondary source of mechanical
irritation or inflammation in the still-intact nerve trunk.
Clinicians should search for entrapped or pinched nerves,
in order to eliminate the acute sources of neuritis in-
cluding foreign bodies, mobile bone fragments, and in-
fec tion. Triggered pain lasts for more than a few weeks
or emerges after a delayed period of anesthesia and is
usually accompanied by sensory neuropathy. This pain
component is like conventional tic douloureux in that fine
tactile or mild heat stimuli evoke brief hyperesthesias and
sharp “tingling” or stabbing sensations. These pains may
be due to either peripheral neuromas or secondary
deafferentation pathology in the brain stem. In these
cases, clinical examination is of considerable importance
because pain triggered by manual palpation at specific
points along the neurovascular bundle is suggestive of
neuroma.

However, in cases of tooth extraction or alveoloplasty
that have no possibility of neuroma as in the present case,
posttraumatic trigeminal neuralgia can be induced by
deafferentation pathology phenomenon. In other words,
spontaneous or random spasms of pain are probably
linked more to the central neuropathy. Peripheral
microsurgery is highly beneficial for patients who have
neuromas, whereas centrally acting pharmacological
agents are more appropriate for patients with central
neuropathology [1,12].

Hence, the patient in this case was administered oral
carbamazepine, which is effective for the treatment of
trigeminal neuralgia to reduce excessive pain in the form
of posttraumatic neuralgia. At the same time, I.V. infusion
Neuralgia in patient with extraction was accompanied with antibiotics, anti-inflammatory drugs, and sedatives to improve systemic symptoms and neuritis conditions, with good clinical results.

The neuritis mentioned here means “inflammation of the nerve” and pertains to acute reversible irritations to maxillofacial nerves. Neuritis occurs in the sensory, motor, or autonomic nerves and results from peripheral pathology that infect, compress, entrap, or erode the adjacent nerves. Neuritis is significant because it is an indication of an acute pathological condition; once it persists, it induces degenerative and irreversible neuropathy. Sensory neuritis—characterized by a decline in pain thresholds—is almost always manifested as pain, but its characteristics depend on the location and nature of the primary lesion. Neuritis etiology includes trauma, infection, paranasal sinusitis, otalgia, salivary gland disorders, mucosal disorder, motor neuritides, and myofascial dysfunction [1,2].

In conclusion, even after routinely performed tooth extraction or alveoloplasty in clinical dentistry, neuritis can occur in the trigeminal nerve, as can posttraumatic pain syndrome (e.g., trigeminal neuralgia, neuropathic pain, phantom pain). Therefore, continuous monitoring and management are required until the wound has completely healed.

REFERENCES