A 39-year-old male presented with severe pain in right posterior mandibular teeth and temporal area. Initially, the pain in the mandibular teeth was moderate, but the concomitant headache was unbearably severe. His medical history was non-contributory. The clinical and radiographic examination failed to reveal any pathology in the region. There was no tenderness to palpation in the temporalis and masseter muscles or temporomandibular joints. The clinical impression was migraine. The pain in the teeth and headache were aborted using ergotamine tartrate and sumatriptan succinate. Atenolol prevented further pain, while amitriptyline and imipramine had no effect. Migraine can present as non-odontogenic pain in the mandibular teeth, although not as frequently as in the maxillary teeth. A correct diagnosis is essential to avoid unnecessary dental treatments and to manage pain effectively. Clinicians should be able to identify migraine with non-odontogenic dental pain and establish a proper diagnosis through a comprehensive evaluation.

**Key Words:** Facial pain; Headache; Migraine without aura; Toothache

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**INTRODUCTION**

The majority of toothaches are caused by inflammation or infection of the pulp and periradicular tissues. However, there are various non-odontogenic sources that may be responsible for pain felt in the teeth. Non-odontogenic toothaches may be caused by muscle, maxillary sinus, neuro-pathic, neurovascular, cardiac, and psychogenic problems. Headache disorders can present with pain in the orofacial area because of at least two reasons: it shares the trigeminovascular system and by pain mechanisms, such as neurogenic inflammation. It has been reported that several types of headaches including migraine, tension type headache, cluster headache, and paroxysmal hemicrania can cause toothache.

Migraine is characterized by unilateral pain with pulsatile quality, accompanied by symptoms, such as nausea and vomiting. The diagnostic criteria for migraine according to the International Headache Society are shown in Table 1.

During the migraine attack, stimulation of the trigeminal nerve may induce the release of vasoactive peptides and cause referral pain to any of the three branches. This results in pain in the orofacial structures, such as orbit, maxillary sinus, cheek, teeth, and jaws.

Among the neurovascular diseases that may cause pain in the tooth area, cluster headache and paroxysmal hemicrania are comparatively easily distinguished, because of the distinct characteristics, such as the location, intensity, frequency, and duration of the pain. However, migraine may be difficult to differentiate because they are characterized by pulsating pain, which is like pulpal pain.

Dental pain associated with migraine have been reported infrequently and mostly in the maxillary teeth. In this case presentation, we describe a rare case of...
migraine-associated non-odontogenic pain in the mandibular teeth.

**CASE REPORT**

A 39-year-old male visited the Department of Oral Medicine, Chonnam National University Dental Hospital complaining of recurrent pain of his lower right molars and a concomitant headache of the right temporal area (Fig. 1). The pain symptoms had been present for nine months. Pain attacks always began from the mandibular molar on the right side, and then a headache on the right unilateral side followed the toothache. The headaches were far more severe than the preceding toothaches. They were described as unbearably painful (NRS=9/10), nearly causing tearing and disabling him. Each pain episode lasted several hours, unless controlled with analgesics. The pain usually subsided 30 minutes to one hour, after taking an over-the-counter medication, such as a Tanaxen tablet (acetaminophen 500 mg and caffeine 65 mg; Yuhan Corp., Seoul, Korea). The pain symptoms usually occurred once a day, recurring four days a week. There was no nausea, photophobia or phonophobia around the time of the toothache and headache.

He had esophageal reflux disease, which had been treated pharmacologically, for about one year at a local community hospital. He had suffered a traumatic injury of his head from a traffic accident 15 years ago, but he completely recovered from the injury without complication. He also had a smoking habit lasting 10 years. No other specific systemic diseases were reported.

Clinical examination of the head, face, jaw, teeth, gingiva, and oral mucosa did not suggest significant findings (Fig. 2). No tooth was tender to percussion. Upper and lower teeth on right side responded within normal limits to palpation and bite test and showed positive response to vitality tests. Dental occlusion was favorable. Slight gingival recession was noted on the palatal side of the right maxillary first and second molars. None of the temporalis muscles,

<table>
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<th>Table 1. Diagnostic criteria for migraine</th>
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<td>A. At least five attacks fulfilling criteria B-D</td>
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<td>B. Headache attacks lasting 4-72 hours (untreated or unsuccessfully treated)</td>
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<td>C. Headache has at least two of the following four characteristics:</td>
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<td>1. Unilateral location</td>
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<td>2. Pulsating quality</td>
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<td>3. Moderate or severe pain intensity</td>
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<td>4. Aggravation by or causing avoidance of routine physical activity (e.g., walking or climbing stairs)</td>
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<td>D. During headache at least one of the following:</td>
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<tr>
<td>1. Nausea and/or vomiting</td>
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<td>2. Photophobia and phonophobia</td>
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<td>E. Not better accounted for by another ICHD-3 diagnosis.</td>
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ICHD, International Classification of Headache Disorders.
masseter muscles, or temporomandibular joints of either side were tender to palpation. Panoramic radiography did not reveal significant pathology, except a periapical rarefaction of the mandibular lateral incisor on the left side (Fig. 3). The root canal of the involved tooth was radiopaque with filling materials. The rarefaction lesion had a thick sclerotic border.

A careful review of the patient history and examination findings gave the clinical impression of probable migraine without aura, according to the International Classification of Headache Disorders. The preceding pain of the mandibular posterior teeth on the same side of the headache was thought to be an atypical manifestation of referred pain.

To abort pain episodes, several tablets of Cafergot (Novartis, Basel, Basel-Stadt, Switzerland) (ergotamine tartrate 1 mg and anhydrous caffeine 100 mg) were prescribed with the usage of ‘pro re nata’ (as needed). In addition, we prescribed amitriptyline 10 mg once a day at bedtime, for one week for prophylactic purposes.

At the follow-up visit one week later, the patient reported that amitriptyline had no effect in preventing the pain. On the contrary, Cafergot was very effective in resolving the toothache and in suppressing the headaches. If he administered Cafergot at the time of initial toothache development, the toothache was soon relieved and headache did not follow. However, if he took Cafergot after he had felt headache, pain resolved slowly after an hour. At this visit, another tricyclic antidepressant (TCA), imipramine, was prescribed instead of amitriptyline along with Cafergot. He was directed to take 12.5 mg of imipramine a day at bedtime, for eight days.

At the third visit, he stated that there was no improvement in recurrent pain symptoms, although he took imipramine every day. During the previous eight days, he had to take Cafergot five times (one tablet each time) to control the pain. At this time, abortive and preventive medications were replaced with oral sumatriptan succinate (50 mg tablet, pro re nata) and atenolol (25 mg tablet per day), respectively.

At the fourth visit nine days later, the patient reported that he had no pain during the past four to five days and he had not suffered from a severe headache. He took sumatriptan succinate twice, only for mild to moderate pain attacks. Furthermore, there had been no pain attack for the last three days, although he could not take atenolol, due to a lack of medication. Blood pressure was 117/89 mmHg with pulse rate of 89 beats per minute (bpm). The daily dosage of atenolol was reduced to 12.5 mg from this visit on. At the fifth visit five weeks later, he experienced neither toothache nor headache after the last visit.

At the sixth visit three months later, he reported that the headaches had nearly disappeared, after he started taking atenolol. He had taken oral sumatriptan succinate only twice during the last three months. He tried taking atenolol 12.5 mg every other day for 1 week and found no pain recurred. At this time, propranolol 40 mg per day was prescribed, instead of atenolol for one month to check whether another beta blocker also had similar effectiveness.

However, he came back one month later, having not followed the doctor’s order during the last follow-up period. Blood pressure was 110/79 mmHg with pulse rate of 85 bpm. These values were not different from those measured during the time he had taken atenolol. It was inferred that atenolol as little as 12.5 mg per day had no significant influence on his blood pressure. He reported an interesting symptom related to smoking at this visit. When he smoked a cigarette, headache pain arose faster than when not smoking. He had no pain during the time he did not smoke. Atenolol was prescribed for two months because he preferred it to propranolol.

**DISCUSSION**

The diagnosis of the present case presentation was ‘probable migraine without aura’, which differs from migraine.
without aura in that its attacks fulfill all but one of criteria A-D for migraine without aura. The patient did not show any of nausea, vomiting, photophobia, and phonophobia, however, other clinical characteristics were all well within the criteria for migraine without aura.

Migraine is regarded as a neurovascular disorder with alterations in trigeminal sensory processing. Until recently, the mechanism of migraine has not been clearly elucidated. It seems to be a disorder of the trigeminal sensory system occurring at the brainstem level, and is thought to induce neurogenic inflammation. When an antidromic transmission occurs in the primary nociceptive neurons, vasoactive neuropeptides, such as substance P and calcitonin gene-related peptide are released and cause vasodilation. These substances, additionally induce the release of algogenic substances, such as serotonin, histamine, bradykinin, and prostaglandin. These algogenic substances sensitize primary afferent nociceptors and this leads to central sensitization, which is associated with allodynia and hyperalgesia. Therefore, when migraine occurs, hypersensitivity of the intracranial and extracranial regions is present, where the trigeminal nerve branches are distributed. This can be explained by the sensitized second-order neurons receiving converging inputs in other adjacent regions.

Orofacial pain associated with migraine can occur anywhere in the area where the trigeminal nerve is distributed and is more prevalent in the maxillary branch. In rare cases, migraine-related toothache has been reported in the mandible. In this case, there were no abnormal findings in the clinical and radiological examinations and the toothache was always accompanied by headache. As such, it could be diagnosed as toothache associated with migraine. The results of pharmacological therapy supported the diagnosis of migraine and related dental pain.

Pharmacologic treatment of migraine can be divided into drugs that abort migraine and those that prevent migraine attacks. Analgesics may be effective for the treatment of migraine, and can be used alone or in combination with caffeine. However, most of these drugs are over-the-counter, so overuse can lead to medication overuse headaches.

Ergotamine is an alpha-adrenergic blocker and vasoconstrictor of the cranial smooth muscle and is considered in the treatment of patients with moderate or severe migraine. Caffeine is added to enhance absorption and potentiate analgesia. Acute treatment of headaches with caffeine is sometimes effective, but an excessive use of caffeine is known to induce dependence or increase the frequency of migraine and should be used with caution.

Sumatriptan is a 5-HT_{1B/1D} receptor agonist, the first drug in a new class of specific antimigraine drugs. Sumatriptan reduces increased blood flow and neurogenic inflammation of the trigeminal nerve terminals. This inhibits the neuronal release of vasoactive peptides. The two abortive medications used in this case, both ergotamine tartrate with anhydrous caffeine and sumatriptan succinate were effective.

Many classes of pharmacological agents including anti-epileptic drugs, beta blockers, calcium channel blockers, antidepressants, and serotonin antagonists have been used in the treatment of migraine for prophylactic purposes. Medication is appropriately selected and adjusted to the medical condition of the patient. Conditions may include depression, anxiety, hypertension, cardiovascular disease, and sleep disorders. Possible therapeutic mechanisms include stabilization of the reactive central nervous system, enhancement of anti-nociceptive pathways, and inhibition of peripheral sensitization.

In this case, two kinds of prophylactic medications were tried. Atenolol, belonging to beta blockers, as low as 12.5 mg/day was sufficient to prevent pain attacks, although a generally recommended adult dose with established efficacy is 50-150 mg/day. TCAs, both amitriptyline 10 mg/day and imipramine 12.5 mg/day, had little effect after one week of administration. However, the duration of administration of these two agents seems not enough long to evaluate their effectiveness. They were tried but switched to another agent hastily to find out more effective ones. In general, TCAs are known to take several weeks to show their effectiveness.

Interestingly, the patient reported an increase in headache after smoking. The relationship between migraine and smoking is still controversial. However, recent studies have reported that smoking may be a precipitating factor of migraine. In addition to smoking, lifestyle factors associated with migraine include dieting and fasting, exercising and physical activity, stress, and sleep pattern. It is necessary to modify these factors, which can trigger headaches, for the treatment of migraine.
Migraine is a common headache disorder. If migraine is accompanied by pain felt in the teeth, face, or maxillary sinus, patients may visit dental clinics for evaluation and treatment of such pain. If there are no abnormalities of the teeth and its surrounding structures, clinicians must consider non-odontogenic pain including neurovascular disorders, such as migraine. A differential diagnosis is crucial in patients with non-odontogenic toothaches to avoid inappropriate and unnecessary dental treatments including root canal therapy, periodontal treatments, and tooth extraction, as these would fail to alleviate the symptoms of the patients. Dentists should be aware that migraine may present as pain anywhere within the areas innervated by the trigeminal nerve. They should be able to differentiate pain associated with migraine with common dental pain conditions. Clinicians should be able to establish an accurate diagnosis through a comprehensive evaluation and careful history evaluation. Prescribing drugs specific to migraine, such as triptans, may be useful for diagnostic purposes.

In conclusion, migraine can present as a toothache in the mandibular posterior teeth, like in this case presentation, without a definite odontogenic cause. Proper understanding of the disease and a comprehensive evaluation of the patient can help correct diagnosis and can prevent unnecessary dental treatments. Such pain is effectively managed with migraine therapies and may require a referral to a specialist in orofacial pain.

**CONFLICT OF INTEREST**

No potential conflict of interest relevant to this article was reported.

**REFERENCES**