Introduction
The international classification of headache disorders defines migraine as “a common disabling primary headache disorder.” The term primary refers to a pathology that is not a symptom produced by another disorder, but is a disorder itself. In fact, secondary migraine must be diagnosed and coded according to the causative disorder.

Migraine is characterized by headache lasting 4-72 hours, with at least two of the following characteristics: unilateral location, pulsating quality, moderate or severe pain intensity, and aggravation by or causing avoidance of routine physical activity. During headache, at least one of the following must be present: nausea and/or vomiting, photophobia, and phonophobia.

Migraine headache can be preceded by the appearance of visual and/or sensory and/or speech symptoms that develop gradually over more than 5 minutes and that last for less than 1 hour. In this case, it is diagnosed as migraine with aura.

However, some reports have been found in the literature where patients diagnosed with migraine were successfully treated by correcting dental occlusion and reducing dental parafunctions. Such results may suggest that in some cases, headache related to dental occlusion and dental parafunctions is able to mimic a primary migraine headache, and therefore treatment of the causative disorder will resolve the headache.

In this case study, we will try to understand if there is a relationship between migraine headache, dental occlusion, and dental parafunctions. We will analyze the reports that have been published in the literature, and describe a clinical case of a patient affected by migraine with aura with concomitant temporomandibular joint (TMJ) and masseter muscle pain, and successfully treated by the use of a dental occlusal appliance (DOA).

Case Report
A 59-year-old Caucasian female patient came to our dental office for evaluation. Her chief complaint was a severe headache that had been diagnosed as migraine with aura by different neurologists after assessment by two headache clinics in Pisa and Cagliari, Italy.

The headache was described as an intense throbbing pain located in the forehead and temples, bilaterally, associated with photophobia and phonophobia, nausea, and occasional vomiting. The headache usually lasted for days if untreated and tended to start in the morning upon waking.

Initially, the migraine was not preceded by aura, but then attacks started alternating with episodes of migraine preceded by a visual aura that was described as the appearance of small pulsating scintillating flashes starting a few minutes before onset of the headache and persisting during the headache.

Further investigations, including neurological and ophthalmic evaluations, magnetic resonance imaging of the head, and echo-color Doppler of the carotid arteries, revealed the presence of an arachnoid cyst located in the left cerebral hemisphere that was considered unrelated to the symptomatology of the patient.
Several medications were tried for the treatment of headache, including nimesulide, ibuprofen, and indomethacin. The only drug that was found effective was sumatriptan, which succeeded in aborting migraine attacks; however, an almost daily dose of the medicine was needed to control headache.

The presence of dental malocclusion—more precisely, a reduction of the vertical dimension of occlusion (VDO) with evidence of a deep overbite—made a dentist provide the patient with a DOA to use at night during sleep. The device had to be worn on the upper teeth, and it had even contact with all of the lower teeth. The use of this appliance gradually reduced the intensity and frequency of migraine headaches, which ceased in a few weeks; nonetheless, aura attacks continued but were not followed by headaches. The aura episodes started as previously described with the appearance of small pulsating scintillating flashes, but then developed with the progressive expansion of the flashes that proceeded with zigzag movements in a circle, delimiting areas of black spots (scotomas) gradually enlarging, sometimes comprising the entire visual field, producing transient complete blindness of duration up to 40 minutes.

At the time we first saw the patient, she also reported some masseter muscle fatigue and soreness on the right side, especially during mastication, and temporomandibular joint dysfunction pain on the right side, exacerbated by mandibular movements. She also reported nighttime tooth clenching. A reciprocal click was palpated on the right TMJ during opening and closing of the mouth, suggesting anterior displacement of the articular disc. Panoramic X-ray showed osteoarthrosis of both TMJs.

The old DOA that the patient was wearing at night increased the VDO by an amount that was considered excessive. Therefore, a new appliance was prepared that moderately increased the patient’s VDO, and the subject was instructed to wear it every night during sleep; the new appliance also eliminated all tooth contacts other than the ones between the upper and lower incisors, to maximally reduce clenching activity. Additionally, serrapeptase, an antiedematous medication, was prescribed to reduce TMJ pain.

During the following month, the patient was free from headache and the visual aura started improving. In fact, the duration and frequency of aura symptoms began to decrease, and the small scotomas failed to enlarge to the point of covering the entire visual field. TMJ and masticatory muscle pain also subsided. During the second month, no more headache and aura symptoms occurred; however, mild masseter muscle fatigue was occasionally present. At three- and six-month follow-ups, no recurrence of headache and aura symptoms was reported and TMJ pain was resolved, but there was still some masseter muscle fatigue present sporadically. No further treatment was proposed other than continuing the use of the DOA at night during sleep.

**Discussion**

The case showed how the use of a DOA at night during sleep was effective in relieving both headache and aura symptoms in a period of two months.

The diagnosis of migraine with aura had been made by several neurologists in two different headache clinics in Italy, and the clinical appearance seemed to confirm that diagnosis. In fact, the criteria of the International Headache Society were fulfilled: The headache lasted for days if untreated; it was pulsating, severe, and caused avoidance of routine physical activity; it was associated with nausea and, occasionally, vomiting, photophobia, and phonophobia. A visual aura was also present. To rule out the presence of other disorders that might be responsible for the headache and the aura symptoms, investigations were performed, including neurological and ophthalmic evaluations, magnetic resonance imaging of the head, and echo-color Doppler of the carotid arteries, and the results were negative. Nonetheless, a dental evaluation was not proposed in either of the headache clinics.

The presence of malocclusion made a dentist decide to try a DOA to restore the patient’s proper VDO, and nighttime wearing of the device resolved her headache. We had the fortune to evaluate the patient after the headache was resolved, which is why although the diagnosis of migraine might have seemed correct, we decided to make a new DOA. Another reason for the new device was the concomitant presence of TMJ pain, masseter muscle fatigue and soreness, together with the report of nighttime tooth-clenching activity by the patient. We changed the thickness of the device because, to our judgment, the previous one excessively increased the patient’s VDO, and we eliminated all tooth contact other than the ones between the upper and lower incisors, to maximally reduce clenching activity.

The results obtained were very positive. Both headache and aura symptoms resolved, TMJ pain subsided completely, and masseter muscle fatigue decreased and became episodic with no further need of treatment.

In light of the results of this single case, correction of dental occlusion and protection of the structures of the stomatognatic system from the stress of tooth clenching seems to be useful to eliminate migraine headache.

Of course, no conclusions can be drawn from a single case; however, in the literature, similar cases have been reported. In the two studies of Lamey et al., migraine patients were treated by the means of acrylic DOAs. In the first study, 19 patients with migraine with aura (classical migraine) with headache attacks occurring more frequently on waking were selected. Therapy consisted of the use of a DOA to wear during sleep that produced a good clinical response with considerable reduction of the intensity and the frequency of the headaches. The second study was carried out with a placebo-controlled design. Two different appliances were made: an effective DOA with tooth coverage, and a placebo DOA without tooth coverage that only contacted the palatal mucosa. The placebo DOA did not produce any change in the subjects’ dental occlusion. Only migraine patients who used the DOA with tooth coverage reported significant reduction of the number of headache attacks to about 40 percent of that previously experienced. Intensity of headache was not evaluated.

In a study by Quayle et al., 57 patients suffering from different types of headaches were selected and treated with a soft DOA to wear at night during sleep. Among them, the subjects diagnosed with migraine reported marked improvement or complete relief of headache symptoms; on the other hand, subjects diagnosed with tension-type headache did not benefit from the therapy. Unexpectedly,

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Forsell et al. describe the opposite outcome. They treated 35 patients with migraine, 36 patients with tension-type headache associated with pericranial tenderness (muscle contraction headache), and 20 patients with both migraine and tension-type headache (combination headache), performing occlusal adjustments to surfaces of the teeth. The experiment consisted of a placebo-controlled double-blind trial, where effective occlusal adjustment was carried out by adjusting the occlusal surfaces of the teeth to give bilateral simultaneous contacts between the upper and lower teeth; conversely, placebo occlusal adjustment was performed by adjusting the surface of the teeth that do not come in contact with the antagonist teeth, and therefore without changing dental occlusion. Differently from the Quayle study, Forsell et al. found the frequency and intensity of headache was reduced in 79 percent and 53 percent, respectively, of the patients affected by tension-type headache and both migraine and tension-type headache. No difference was detected between placebo and effective occlusal adjustment for migraine patients.

Interestingly, abnormalities of dental occlusion were not more prevalent in migraine patients, according to Steele et al., but TMJ and masticatory muscle tenderness—together with tooth-clenching and grinding habits—were found more frequently in migraine patients, with two-thirds reporting dental parafunctions.

The hypothesis that dental parafunction, especially tooth clenching, can be a precipitating factor for migraine was studied by Shankland, who reported 62 percent reduction of migraine episodes using a device fitting in the upper incisors and contacting only the lower incisors, keeping the remaining teeth apart. Such a device had to be worn during sleep and had the intent of reducing muscular activity. Shankland’s hypothesis seems to be confirmed by Lamé et al.’s findings that display how masseter and lateral pterygoid muscles were 70 percent larger in migraine patients compared with a control group. Also, maximal bite force and EMG activity of the masseter and temporalis muscles during maximum voluntary contractions were found to be significantly higher in migraine patients. These findings suggest higher activity of the masticatory muscles in migraine patients, which might be parafunctional (tooth clenching) rather than functional.

The mechanism through which tooth clenching could precipitate migraine headache has not been demonstrated, but two hypotheses can be made: Tooth clenching might be a trigger for migraine attacks, increasing norepinephrine release into muscle spindles and vasoconstriction. If we accept the association between tooth clenching and stress, norepinephrine is also released by the adrenal cortex. These changes stimulate the cervical sympathetic ganglia to further produce norepinephrine, as supported by some animal studies.

Circulating and localized norepinephrine would then generate headache, according to Shankland and Anthony. A second hypothesis is suggested by the fact that muscle soreness and fatigue can produce not only localized but also referred pain. As thoroughly described by Travell and Simons, such pain can be frequently referred to the head, giving headache, and the clinical picture can mimic migraine headache. If this is true, the hypothesis is that muscle fatigue caused by malocclusion and dental parafunction produced the secondary migraine.

In both cases, use of the DOA corrected the patient’s malocclusion, restored the correct VDO, and reduced tooth clenching, with the result of decreasing masseter muscle fatigue and, consequently, headache.

Conclusion

The results reported in the literature and the outcome of the case described show how primary migraine can be confused with migraine secondary to dental malocclusion and parafunction. For this reason, dental evaluation is always advised for headache diagnosis.

References