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Review Article & Clinical Report

Medication Overuse Headache and Myofascial Pain: Frequent Under-Diagnosed Conditions Related to Chronic Headache Patients. A Review of The Literature and Clinical Report

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Medication Overuse Headache and Myofascial Pain: Frequent Under-Diagnosed Conditions Related to Chronic Headache Patients. A Review of The Literature and Clinical Report

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Abstract

Primary headaches become chronic when contributing factors are under-diagnosed, as a medication overuse or the presence of Myofascial Pain. This case report illustrates a 40 years old male patient complaining about headaches and facial pain for 30 years. After an accurate examination, Medication overuse headache (MOH), Myofascial Pain (MFP), Local Mialgia, and joint disorders were diagnosed, followed by the establishment of a conservative treatment plan. The mechanisms and pathophysiology of MOH, as well as its possible relationship with MFP, were discussed. With basis in the case report and literature, it was concluded that chronic headache patients must be evaluated in an attempt to diagnose MOH and MFP, since both are common conditions found in these individuals. In addition, their etiologies are multifactorial and the treament plan should include the use of different conservative treatment modalities.

Keywords: Medication Overuse Headache, Myofascial Pain, Caffeine, Chronic Headaches, Temporomandibular Disorders

Introduction

Medication overuse headache (MOH), according to the International Classification of Headache Disorders[1], is the preferred name for a particular headache type, described typically as headache that is present daily or almost daily (15 or more days of each month) of varying intensity, associated with excessive use of simple analgesics (for 15 or more days per month) or any other migraine abortive medication (such as triptanes, ergotamines, opioids, and caffeine/codeine-containing combined analgesics, for 10 or more days per month), for at least three consecutive months. The MOH is a secondary headache syndrome that results from an underlying primary one, such as migraine or tension type headache (TTH). Thus, the clinical features of the MOH depend on the specific characteristics of each of these primary headaches. Frequently, MOH is associated with considerable disability, its prevalence has been increasing worldwide, with higher proportions among women, and the peak prevalence by age is reached in the middle of the fifth decade.[2,3]

The pathophysiology of MOH is still not completely understood. The misuse of pain relief medications has been proposed as an etiologic mechanism.[2-6] In fact, the frequent use of acute medications to manage headaches may lead to the progression of pain from an episodic disorder to a chronic condition.[6] When triptans and opioids are overused, MOH can be triggered faster. However, the abuse of any kind of the above-mentioned drugs can lead to this condition.[2,4] In this context, the type of medication overused, as well as its frequency of use, may influence the risk of headache chronification and the duration of withdrawal symptoms during treatment. These features, together with the unique and selective vulnerability of migraine patients to these phenomena, highlights the possibility that the mechanism(s) by which acute medications lead to this progression may differ depending on the particular drug, and may be similar to the underlying biology of acute attacks of migraine.[5] It has also been suggested that the oversensitivity to the medications taken for the acute treatment of headache attacks may have a genetic basis, making some people more susceptible to this condition.[3] Other potential mechanisms for the development of MOH would include an increase in evoked cortical spreading depression (CSD), a possible role for neurogenic inflammation, peripheral and central sensitization and descending facilitation.[5] A more throughout examination comparing the effect of different classes of medication on each of these factors could provide further insight into the pathophysiological mechanisms of both MOH and migraine.

Despite the overuse of pain relief medications, the abuse of some substances such as caffeine, has also been proposed as an etiologic factor for MOH. Caffeine may be consumed

daily in the form of coffee, tea, cocoa, chocolate, energy drinks, as well as many painkillers and anti-headache medications. Some authors suggested that caffeine overuse could cause chemical dependence and in some cases, increase the burden of migraine headaches and/or lead to migraine chronification.[7-10] This could be explained by the fact that sudden caffeine withdrawal would trigger migraine attacks.[9,11] It is also important to highlight that the misuse of analgesics medications containing caffeine may have play an additional negative role in the treatment of MOH patients, especially in those who were originally suffering from episodic headaches.[6] Thus, the relationship between caffeine and the etiology of headache is complex, controversial, and still misunderstood.

Some authors have pointed out the etiological relationship of chronic headaches with myofascial pain (MFP).[12,13] MFP is a regional neuromuscular disorder with a high prevalence in patients with chronic pain, including headaches.[12,13] This disorder results from hyperalgesic trigger points (TrPs), which have been characterized as hyperirritable points located within a taut band of skeletal muscle or fascia.[12-14] The referred pain pattern from these hyperalgesic TrPs in head and neck muscles produces pain features that are usually found in patients suffering from chronic headaches.[12,13] In fact, there are clinical evidence demonstrating the that the referred pain elicited by TrPs reproduced both TTH and migraine attacks.[12] It was also verified that MFP is associated with chronic headaches in aproximately 75% of these patients.[12] Since this neuromuscular disorder is extremely debilitating for the patient, this could also contribute to the increased consumption of pain relief medications by chronic pain patients.

Treating the patient suffering from MOH can be challenging, especially because detoxification of medication and/or substance abused is needed. This process may be extremely uncomfortable for patients because withdrawal reactions and worsening of headache frequently occur.[6] Therefore, the use of preventive medications has been indicated to limit the intensity and frequency of the headache episodes.[3,6] Also, when associated with MOH, the diagnosis and consequent adequate management of MFP is indicated in order to increase treatment success.[12-14]

Considering the information above, this article describes a patient whose chief complaint was headache for more than 30 years. The established diagnosis, treatment plan proposed, and patient response to treatment are discussed. In addition, this article debates the mechanisms and pathophysiology of MOH and its possible relationship with MFP.

Clincal Report

A 40 years old man was referred to the Temporomandibular Disorder and Orofacial Pain Clinic of the Araraquara Dental School, Univ. Estadual Paulista-Unesp, with a history of temporal left-sided headache and facial pain for 30 years. Although there are no pictures or name that would identify the referred patient, informed consent document was obtained authorizing the use of his health records.

The pain complaint was first noticed during childhood and happened on a daily basis, when he woke up in the morning or shortly thereafter, with a 40 to 50 minutes duration. Its intensity was moderate with a pulsating quality. Phonophobia, photophobia, nausea, and vomiting were not associated. The pain was aggravated by masticatory function or during sleep and relieved by decreasing chewing or eating a soft diet, analgesic medication, removing denture prostheses and, in the past, caffeine consumption. The clinical characteristics of the headache episodes were: pain intensity (5, in a 0-10 pain scale), frequency (daily) and duration (around 50 minutes). He also reported clicking during mouth opening and closing and pain on the left temporomandibular joint (TMJ).

During personal interview, he reported staying home almost all day long, with no exercising or working, low water intake, extremely high consumption of coffee (approximately 3 liters a day), and daily use of analgesic medication (Acetylsalicylic acid and Paracetamol). The patient also reported to sleep 8-10 hours a day, despite frequently waking up tired. He also had low energy level and fatigue. Past medical history revealed neurological disturbance and symptoms of depression. Physical examination revealed pain during lateral palpation of the left TMJ, reproducing his pain complaint, and tenderness in masticatory muscles, with hipersensitive points associated with pain referral and local twich response, indicating the presence of TrPs. Indeed, referred pain evoked during palpation of active TrPs in the masseter and temporalis muscles could reproduce part of his headache complaints.

Based on the clinical history and physical examination, the following diagnosis were established:

1. Capsulitis and Disc Displacement with Reduction on left TMJ,
2. Local Myalgia,
3. MFP,
4. MOH.

Treatment plan

The first step of the treatment plan included detoxification, through discontinuation of all pain relief medications that he had been using on a daily basis and a gradual decrease in the consumption of coffee. The process of detoxification was carried out by explaing to the patient that his headache was probably related to the excessive use of medications and extremely high consumption of coffee.

Preventive and abortive medications for headache (different from that usually taken by the patient) were also prescribed. Preventive medication (Cyclobenzaprine) was prescribed to limit headache occurrence and severity, as well as to improve sleep quality. Cyclobenzaprine is a centrally acting skeletal muscle relaxant, closely related to tricyclic antidepressants, and its mechanism of action seems to be the inhibition of presynaptic norepinephrine/serotonin and improvement of sleep quality.[15] Patient was instructed to take 10 mg/day, 1-2 hours before bedtime, for 30 days.

Abortive medication was indicated to interrupt the headache attacks. Ibuprofen was prescribed, a nonsteroidal anti-inflammatory drug, which mechanism of action seems to be the inhibition of the rate-limiting enzyme complex cyclooxygenase [COX], resulting in the reduced synthesis of prostaglandins in the periphery.[15] The treatment of capsulitis on left TMJ included the prescription of another nonsteroidal anti-inflammatory medication – Etoricoxib, a COX-2-specific inhibitor. Patient was instructed to take a 400 mg capsule of Ibuprofen, within a 6-hours-interval, until headache completely subsided, and a 90 mg capsule of Etoricoxib/day for 7 days.

Detoxification and pharmacological therapy were combined with patient education, home care therapy, biofeedback-assisted relaxation, and behavioral modification therapy, including recognition and awareness of daytime parafunctional habits, such as keeping teeth gently together or even clenching. Patient was asked to keep his teeth gently apart, lips gently touching and jaw muscles relaxed. Stretching exercises for the masticatory muscles were recommended in combination with cold and heat therapies. He was finally instructed to engage in aerobics exercises, such as walking, riding a bike or swimming and to increase water intake. All exercises were performed in such a way that no pain was present during the activity in order to decrease the chances of Central Nervous System (CNS) stimulation.

Follow-up

The first follow-up evaluation was performed 2 weeks after the initial evaluation when patient reported improvement in the complaints in terms of head pain intensity (5 to 1, in a 0-10 scale), frequency (daily to once a week), and duration (50 to 30 minutes). The lateral palpation of the left TMJ was repeated and revealed no pain, but muscle examination still showed the presence of TrPs. The patient's cooperation was remarkable for the proposed treatment. He reported withdrawing analgesic medication, decreasing coffee to 150 ml a day, taking the medications prescribed, increasing water to 1 liter a day, performing the stretching exercises with thermic therapy daily and practicing physical activity three times a week.

Six weeks after initial evaluation patient reported no headache attacks and muscle examination failed to show the presence of either active or latent TrPs. During the next four months, patient returned for regular follow-ups and reported no headache attacks and no pain on the left TMJ or TrPs. The patient also mentioned being completely free of medication, consuming few doses of coffee, and cooperating with the treatment plan.

Discussion

Although the pathophysiology of MOH is incompletely understood, several factors have been related to its etiology, such as misuse of headache medication, central sensitization, abuse of some substances such as caffeine, psychological factors (anxiety and depression), disinhibition of pain impulses, cutaneous allodynia and intracranial hypersensitivity.[2-10,12,16] The pathophysiology of this type of headache could also be related to episodic headaches becoming chronic. In this case, the wish to relieve pain and perform daily activities normally may lead the patients with episodic headaches to misuse acute medications. The headache' episodes may also stimulate fear and anxiety related to work loss, severity of pain, and interference with social activities, for example. Thus, to reduce the fear, patients may take acute medications preemptively at the first warning of an impending headache or in anticipation of an attack.[6] Once chronic headache has been developed, patients overuse medications for pain relief and become susceptible to rise MOH. In addition, with the development of chronic pain, diferent pain receptors are activated, such as the N-methyl-D-aspartate receptors [NMDA], and the abortive medications that are usually effective for acute pain, including opioids, are not going to be totally effective anymore.

In this context, MOH is not considered as a single entity, as each class of drug overused may cause MOH via a different mechanism and with different clinical characterisctis.[3] Migraine

patients, for example, who usually overuse triptans, have reported daily headaches mimicking a migraine attack or an increase in the frequency of migraines. As the headaches become more frequent, the the pattern of characteristics (nausea, vomiting, photophobia, phonophobia) changed and the pain can become hard to distinguish from that of TTH. Those patients diagnosed with chronic TTH overusing analgesics, have reported an increase in frequency of the attacks, but the clinical features of the pressing holocranial pain do not change.[17] Thus, the diagnoses of MOH only with basis in signs and symptons reported by the patient must be difficult. It is of utmost importance to identify which class of pain relief medication has been overused.

The opioids are a particularly problematic drug class consistently associated with the worsening of headaches. Recent evidence indicates that chronic opioid administration may exacerbate pain in the long term by activating toll-like receptor-4 on glialcells, resulting in a pro-inflammatory state that manifests clinically as increased pain. Thus, from the available evidence, it seems that opioid-overuse headache is a phenomenon similar to opioid-induced hyperalgesia, which derives from a cumulative interaction between central sensitisation, due to repeated activation of nociceptive pathways by recurrent headaches and pain facilitation due to glial activation.[18] Future studies are recommended to better elucidate the link between each class of medication and the pathophysiology and clinical presentations of MOH.

There is still scientific evidence about the role that central sensitization plays in the etiology of chronic headaches[12], such as MOH. Consequently, MFP, throughout the constant peripheral nociceptive stimulation on CNS, might have an etiologic role in pain chronification, through the increase or maintenance of this central sensitization. MFP and TrPs could also be involved with headaches as the exclusive source of pain, as a contributing element, or as a precipitating factor.[12] Substances like caffeine may also contribute to the development of MOH, since this substance is considered a central stimulator.[7,16,19] The findings of Scher et al.[18] suggested that caffeine is powerfully linked to chronic daily headache underscoring its potential role in episodic-chronic transformation. The authors also suggested that dietary and medicinal caffeine consumption appears to be a modest risk factor for chronic daily headache onset.[18,20] Besides the role of caffeine in the onset and perpetuation of headaches, there is also evidence showing that its withdrawal is also associated with better efficacy of acute treatments for headaches.[10] Thus, it is recommended that patients suffering from headaches should be aware of the possible effects of this substance in the etiology of headaches.

A previous study emphasizes that patients with MOH present a higher concentration of 5-HT2 receptors in platelet membranes compared with episodic migraine patients, suggesting that this could be found in central serotonergic receptors as well, and explain the transformation of episodic migraine into MOH.[21] Srikiatkhachorn et al.[22] also suggested that the overuse of relief medication could lead to an up-regulation of central serotonergic receptors, suppressing therefore the function of serotonergic pathways involved in central pain modulation. Furthermore, Hering et al.[23] observed an increase in blood level of serotonin after withdrawal of headache medication in MOH. Platelet membrane transduction of patients with daily headache was analyzed, suggesting that the misuse of medication would result in a modification of this process, possibly playing a role in such a headache pattern modification.[23]

Another factor related to MOH is a history of previous episodes of migraine headaches. Relja et al.[7] diagnosed migraine without aura as the primary headache, pre-existing to MOH onset, in 74.3% of patients. Moreover, during MOH episodes, the migraine-like component is usually more frequent, more prolonged, and more intense than any episodic migraine which might have been experienced in previous years. In this condition, the patient misuses the medication for pain relief and this medication used for today's headaches "rebound" and cause tomorrow's pain.

A multifaceted treatment approach has been proposed by some authors, with the following objectives, generally providing to be most effective: 1. discontinuation of overuse acute medication, 2. termination of persistent headache, 3. behavioral therapy to address the underlying overuse patterns, 4. use of preventive medication to decrease intensity and frequency of headache, 5. instruction in appropriate use of acute medication.[2,3,6] The withdrawal of medication overuse may be thwarted by substantial and intolerable symptoms and the coexistence of psychiatric conditions often adds to the complexity of the situation.[2,3,6] Mathew et al.[24], studying a group of 200 patients taking daily symptomatic medications, observed initial withdrawal reactions, commonly in the first 4 days, that included nervousness, increased headache, nausea, vomiting, insomnia, diarrhea, and tremor in most of the patients. Various treatment protocols have been proposed to relief initial withdrawal reactions, including the use of repetitive intravenous [i.v.] dihydroergotamine (DHE) injections, oral prednisone, repetitive i.v. prochlorperazine, pharmacological therapy combined with biofeedback-assisted relaxation, behavioral treatment, i.v. lidocaine infusion and naratriptan.[6] In the present case report, maybe because pain duration was short [50 minutes] or because preventive medication was indicated together with withdrawn of the 2 analgesic medications and coffee, patient did not complaint of any reaction.

Also, Ibuprofen and Etoricoxib was prescribed, and this may also have affected patient's response.

In the present case report, no occlusal appliances were utilized. However, it could be indicated and important in patients with chronic headaches and TrPs localized in the masticatory muscles, such as masseter, temporalis, lateral and/or medical pterygoid. Patients respond in a different way to different kinds of treatment modalities. It is likely that some patients would benefit from the proper use of an occlusal appliance to decrease the load over the muscles when they sleep and to function as a behavioral modification appliance when used during the day.[25] It is important to note that patients should never be asked to eat with the appliance in their mouth. It is our recommendation that, in cases in which an occlusal appliance will be used, the material should be hard and not soft, the coverage should be complete and not partial, and, finally, it could be fabricated in the maxillary or mandibular arch. However, we recommend the mandibular arch simply because it will interfere less with patient's phonetics and esthetics, important considerations in instances where the patient will wear the device during the day. Despite this, there are recent studies showing that patients can and will get better with the use of medications and self-care management regardless the use of any device.[25]

It would be also important to note that most patients will need more than 30 days of preventive medications, usually 90 days. Chronic pain patients usually do not have insomnia, but rather poor sleep quality. Medications for insomnia do not work well with them. In order to improve sleep quality, medications such as the tricyclic antidepressants in very low doses, such as nortriptyline 10 mg or amitriptyline 25 mg, are the first line drugs. Cyclobenzaprine has been shown to be effective in low doses from 5-25 mg a day, taken 1 hour before bedtime for patients with MFP and associated headaches, as well as Fibromyalgia. These medications decrease the number of awakenings and arousals and provide patients with the deep stages of sleep (3 and 4 or more recently stage 3 only).[26]

Several factors need to be taken into consideration when evaluating patients with chronic pain. Based on the fast response that the patient reported here had to the treatment, we can attribute his chronic pain to a lack of adequate treatment and proper diagnosis. Possible, the main success point of the treatment was the establishment of the two etiologic contributing factors related to pain chronification: the misuse of medication/substance and the presence of TrPs. Thus, MOH and MFP should always be considered as potential contributing factors in patients with chronic headaches.

Despite the limitations of this case report, the authors were able to follow-up this patient for 1 year after symptoms had subsided. It was demonstrated that successful detoxification is necessary to ensure improvement in the headache status when treating patients with MOH. The patient must completely avoid the pain relief medication as well as excessive caffeine intake, which might be related to the MOH and central sensitization. Moreover, when it is also associated with TMD, such as MFP, the treatment protocol should also include management of this disorder. Treatment plan should be conservative and also based on behavioral modifications and self-care management. Depending on the etiologic contributing factors and the affected muscles, proper home physical therapy exercises along with increase in sleep quality, control of traumatic factors (daytime clenching and other parafunctions), and TrPs injections may be enough for their management. When TrPs would be located in the masticatory muscles and would be activated during sleep bruxism, the use of an occlusal appliance might be indicated. However, as observed here, not all patients would need a TrP injection or an occlusal appliance.

Conclusion

The combination of conservative treatment approaches for MFP, analgesics, caffeine detoxification and the use of a preventive medication for pain could be the best treatment protocol for most patients with MOH and associated MFP.

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