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Case Report

Pathological Parafunction: Diagnosis, Clinical Presentation, and Treatment Options

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Abstract

The term "parafunction" refers to nonfunctional, repetitive behaviors [1] affecting oral structures which include digit sucking, tongue thrusting, bruxism, mouth breathing and nail biting [2]. Bruxism is defined as a repetitive jaw-muscle activity characterized by clenching or grinding of the teeth and/or by bracing or thrusting of the mandible [3]. This can occur during while awake or asleep.

This paper proposes the creation of an additional term, "pathological parafunction", to describe a severe form of parafunctional behavior, and describes associated signs, symptoms, and recommended treatments. Case reports are included to demonstrate clinical examples of this condition and its response to treatment.

Introduction

Bruxism and clenching are the most common of the parafunctional activities and have a prevalence of 50-90% in the general population [4] with no significant differences between sexes [1]. These behaviors involve use of the masticatory muscles, namely the masseter and temporalis. These muscles are the primary jaw closing muscles, thus when a patient is clenching, these muscles are being used and can often be sore or tender as a result. In addition, the lateral and middle pterygoid muscles are used during excursive movements of the jaw, and are used during teeth grinding events.

Effects on TMJ

The act of clenching or biting exerts force on the temporomandibular joints (TMJ's). Pain in the TMJ or masticatory muscles is referred to as temporomandibular joint disorders (TMD's). While 90% of people engage in parafunctional behavior, only 5 to 12% of the population has TMD [5]. This suggests that not all parafunctional behaviors cause significant pain.

Evidence of parafunction includes tongue scalloping, cheek ridging, dental attrition as evidenced by wear facets on teeth, and in some cases, occlusal changes (if caused by digit sucking or tongue thrust). Occasionally, bed partners will report grinding sounds coming from the person who is bruxing [1]. These clinical symptoms are typically present in patients who engage in parafunctional behavior. In cases of severe parafunction, or pathological parafunction, patients present with even more extreme clinical manifestations including chipped, cracked, or fractured teeth, masseter hypertrophy, and/or broken oral appliances. In addition, the presence of maxillary or mandibular tori can be influenced by bruxism and significant bruxism can increase incidence of tori [6].

Contributing Factors

Several factors may contribute to parafunction including stress/anxiety, genetic predisposition, and medications/stimulants. Particularly, certain antidepressants such as selective serotonin reuptake inhibitors (SSRI's) and ADHD medications like methylphenidate can play a role in increased levels of parafunction. Excessive caffeine intake can also contribute to these symptoms.

Oral Appliances

Oral appliances are typically made to protect the oral structures from damage caused by parafunction, and to unload the TMJ to prevent or address pain. These appliances are often made out of hard acrylic, and are fabricated to withstand occlusal forces at night (not to be worn during mastication). As such, wear facets are

often seen on devices indicating areas of tooth contact. Devices, if made appropriately, have an expected lifetime of three to five years. In patients who exhibit pathological parafunction, these devices have a much shorter lifespan as they are exposed to much heavier occlusal forces. In addition, the amount of wear seen on the devices is substantially higher than seen in normal cases of parafunction, and serve as an indicator of pathological parafunction. It is not uncommon to see cracked or completely fractured devices in cases of pathological parafunction.

Consequences of pathological parafunction can include jaw pain, headaches, breaking dental work, and fractured teeth. As a result of the strong masticatory muscle contractions sustained during these behaviors, patients often present with pain in the orofacial region. Pain is often described as dull, achy, sore, and tense or tight and is often located in the masseters, temporalis, TMJ capsules, and/or frontal region. In addition, patients often have comorbid headache.

Treatment of typical parafunctional cases is optional and often includes oral appliance therapy to protect dental structures from wear. On the other hand, pathological parafunction requires treatment as pain is most often an accompanying symptom. Rather than making a single arch oral appliance which is likely to quickly break or crack, making multiple (at least three) oral appliances to be switched out nightly can help not only to extend the lifetime of the devices, but also to interfere with the feedback loop mechanism driving the parafunctional behavior. If multiple appliances are made with varying vertical dimensions, and patients switch between the devices nightly in random order, the force/pattern of masticatory muscle contraction cannot be consistent and is therefore slightly impeded by the multiple appliance method. Alternatively, a dual flat plane orthotic can be made in which patients wear both upper and lower orthotics simultaneously to prevent any tooth contact on the appliances which extends the lifetime of the appliance but does not take advantage of the confusion that is obtained with the multiple device method.

Other Treatment Options

To address the pain that inevitably accompanies pathological parafunction, injections of Onabotulinum toxin A (Botox) are very helpful in reducing the strength of the masticatory muscles and therefore reducing the parafunctional power. Placement of 10 to 20 units is advised in each muscle and injection sites should be evenly spread out throughout the body of the masseter and temporalis muscles to distribute the Botox evenly, rather than injecting into a single site. These injections should be performed at 12-week intervals and repeated as the effects will wear off around between 12 and 16 weeks.

Additionally, pharmacotherapy may be implemented in cases of severe pathological parafunction. For instance, 0.3mg clonidine 1 hour before bedtime has shown to decrease rhythmic masticatory muscle activity and thereby be useful in cases of sleep related bruxism [7]. 0.5 to 1mg clonazepam at bedtime has been shown to reduce bruxism-related motor activity [8], however long-term dependence and withdrawal upon cessation is a drawback to consider before initiating treatment. Buspirone is another option to address sleep related bruxism, particularly when it is induced or exacerbated by SSRI medications. If patients are unable to tolerate dose reductions or elimination of SSRI's, the addition of buspirone, in the anxiolytic class, at doses of 15-30mg daily, can help alleviate SSRI induced bruxism through its partial agonism of 5HT1A receptors [9].

Case Reports

The following case reports demonstrate patients with pathological parafunction who responded positively to treatment.

A 66 year old female presented to the clinic for bilateral jaw pain and tinnitus. She reported a long history of bruxism induced dental fractures and had been wearing an upper and lower arch oral appliance fabricated by her dentist. She presented with bilateral masseter hypertrophy and constant, aching pain in bilateral masseter. Her tinnitus was bilateral and she reported increased jaw pain and tinnitus by the end of the day. She was taking escitalopram 10-30mg qd for anxiety. Upon clinical examination, she had bilateral tongue scalloping, bilateral mandibular tori and bilateral masseter hypertrophy. She had pain upon palpation of bilateral masseter, temporalis, suboccipital muscles, and sternocleidomastoids.

A trial of buspirone 15 mg was initiated to be taken once daily at bedtime to address suspected SSRI-induced bruxism. Botox was injected in bilateral masticatory muscles and associated muscles of facial tension/expression for a total of 200 units distributed amongst 24 sites in the masseter, temporalis, procerus, corrugator supercillii, auricularis superior, auricularis posterior, and temporoparietalis. After Botox took effect, the patient reported a 50-70% decrease in tinnitus and significant reduction in jaw discomfort. At the time, she was in the midst of dental treatment to restore fractured crowns. Upon completion of dental treatment, custom maxillary and mandibular orthotics were fabricated to be worn together and she reported no further dental fractures secondary to bruxism.

Currently, patient's symptoms remain well managed with regimen of Botox injections every 12 weeks and continued orthotic use. She reports increase in jaw tension/tinnitus in absence of Botox at 12 week intervals. Her tinnitus is intermittent and she reports less impact on her hearing and quality of life. She has not fractured any teeth in the past 4 years since initiating Botox treatment. She has switched between different SSRI medications to address her anxiety and continues taking 15-30mg buspirone per day. She reports 80% improvement in jaw pain/bruxism and 100% improvement in dental fractures since initiating treatments. Lately, she has not had any tinnitus in last 6 months.

A 36 year old female presented to the clinic for a consultation of jaw pain and significant bruxism. She had bilateral masseter pain, which was worse upon awakening, and reported significant daytime and nighttime parafunction which she attributed to her high stress job. She had worn several oral devices over the years and wore through them quickly due to parafunction. She endorsed headaches triggered by jaw pain.

Upon examination she had pain upon palpation of bilateral

TMJ capsules, masseter, trapezius, and suboccipital muscles. Her mandibular range of motion was within normal limits (50mm active opening, 52mm passive opening) and she had bilateral TMJ clicking. She had generalized dental attrition, bilateral cheek ridging and tongue scalloping, and grade 4 tonsils.

A trial of tizanidine 4 mg was initiated to be taken nightly to address jaw pain. 3 flat plane orthotics were fabricated at varying thicknesses and patient wore them at night and alternated randomly between devices as advised. Botox was injected in the same protocol as above and patient reported reduction in clenching and pain as a result. She continued to get Botox injections every 12 weeks as part of care plan. A trial of clonidine 0.3mg qhs was initiated to be taken nightly to reduce bruxism which she continues at present and finds it helpful.

Within 18 months of receiving multiple orthotics she already had significant wear (holes) in some of the devices. A dual flat plane orthotic was discussed which would be worn instead of single arch appliances to extend the lifetime of the devices by eliminating direct tooth contact on the device. Patient has not yet gotten this device but continues to manage symptoms with Botox and clonidine.

These cases serve as prime examples of the clinical presentation of patients exhibiting pathological parafunction and their response to appropriate treatments.

Conclusion

In conclusion, typical parafunction cases are easily distinguishable from more severe cases which authors propose to call pathological parafunction. These extreme cases of parafunction have dire consequences on oral structures and will require careful monitoring and preventive treatment. Oral appliances, Botox injections, and pharmacotherapy are treatment modalities to consider when encountering such exaggerated forms of parafunction. In doing so, providers can prevent damage to oral structures and the formation of painful conditions.

Acknowledgement

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Conflict of Interest

No Conflict of Interest.

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