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## CASE REPORT

# Methylphenidate-Induced Sleep Bruxism Unresponsive to Adjunctive Guanfacine Rapidly Treated with Once Daily Buspirone in an 8 years old Girl

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#### Abstract

Attention deficit hyperactivity disorder (ADHD) is characterized by impairing inattention, hyperactivity, and impulsivity. Stimulants, including methylphenidate (MPH) are commonly used for its management. MPH has been previously associated with both sleep bruxism (SB) and awake bruxism (AB) in high doses. Here we report emergence of SB at a low dose of sustained release MPH which was unresponsive to guanfacine but rapidly responded to buspirone (BUS).

Keywords: Bruxism, Attention Deficit Hyperactivity Disorder, Side effect, Buspirone, Methylphenidate

#### INTRODUCTION

Bruxism is a repetitive masticatory muscle activity characterized by clenching/ grinding of the teeth resulting in head, neck and facial pain, damage to temporomandibular joint and wear in dentition. It is divided into two types: sleep bruxism and awake bruxism. SB is involuntary and classified as a sleeprelated movement disorder while AB is semi-voluntary with awareness of clenching (1). The prevalence of SB in children reported in studies varies widely. In addition, SB has been shown to be more common among children compared to adults (2), with the prevelance rates ranging from 13% to %49 (3).

Central dopaminergic (DA) dysfunction has been postulated (4). It is commonly associated with dental problems (e.g., temporomandibular joint dysfunction), stress, other parasomnias, gastroesophageal reflux disease, and also psychotropic medications. These medications can cause bruxism by affecting the masseter's activity by direct action on DA pathways or indirectly through 5-hydroxytryptamine/serotonin (5HT) pathways (5).

Methylphenidate (MPH) is a stimulant agent used for the management of attention deficit/ hyperactivity disorder (ADHD) and its most common adverse effects were listed as insomnia, headache, stomachache, and reduced appetite (6). MPH has been previously associated with both SB and AB in high doses (1). Studies have reported successful treatment of sleep bruxism with buspirone in children, with cases of a 6-year-old girl and a 7-year-old boy showing disappearance of sleep bruxism symptoms with buspirone treatment (7, 8). Additionally, buspirone has been found to be effective in rapidly treating fluoxetine-induced sleep bruxism in a 6-year-old girl (7). The addition of buspirone may be an effective option to alleviating bruxism, especially in patients who cannot tolerate dose reduction or discontinuation (9).

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### CASE

An 8-year-old girl who was attending second grade was referred to the clinic with complaints such as failure in the ability to sit quietly, maintain attention, stop impulsive behaviors, make plans, follow directions and interact appropriately with other children. These symptoms caused the patient's dysfunction in school, home and social life. She was adopted while she was 2 years old by her parents and inattentiveness, hyperactivity and impulsivity were present since preschool. She had complaints such as aggressive behavior, sleep problems, including resistance to falling asleep, activity during sleep, waking up early, poor peer relations associated with hyperactivity, impulsivity and attention problems in the preschool period. However, he was not admitted to any clinic during this period. Physical examinations and laboratory evaluations were normal. The patient's height was 123 cm and weight was 25 kg, which were found to be within the normal range compared to his peers. The case was evaluated and Atila Turgay DSM-IV-Based Child and Adolescent Behavior Disorders Screening and Rating Scale parent and teacher forms and teacher information form were given. As a result of the scale, the attention deficit assessment area was 6/9, the hyperactivity assessment area was 7/9, and the behavior problems area was 3/8. She was diagnosed with ADHD – combined presentation in accordance with the DSM-5 criteria (10) based on clinical evaluations and parents'/ teachers' reports. She was prescribed MPH IR 10 mg/ day for a fortnight with minimal benefit and MPH XR 10 mg/ day thereafter. Parents reported moderate clinical improvement after three weeks. However, problems with inattention persisted while the parents' reported emergence of biting pencils, pens and nail biting which did not lead to dysfunction. Behavioral recommendations were made for these newly emerging parafunctional behaviors. OROS MPH 18 mg/ day was prescribed at the fifth week which immediately led to SB which was at such severity to awaken the parents and to lead to jaw stiffness. The patient complained of severe jaw pain in the morning. Treatment options including reduction in MPH dose, discontinuation of MPH, adjunctive use of guanfacine extended release (GXR) 1 mg/ day were discussed with the family and the patient. Among the treatment options offered to the patient, the patient and his family preferred the addition of guanfacine in addition to the current treatment. guanfacine ER 1 mg/g once a day was added and weekly titrations were adjusted to 4 mg/day in the 4th week.

however, although there was a significant improvement in ADHD symptoms, the complaint of teeth clenching continued. The family did not want to discontinue MPH as they were concerned about the loss of well-being achieved with methylphenidate treatment. Guanfacine was discontinued and buspirone 5 mg once daily at night was added in addition to methylphenidate. The patient's complaints dramatically disappeared completely on the second day. The patient did not experience common side or adverse effects of buspirone as reported by the FDA, such as dizziness, headache, nausea, nervousness, lightheadedness, and excitement. After one month of the combination of buspirone and MPH treatment, buspirone was discontinued and MPH treatment was continued without dose change. The patient's complaint of bruxism did not recur during the follow-up period.

#### DISCUSSION

Bruxism, a condition characterized by teeth grinding or clenching, has been found to be more prevalent in children with ADHD (11). This is consistent with the observation that children with ADHD are more vulnerable to suffer from bruxism and dental trauma due to their hyperactivity (11,12). Furthermore, children with ADHD have been found to have poorer oral hygiene and more adverse oral-health attitudes and behaviors compared to children without ADHD (12). Studies have also shown that children with ADHD have significantly higher decayed, missing, or filled surfaces (DMFS) and decayed surfaces (DS) compared to controls (13). Additionally, children with ADHD have been found to exhibit a higher prevalence of dental caries and poor oral health-related quality of life (14). When treating children who are already predisposed to such conditions due to their illnesses, it is essential to quickly recognize the side effects that may be caused by the medications they are taking and treat them with effective management.

The causes of bruxism are poorly understood and it is considered to have a multifactorial etiology (15). Defectiveness of the central dopaminergic neurotransmitter systems, especially in the mesocortical tract, has been usually blamed for the etiopathogenesis of bruxism (16,17). The inhibition of noradrenergic neuronal activity in the locus coeruleus has been shown to decrease dopamine synthesis in the striatum and to regularize the firing rate, also explained as increased synaptic efficacy (18). Alpha-2 adrenergic agonists may blunt stress-induced increases in dopaminergic neuronal activity in the medial prefrontal cortex (19). Guanfacine like other alpha-2 adrenergic agonists reduces norepinephrine release from the locus coeruleus, which could prevent orexin activation of sensory and motor neurons controlling jaw-elevator muscles involved in mastication (20, 21). Finally, alpha-2 adrenergic agonists is also known to reduce the amplitude of the masseteric jaw-closure reflex and the digastric motoneural discharge, in the jaw-opening reflex, during intraoral stimulation (22,23). However, because the noradrenergic neuronal pathway is an integrative part of a larger network of interconnecting pathways, it is probable that other systems will also be indirectly influenced. Each patient is unique and bruxism may be associated with some or all of these factors.

MPH was previously reported to be associated with both AB and SB while alpha-2 adrenergic agonists and BUS were previously reported to alleviate it (1,24). Here we report a case of SB which emerged after MPH and refractory to GXR responding to BUS. Our case underlines the importance of MPH in emergence of SB and the beneficial effects of BUS.

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