Review Article

Sleep related bruxism—comprehensive review of the literature based on a rare case presentation

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Contributions: (I) Conception and design: All authors; (II) Administrative support: All authors; (III) Provision of study materials or patients: All authors; (IV) Collection and assembly of data: All authors; (V) Data analysis and interpretation: All authors; (VI) Manuscript writing: All authors; (VII) Final approval of manuscript: All authors.

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Background and Objective: To perform a comprehensive review of the literature on sleep related bruxism (SRB) based on the presentation of a rare case. SRB is currently classified by the American Academy of Sleep Medicine (AASM) as a sleep related movement disorder. Multiple etiological factors have been reported as contributing to the development of this disorder. Genetics may be one of them. We describe the literature on this entity as related to an unusual case of the disorder being transmitted over four generations in a family.

Methods: A comprehensive history and clinical features in the patient, as well as the possible genetic factors were delineated. A thorough literature search on the topic was performed based on PubMed, Web of Science, Embase, Scopus, Medline.

Key Content and Findings: The SRB in this family has been transmitted through the four generations; however, one of the twins in the fourth generation is exhibiting symptoms. The sensory, autonomic, and motor mechanisms involved in the pathophysiology and presentation of SRB is currently poorly understood. We attempt to elucidate the known mechanisms and explore the possible neurophysiological pathways as related to this entity.

Conclusions: SRB can have a strong genetic predisposition. The reason for the entity to skip one of the twins in the fourth generation is unknown.

Keywords: Bruxism; sleep disorder; tooth grinding; genetics; pediatric

Received: 10 October 2021; Accepted: 15 July 2022; Published online: 08 September 2022.

doi: 10.21037/fomm-21-102

View this article at: https://dx.doi.org/10.21037/fomm-21-102

Introduction

Sleep related bruxism (SRB) is an entity that has been a popular topic of debate in the dental and medical community in the recent several decades. While the American Academy of Sleep Medicine (AASM) has classified SRB as a sleep-related movement disorder (1), there is recent literature refuting this, and even classifying it as normal (2). Broadly, it is considered to differ from the wake bruxism by the fact that the latter is a "habit", and therefore could be potentially controlled by interventional habit controlling methods (2). Attempts have been made to classify bruxism according to the various factors such

as diurnal variation (wake and sleep-related), presence or absence of mandibular movements (static and dynamic), and etiology (primary and secondary) (3). It is of interest to the medical and dental community, both with reference to the probable abnormal nature of these movements during sleep, and due to the proposed destruction of the dentition, the temporomandibular joints (TMJs), and dental restorations. In addition, the observed association of SRB with other sleep related movement disorders, and possible medical comorbidities is of immense interest to the medical profession (4).

Sustained jaw clenching and/or repeated mandibular movements are hallmarks of SRB (5). The diagnostic criteria that are followed by various medical specialties vary vastly, and the current absolute lack of consensus amongst clinicians and researchers, has added to the existing confusion regarding the classification, diagnosis, and management modalities of this sleep related movement disorder. A diagnosis of SRB made based on patient, bed partner, or parental report is probably largely inaccurate. The dental clinician traditionally has resorted to such reports in combination with the observed changes in the dentition in an attempt to come to a diagnosis. An overnight polysomnography (PSG), although once considered the golden standard in the diagnosis of SRB, has been questioned in terms of the necessity for employment in a patient suspected of the same (1). The pathophysiology of SRB is unknown, with certain speculations and hypotheses floated in the literature. Recent attempts to include SRB as a "normal" or "protective" phenomenon has added more chaos to the already crowded arena of hypotheses (6). Consequently, there are no standardized management modalities either. It is the opinion of the authors that SRB should be looked at in the context of a sleep related movement disorder, and further succinct scientific studies are paramount in elucidating the mystery of this phenomenon. In this manuscript, we describe an interesting case of SRB, which has apparently traversed four generations. Here, we look at SRB as a probable movement disorder, shed light into the possible motor pathways, and discuss the genetic factors involved in SRB.

Methods

The search for this narrative review was performed between January 1st, 2021, and July 14th, 2022. The databases searched included PubMed, Google Scholar, Ovid, Embase, Science Direct, and textbooks on sleep disorders. The search terms included bruxism, sleep related bruxism, awake bruxism, sleep related movement disorder, pediatric bruxism, motor pathways of mastication, mandibular movements, rhythmic masticatory muscle activity (RMMA). The complete articles written in the English language were retrieved. Only articles published in English between 1970 and 2022 were included. Exclusion criteria included articles other than in English; articles published prior to 1970 and articles whose complete published form were not available.

The case

Twelve-year-old Indian fraternal twin girls presented along with parents with a chief complaint of "grinding her teeth in sleep at nighttime" in one of the twins.

History of presenting illness

According to a parental report, the affected child started SRB at an early age of 8 to 9 years. The child reportedly had bruxism episodes consistently "almost every night". The episodes were reported as "bursts". Most of the episodes were of a dynamic nature ("grinding") with occasional static episodes ("clenching"). The child also complained about sporadic day time facial pain episodes and painful legs.

General features

The child appeared well nourished, well groomed, was very responsive to questions and cognizant. She was articulate and well oriented in terms of time and space. She was very expressive and devoid of anxiety at the first visit, although parents had given a history of anxious nature.

Medical history

Reported medical history was normal except for stomach hyperacidity and "pes planus". Patient is on iron supplementation to manage her deficiency, which she has relatively poor compliance due to the side effects such as constipation.

Family history

The twins' father, grandfather and great grandfather have reported a history of SRB. The grandfather of the twins also had reported daytime bruxism. From the pedigree (see *Figure 1*), SRB seems to follow an autosomal dominant

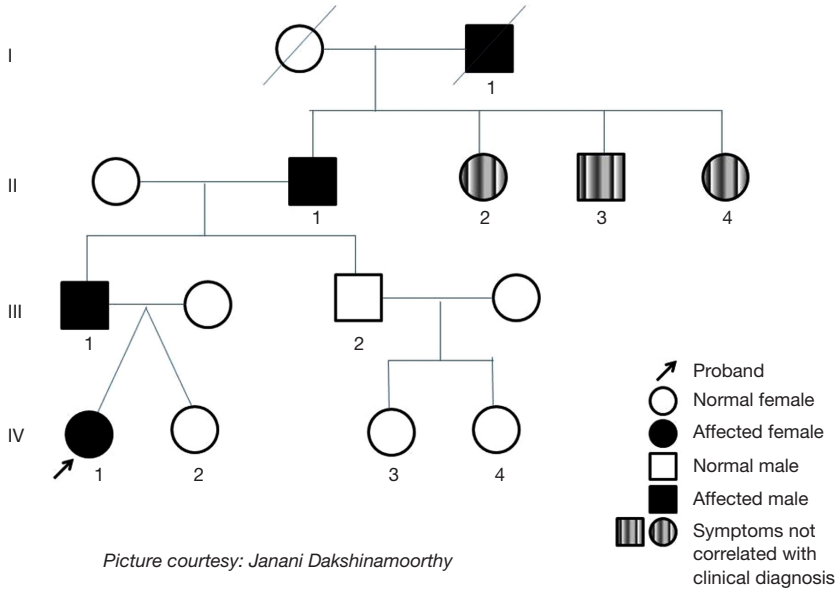

Figure 1 Pedigree of the family based on phenotypic representation of bruxism.

pattern of inheritance. It is observed that the proband (IV:1) has a father (III:1), grandfather (II:1) and a great grandfather (IV:1) who also have been diagnosed with SRB. It was also noted that the father's brother (III:2) and his lineage did not show any clinical features of SRB. The siblings (II:2, II:3 and II:4) of the grandfather (II:1) were identified to have generalized attrition indicating the possibility of SRB. However, a diagnosis of SRB by report could not be verified in these siblings. The great grandfather (I:1) was reported to have a habit of grinding his teeth at night.

The affected twin child's apparent psychological profile is very similar to her dad's, in terms of common likes for apparel, type A personality, academic excellence and extrovertedness. They also have other common likes and dislikes. The common sleep trend between the father and the affected twin child includes somniloquy. One common factor among these four generations of individuals is hyperacidity/acid reflux. The unaffected twin does not suffer from acid reflux or hyperacidity. Both father and the affected twin child have a history of anxiety.

Dental examination

A comprehensive dental examination revealed normal soft and hard tissue structures, consistent with the twins' age. There was no significant wear pattern observed in either child. TMJ and muscle examination were normal.

Investigations

An overnight PSG could not be ordered due to the fact that the child was not showing any other signs/symptoms of obstructive sleep apnea (OSA)/snoring; also due to safety concerns with the COVID-19 pandemic.

Diagnosis

The diagnosis of SRB in this case was entirely based on a parental report. There was no indication for a PSG due to the lack of evidence for snoring and OSA.

Review of literature

Definition of bruxism

SRB is defined by AASM as "an oral parafunction characterized by grinding or clenching of the teeth during sleep that is associated with an excessive (intense) sleep arousal activity" (1) and included as such in the International Classification of Sleep Disorders (3rd edition, 2014). There has been abundant discussion and confusion in the literature in defining and classifying bruxism. There are two distinct entities that have been identified namely, awake bruxism and SRB (7). Bruxism occurring in children is referred to generally as pediatric bruxism (PB). There is anecdotal evidence in the dental literature for possible

genetic association with SRB. The American Academy of Orofacial Pain (AAOP, 2008) defines bruxism as "diurnal or nocturnal parafunctional activity including clenching, bracing, gnashing, and grinding of the teeth" (8). A recent international consensus statement does not consider SRB and awake bruxism to be movement disorders (2).

Primary/idiopathic sleep bruxism

This entity has been loosely defined in the literature, with considerable variations and controversies regarding its possible etiology. There has been strong suggestion in the literature of the role of the reticular activating system, and thereby the brain stem, contributing to increased motor and autonomic nervous system activity in SRB (9). There have been suggestions that SRB may be a result of improper processing of feedback from peripheral tissues such as teeth and the surrounding structures (10).

Secondary sleep bruxism

A class of drugs called selective serotonin reuptake inhibitors (SSRIs) have been implicated in the literature as etiologically related to secondary bruxism (11-16). Some recent articles, doing systematic reviews have concluded that a few medications such as duloxetine, venlafaxine, paroxetine, barbiturates may be related to higher risks of SRB (17). However, contrary to other literature, these authors reported lack of association of SRB with some SSRI medications such as citalopram, escitalopram, fluoxetine, sertraline. That said, it seems clear that drugs acting on the dopaminergic system promote awake bruxism in human patients (18,19).

Pediatric SRB

By definition, this entity occurs in children. There is no consensus in the current literature as to the diagnostic criteria or management of PB. Our experience is that explanations given to patients and parents by dental professionals (including teaching institutions) have not been based on rigorous scientific research. These explanations range from "because of mixed dentition", "psychology" to "habit" to "intestinal worms/parasites" to "chronic abdominal distress", and more. Recent literature has started looking at risk factors involved in bruxism in children. In these studies, risk factors were extremely varied, rendering a firm consensus challenging. Identified risk factors included male gender, genetics, anxiety, secondhand smoking, snoring, sleeplessness, and headache (20,21). Other studies have pointed to the significance of childhood stress in the etiopathogenesis of pediatric sleep bruxism (22).

Synonyms

Tooth grinding; clenching.

Epidemiology

The prevalence of PB is not well understood. Recent literatures including systematic reviews have reported the regional prevalence of PB (23,24). The highest reported prevalences in the descending order were Finland (40%), United States (37%), Brazil (35%), and Hong Kong (6%) (23,25). Studies show contradictory results, as to the association of bruxism with age (26-29). One of the reasons for the vastly varied findings in the prevalence rates is the differences and inconsistencies in diagnosis, primarily due to lack of universal diagnostic criteria for PB (25). Moreover, inconsistencies and errors in various parental report forms may be skewing the results (27,28,30). Studies from Brazil found high prevalence of PB in school children, with children having a habit of nail biting and biting objects at a possible higher risk of having PB (31,32). Gender differences in the prevalence of PB have been reported to be higher in males than females (30). Further, the same authors found a reduction in prevalence in both genders with age. Multiple studies over the last decade have resulted in diagonally opposite conclusions as to the incidence and prevalence, and association of PB with gender and age (27,28,30,33). A recent study shows the prevalence of PB as approximately 28% (34). The prevalence of sleep bruxism in adults was reported to be 9% in the general population, and as in children, adults also show no gender difference in SRB, and the prevalence reduces with age (35,36). A systematic review of epidemiology of adult bruxism shows rather inconclusive results. It was found to have a wide range of 8–31% for clinically identified bruxism, 22–31% for awake bruxism and approximately 13% for bruxism by report (36).

Etiology/pathophysiology

As the name indicates, the etiology of primary bruxism is not clearly understood. Secondary bruxism is associated with certain systemic diseases/or medications (37-39). The pathogenesis of bruxism is hypothesized to be multifactorial. Association of SRB with systemic disorders, sleep disordered breathing, restless leg syndrome, gastric reflux,

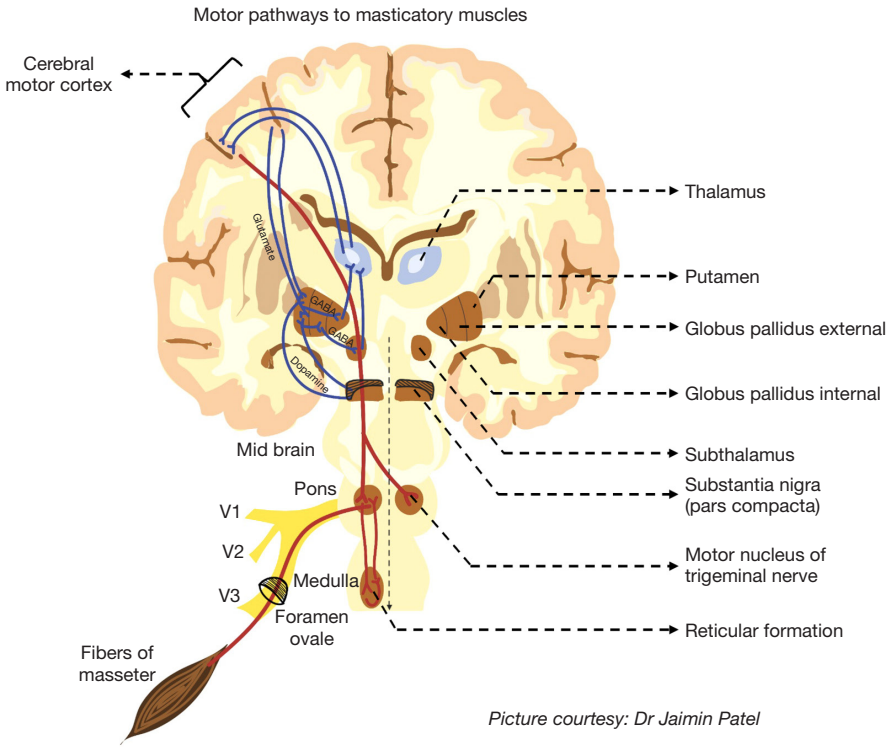

Figure 2 Motor pathways to masticatory muscles.

and neurologic disorders, have been suggested (40,41). Although not clear in terms of association, increased urinary catecholamine levels were found in SRB patients, compared to normal individuals (42). The trigeminocardiac reflex (TCR) has recently been linked to various sleep disorders like OSA, SRB and rapid eye movement (REM) related sleep apnea (43).

Proposed motor pathways (in the context of movement disorder classification)

The motor pathways (see *Figure 2*) for volitional movements are widely considered to start from the pyramidal cells of the motor cortex (areas 4 & 6) (44). The role of the basal nuclei, their associated structures, and the related dopaminergic, glutamatergic and gabaergic pathways, in volitional movements, is well known (45,46). Diseases that cause abnormal functioning of these systems (such as parkinsonism) have been known to cause significant movement disorders such as dyskinesias, dystonias, athetosis, chorea and others (47,48). The critical role of the "direct and indirect" motor pathways in performing fine motor movements have also been elucidated (46,49). Consequently, the role of these centers, corticobulbar tracts and associated structures in normal mandibular movements, such as those involved in mastication, phonation, and swallowing are also well understood (50,51). However, the generation of mandibular movements that seemingly simulate these kinetics, such as that occur during SRB are not well understood (9,52). Similarly, the pathogenesis of REM sleep behavior disorders and associated abnormal movements are also poorly understood (53,54). Specifically, the lack of REM-related skeletal muscle atonia, occurring in REM-related bruxism is not understood as well (53).

A role of dopaminergic system in the pathophysiology of SRB has been suggested (55). This concept has been shown in animal models employing dopamine receptor agonists and antagonists (18,19). In line with this hypothesis, is the finding that striatal lesions of movement disorders such as Huntington's disease show consistency with the level of bruxism in these patients (18,56). There have been suggestions in the literature that SRB-related movements may not originate from the motor cortex (2). With regards to the etiology of SRB, abnormalities in the central sensory motor processing have been proposed to be the cause of SRB (9). Central structures including, but not limited

to, the locus coeruleus, the reticular formation, and the raphe magnum nuclei, have been shown to influence this type of sensory motor processing (57). The involvement of the brainstem neurons, by virtue of their effect on descending cortical pathways, has also been suggested in the pathogenesis of SRB (9).

Risk factors and associated conditions of SRB

Bruxism has been reported to be associated with the use of alcohol, tobacco and caffeine (38). SRB is associated with apnea and hypopnea related arousals, and the termination of apneas and hypopneas. OSA is considered a risk factor for SRB (58). Association of bruxism with such rare syndromes as Pallister-Killian syndrome and the associated genotypes have been reported (59). Other risk factors reported as associated with bruxism include poor sleep, prone sleep posture (60), snoring, and mouth breathing (34). Studies suggest that secondhand smoking and sleep disturbances have the strongest association with SRB (61). The same authors found weak associations of occlusal variations, with SRB (62). Further, they also found the history of childhood bruxism, gastro-esophageal reflux disease (GERD), and genetic polymorphisms to be the most important risk factors linked with adult SRB (63). Articles have also looked at changes in the upper airway as being mostly associated with pediatric SRB (64-66). Improper sleep hygiene and a loud external environment have been proposed to be associated with SRB (64,67). Male children with the habit of object biting, nail biting and lip biting have been reported to have a strong association with severe SRB (23).

Studies show an association with psychological factors such as stress, anxiety, and personality traits (68). Some studies demonstrate the association of bruxism with sleep disorders (69). Recent studies do show an association of SRB to chronic stress or sleep quality (70). There have been isolated articles in the literature that cast a doubt on the association between pediatric SRB and anxiety traits (71). Older dental literature referred to the association of bruxism with occlusal abnormalities (72). Literature and research over the past few decades have successfully debunked this notion (57,73,74). Literature suggests that sensitivity to stress might be more important as an association, and predictive of SRB, than anxiety itself (69). There are contradicting reports of a possible association of SRB with OSA. OSA and SRB share common clinical features, and OSA therapy does improve SRB variables (75,76). The association of SRB with limb movements during sleep has been well reported (77,78). The same literature hypothesizes that the control of SRB episodes may be related to motor pathways of jaw movements and limb movements. Also, studies have shown a strong association of SRB with slow movements of the eyes, as that occurs in the N1 and N2 stages of sleep (79).

Genetics of SRB

Recent studies demonstrate a robust relationship between SRB and genetics (35). A positive association with genes that code for the serotonin and dopamine receptors, *5-HTR2A* and *DRD1* respectively, were found (80). In a recent study, there was a genomic association of the *5-HT2A* receptor nucleotide as a risk factor for sleep bruxism (81). Other articles have alluded to the association of *5-HTR2A* polymorphism as a potential risk factor for SRB (82). A recent study suggested an association between bruxism and the enzyme matrix metalloproteinase 9 (MMP-9) in both adults and children (83). Single nucleotide polymorphisms in dopamine receptors were found to be associated with bruxism and were also associated with circadian phenotypes in children (84). Recent studies suggest that bruxism is partly genetically determined, demonstrating that the chance of bruxism in children changes proportionately as the percentage of parental bruxers increases (85).

The Online Mendelian Inheritance in Man (OMIM) website classifies sleep bruxism along with parasomnias, and uses the alternative title "facio-mandibular myoclonus" (OMIM 606840). There is moderate evidence for the role of genetics as a risk factor in the causation of SRB (63,86). Studies demonstrate that neurotransmitters in the central nervous system (CNS) and their associated genes may be factors in the pathophysiology of SRB (57,68,81). The serotonin receptor encoding gene *HTR2A*, catechol-O-methyltransferase (*COMT*), and the dopamine receptor gene (*DRD1*) have been shown to be associated with SRB (80). The same study indicates increased bruxism related episodes that were related to the gene *HTR2A* homozygous mutation, as opposed to heterozygous mutation on rs6313. A possible genetic contribution as etiologically related to primary sleep bruxism was suggested in these recent studies.

Clinical features of SRB

Some of the literature describes how pain is associated with SRB and parafunctional habits (87). It has also been reported in the literature that pain or "tense feeling" is associated with SRB, and other distinct features such as TMJ noises,

stress and smoking being associated with awake bruxism (88). Fatigue of the muscles of mastication with SRB in children/adolescents (self-reported) has been reported in the literature (89). Other orofacial findings in pediatric SRB include ridges on the side of the tongue (90) and on cheek mucosa (91); dental attrition (9); hypersalivation (92) and also hypertrophic masseters (9). TMJ clicks and noises are associated with severe SRB, especially in adolescence (93). Non-carious lesions show association with SRB, compared to controls (94). Features like somniloquy and hypersalivation have been reported to be associated with SRB in children (95). Studies demonstrate a higher prevalence of degenerative TMJ disorders with SRB (96). Anecdotal reports describe the effects of SRB on the dental structures, such as attrition, loss of tooth structure, chipping of enamel, increased tooth sensitivity, and "enamel craze lines", tooth fractures, dental restoration failures, and dental implant failures (97-100).

Diagnosis

The gold standard for qualitative and quantitative diagnosis of SRB is an overnight PSG (101,102). The latest AASM guidelines do not require a PSG for diagnosis of SRB unless there is other clinical evidence of sleep disordered breathing (103). Portable devices may be useful for the diagnosis of SRB in both research and clinical settings (104), however, further research is needed. Novel techniques include the use of sensors in bruxism appliances for monitoring SRB (105). This is an important area in the field as studies demonstrate a diagnostic discrepancy between parental reports compared to PSG (69,106).

Management

A recent systematic review found efficacy with all types of oral appliances, specifically mandibular advancement devices in reducing SRB (107,108). The same authors also report moderate efficacy for botulinum toxin, clonazepam, clonidine, and electrical stimuli of the masseter muscles.

Rapid palatal expansion

Rapid palatal expansion has been proposed as a therapy for pediatric bruxers and showed moderate reduction in the RMMA (Rhythmic Masticatory Muscle Activity) (109).

Medications

Management of concomitant stomach hyperacidity associated with SRB, has shown to be effective in reducing the frequency of RMMA and muscle activity bursts (110). The lack of effectiveness of medications in managing SRB was found in some of the literature (111). The use of botulinum toxin has been proposed as a therapy for SRB, with relatively good short-term results (112,113). However, it is not clear if the botulinum toxin therapy favorably affects the central motor pattern generator of SRB. There is recent evidence in the literature of moderately beneficial use of homeopathic medications rendering positive effects in the management of SRB (114,115). For the management of drug-induced (secondary) bruxism, discontinuation or substitution of the offending drug and possible addition of buspirone has been suggested (15).

Occlusal splints

There is controversy regarding the long-term efficacy of occlusal splints in managing SRB. While it is clear that the splint could act to protect the dentition/restorations, its role in managing SRB activity itself is unclear. The lack of consensus stems from factors such as variation in the definition and diagnostic criteria, and lack of high quality randomized prospective clinical trials. Other articles also conclude by casting doubt on any substantial benefit of splints in the management of SRB regardless of the appliance design or material (116).

Some of the literature talks about the advantage of intermittent rather than continuous use of dental splints, reducing SRB activity better than the latter (102,117). It should be borne in mind that these appliances do have the potential to induce dental occlusal changes and other TMJ changes upon long term use. A Cochrane systematic review concluded that there is no evidence of any long-term efficacy of splints in SRB management, benefiting only in the protection of dental structures (118).

Psycho-social interventions

Psychotherapeutic modalities including biofeedback and cognitive behavior therapy (CBT) have been investigated as management methods for SRB. By and large, CBT is considered one of the most effective methods for conservative management of SRB (119).

Case discussion in the context of literature

As per the parental report, the child started SRB at the age of eight to nine years. This finding is consistent with recent studies on pediatric SRB (31,32). The patient had bruxism

Table 1 Case discussion in the context of the literature

Features	Current case	As per literature
Age of onset	8–9 years old	8–10 years (31,32)
Episodes	Bursts/daily	Bursts (1)
	Dynamic bruxism	Dynamic bruxism (1,57)
Facial and leg pain	Present	Facial pain (57,89)
		Leg pain (87)
Stress/anxiety	Anxious	Stress (22,69,70)
		Anxiety (68)
Pes planus	Present	Not found in literature
Joint hypermobility	Absent	Not found in literature
Stomach hyperacidity	Present	GERD (53,63,123)
Iron deficiency	Present	Conflicting reports on association of Ferritin and SRB (124)
Family history of bruxism	Present	Reported (92,125)
Personality trait	Talkative, extroverted and Type A personality	Reported (68,126)
Concomitant sleep disorders	Somniloquy	Somniloquy (57)
		Hypersalivation (91)
		OSA (58,76,127)
Tooth fracture or wear	Absent	Reported (97,128,129)
TMJ pain	Absent	Reported (87,130,131)
Polysomnography	Not done	Most confirmatory test for diagnosis of SRB (101,102)
Bowel habits	Constipation	Not found in literature
Socio economic status	Upper strata of society	Upper strata of society (28,132-134)

TMJ, temporomandibular joint; GERD, gastro-esophageal reflux disease; SRB, sleep related bruxism; OSA, obstructive sleep apnea.

episodes that were "grinding" and occasionally "clenching" in nature. This finding is consistent with the definition of bruxism by AASM (1). The child complained of sporadic day time facial pain episodes and painful legs. Some literature describes the association of pain with SRB and parafunctional habits (87). As alluded to earlier, pain/"tense feeling" is associated with SRB (88). Also, association of fatigue of jaw muscles with SRB in children/adolescents (self-reported) has been reported in the literature (89). TMJ clicks/noises have been reported to be associated with relatively severe SRB, especially in adolescence (93).

Contrary to some reports (20), the affected twin in our case was articulate and well oriented in terms of time and space. She was very expressive and devoid of anxiety at the first visit, although parents had given a history of anxious nature. Our case findings were not consistent with a few prior studies (60,120), where SRB was associated with tooth caries, dental attrition, malocclusion and respiratory issues, as well as economic status and use of pacifiers. Jaw muscle fatigue was consistent with the literature (89). The child was not having any difficulties in mouth opening, which was not consistent with literature (121). Breathing problems, usually reported in the literature as associated with SRB (34,122), were not present in our case. Sleep disturbances presented the strongest association with SRB (62), but not in our case. There are several features in the medical history of this child that are both consistent and inconsistent with the established literature. The comparison of the medical history features of the present case to the existing literature is given in *Table 1*.

Conclusions

SRB is a sleep related movement disorder which is centrally

generated, and may have a significant genetic component. The pathophysiology remains largely unknown. The theories and apparent consensus on this phenomenon seem to be largely inadequate to explain the various aspects of this entity. Dental occlusion has been ruled out as a factor in the pathogenesis of this condition. High quality randomized controlled trials are needed to evaluate the various management modalities that currently exist for SRB.

Acknowledgments

The authors acknowledge the contribution of Swetha Kannan towards the digitalization of Figure 2.
Funding: None.

Footnote

Provenance and Peer Review: This article was commissioned by the editorial office, *Frontiers of Oral and Maxillofacial Medicine* for the series "Orofacial Pain". The article has undergone external peer review.

Conflicts of Interest: All authors have completed the ICMJE uniform disclosure form (available at https://fomm.amegroups.com/article/view/10.21037/fomm-21-102/coif). The series "Orofacial Pain" was commissioned by the editorial office without any funding or sponsorship. DCT served as the unpaid Guest Editor of the series. The authors have no other conflicts of interest to declare.

Ethical Statement: The authors are accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.

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doi: 10.21037/fomm-21-102
Cite this article as: Thomas DC, Patel J, Kumar SS, Dakshinamoorthy J, Greenstein Y, Ravindran HK, Pitchumani PK. Sleep related bruxism—comprehensive review of the literature based on a rare case presentation. Front Oral Maxillofac Med 2024;6:3.