Antidepressant-induced bruxism: need for buspirone?

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Bruxism is an involuntary grinding of the teeth which can occur during daytime or night. An estimated 85–90% of the population brux at some time during their lives, but it is clinically significant in only 5% of them. In such cases it can result in significant periodontal damage and can produce significant sleep disturbance in the bed partner of the person (Moore et al., 2000). Reports of SSRI-induced bruxism have appeared in the literature (Bostwick and Jaffee, 1999; Ellison and Stanziani, 1993; Wise, 2001). Recently venlafaxine has also been reported to cause bruxism (Jaffee and Bostwick, 2000; Pavlovic, 2004). It has been hypothesized that serotonergic drugs mediate excessive serotonergic action on mesocortical neurons arising from the ventral tegmental area leading to dopaminergic deficit which in turn causes akathisia-like movement of jaw muscles leading to bruxism (Bostwick and Jaffee, 1999). Buspirone a partial agonist at 5-HT$_{1A}$ receptors has been found to improve bruxism caused by SSRIs and venlafaxine (Bostwick and Jaffee, 1999; Jaffee and Bostwick, 2000; Pavlovic, 2004). It has been hypothesized that buspirone-mediated dopaminergic activity in the mesocortical pathway is responsible for an improvement in bruxism (Bostwick and Jaffee, 1999). However, drug-induced movement disorders do respond to reduction in doses of the drugs. It is noteworthy that in none of these reports any attempt to decrease the dose of the culprit antidepressant was made. We hereby report cases of bruxism induced by high dose of escitalopram (first report of escitalopram-induced bruxism) and venlafaxine, who were treated by dose adjustment. Subsequently we discuss the need for buspirone in such cases.

Case 1

Patient A, a 48-yr-old married male diagnosed with generalized anxiety disorder (GAD) and anankastic personality disorder was started on 10 mg/d escitalopram. The dose was increased to 40 mg/d over 2 wk. The patient showed marked improvement in his psychopathology as revealed by a fall in the total score on the Hamilton Anxiety Scale from 35 to 12 over 3 wk after starting the treatment. During the hospital stay he was found to be hypertensive for which he was started on 50 mg/d losartan tablet which resulted in control of the hypertension. He was discharged on 40 mg escitalopram and 50 mg losartan tablet. On follow-up after 1 month the patient reported that he was grinding his teeth at night during sleep as informed by his wife. As the problem was very distressing for the couple the dose of escitalopram was reduced to 35 mg/d but without any improvement over next 2 wk. The bruxism disappeared when the dose was reduced gradually to 25 mg/d over next 4 wk. The dose of losartan was left unchanged. There was no worsening in the patient’s mental and physical status. Currently he is doing well on 25 mg/d escitalopram over the last 3 months without any reappearance of the bruxism.

Case 2

Ms. S, an 18-yr-old female, studying in pre-university school came to a tertiary care centre in January 2005. The presenting complaints were feeling low, loss of interest in study, poor concentration, poor sleep and poor appetite following the death of her paternal grandmother 1 wk earlier. A detailed psychiatric assessment revealed a family history of severe depression and suicidal death in the elder sister of Ms. S with whom she had a close relationship and following which Ms. S had been sharing a closer relationship with her grandmother. Repeated images and memories of the deceased were reported by the patient. In addition she also reported death wishes and suicidal ideations. A diagnosis of complicated grief was made and the patient was started on grief resolution therapy. After the patient consented to pharmacotherapeutic intervention she was put on 37.5 mg/d venlafaxine which was increased to 225 mg/d over the next 3 wk. The increase in doses was necessitated by little improvement in sleep and mood initially which improved considerably at 225 mg/d. After 3 wk the parents who were staying with the patient reported...
that she was grinding her teeth during sleep at night. This behaviour was reported to interfere with the sleep of the parents. The patient reported discomfort in her jaw during morning hours. It was observed by the family for four continuous nights before a decision to decrease venlafaxine to 187.5 mg/d was made. After 3 d of decrease in dose the bruxism disappeared. The patient was next monitored for 1 wk with no relapse of bruxism. The grief resolution therapy was continued. She had significant improvement after 1 month of combination treatment when she was discharged from the hospital.

To our knowledge the first case described here is the first ever reported case of escitalopram-induced bruxism. Escitalopram is the S-enantiomer of citalopram with a potent and highly selective serotonin reuptake inhibition property. Since citalopram has been known to cause bruxism, it is not unexpected with escitalopram (Wise, 2001).

The disappearance of bruxism in both the cases with dosage manipulation raises some interesting issues. For the treatment of bruxism, buspirone has been found to be useful in case reports (Bostwick and Jaffee, 1999; Jaffee and Bostwick, 2000); however, the mechanism of action is unclear. The dopamine–serotonin imbalance hypothesis proposed by Bostwick and Jaffee (1999) states that bruxism is a kind of akathisia induced by hypofunction of dopamine in the mesocortical pathway. This hypofunction is a result of antidepressant-induced hyperactivity of serotonergic neurons of the raphe nucleus projecting to the ventral tegmental area and inhibiting the mesocortical dopaminergic pathway. Buspirone acts as a full agonist at presynaptic, and as a partial agonist at post-synaptic 5-HT1A receptors. This differential action of buspirone on presynaptic and post-synaptic 5-HT1A receptors brings about normalization of dopaminergic action leading to the disappearance of bruxism.

The selectivity of SSRIs for serotonin reuptake varies, with the highest ratio of serotonin to dopamine reuptake inhibition documented for escitalopram (Richelson, 2002). Venlafaxine is predominantly a serotonin reuptake inhibitor at lower doses but the dopamine reuptake inhibition increases with increasing dosage (Stahl, 1997). Extrapolating from these facts, it can be said that the serotonin–dopamine balance in patients receiving these drugs is subject to the dosage and the type of the drug. Thus, changes in direction of dosage can favour this balance towards serotonin or dopamine. As a result the hypodopaminergic action of the mesocortical pathway can be normalized by manipulation of the dosage to bring down the serotonergic action. Given this fact, the need for buspirone in such cases is questionable without giving a trial of dose manipulation.

The first patient described here received high dose of escitalopram. A decrease in the dose led to disappearance of bruxism which in all likelihood resulted from the serotonin–dopamine balance tilting towards dopamine. In the second case, the bruxism disappeared with reduction of venlafaxine dose from 225 mg/d to 187.5 mg/d, which again indicates the tilt towards dopamine. In both cases no relapse of psychopathology was noticed after dose reduction. The cases described by Bostwick and Jaffee (1999) received moderate dosage (100 mg) of sertraline. None of the cases were given a trial of dose reduction. Similarly the case described by Pavlovic (2004) received buspirone without a trial for reduction of the dose of venlafaxine. It has been argued that the addition of buspirone was necessitated by the high level of residual anxiety. However, it should be noted that movement disorders including akathisia are themselves accompanied by a high level of anxiety, dysphoria and agitation which might be mistaken as a worsening of the original psychopathology.

Our observations do not undermine the usefulness of buspirone in treating bruxism, yet we feel that it is always beneficial to manage adverse effects with dosage manipulation wherever possible.

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Statement of Interest
None.

References


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