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## Very Low Dose Aripiprazole (2 mg/d) for Venlafaxine-Induced Bruxism: A Case Report

The association of bruxism with selective serotonin reuptake inhibitors/serotonin norepinephrine reuptake inhibitors (SSRI/SNRI) has been noted for a long time.<sup>1,2</sup> Here we report a case of venlafaxine-induced sleep bruxism and its successful management with very low dose aripiprazole.

### Case Report

Mr U, a 21 years old male, presented with four months' history of a severe depressive episode without psychotic symptoms. An adequate trial of escitalopram failed, and he was initiated on venlafaxine. The dose was gradually increased to 225 mg/d over a period of three weeks. His depressive symptoms improved partially over four weeks (40% reduction in the Hamilton Depression Rating Scale, i.e., from 42 to 25). After four weeks, the dose of venlafaxine was increased to 300 mg/d. In the

following 4–5 days, the patient's caregivers, who had stayed throughout this period with him, noticed the sound of teeth grinding and clenching when the patient was asleep in the night. The frequency of night bruxism increased in the next few days, occurring for 3–4 minutes every hour. Mr U reported discomfort in his jaws after waking up in the morning, but there was no history of awake bruxism, and the patient neither remembered sleep bruxism nor complained of sleep disturbance. Because of an increase in the frequency of suicidal ideas, the patient was offered inpatient care. By this time, he had received Cap venlafaxine 300 mg/d for about ten days. Diagnostic possibility of venlafaxine-induced bruxism was considered, and aripiprazole 2 mg/d was added to venlafaxine 300 mg/d on the second day of inpatient care. The frequency of sleep bruxism decreased from the first day of adding aripiprazole and it completely stopped. As depressive symptoms were persisting, the clinical history was reclarified, and an episode suggestive of hypomania in the past was noted. The primary psychiatry diagnosis was revised to bipolar affective disorder

(BPAD) current episode severe depressive episode without psychotic symptoms. Lithium carbonate (1050 mg/d) was added to venlafaxine (300 mg/d) and aripiprazole (2 mg/d) after about a week of IP care. On this treatment, his depressive symptoms improved completely in three weeks, and he was discharged. Mr U was continued on the same medications for two months after discharge, and he did not have any recurrence of sleep bruxism during this period. Later, venlafaxine dose was decreased to 225 mg/d, and aripiprazole was stopped after a week of decreasing the venlafaxine dose. It has been two months since stopping aripiprazole. Mr. U did not have a relapse of sleep bruxism, and he has been maintaining well on venlafaxine 225 mg/d and lithium 1050 mg/d. We intend to taper off venlafaxine in the follow-up. Score on Naranjo Adverse Drug Reaction Probability Scale was 4, which suggest probable role of venlafaxine in the occurrence of bruxism.

### Discussion

Our report highlights the utility of very low dose aripiprazole (2 mg/d) in the

TABLE 1.

**Antipsychotics for the Management of SSRI/SNRI-Induced Bruxism**

Age (Years), Sex (M/F)	Diagnosis	SSRI/SNRI and Dose (mg/d)	Antipsychotic Agent and Dose (mg/d)
67, F <sup>12</sup>	Nonaffective psychosis with depressive disorder	Paroxetine, 20	Chlorpromazine, 50
63, F <sup>8</sup>	OCD	Escitalopram, 60	Aripiprazole, 10
28, F <sup>3</sup>	MDD	Sertraline, 75	Quetiapine, 25
35, M	MDD	Sertraline, 50	Quetiapine, 25
18, F	MDD	Fluoxetine, 20	Quetiapine, 37.5
45, F	MDD	Sertraline, 50	Quetiapine, 37.5
32, F	MDD	Citalopram, 10	Quetiapine, 25
21, M <sup>*</sup>	Bipolar affective disorder	Venlafaxine, 300	Aripiprazole, 2

SSRI = selective serotonin reuptake inhibitor; SNRI = serotonin-norepinephrine reuptake inhibitor; OCD = obsessive compulsive disorder; MDD = major depressive disorder; \*current report.

management of venlafaxine-induced bruxism. More commonly, buspirone, by virtue of its partial agonism of 5HT<sub>1A</sub> receptors, has been used in the management of SSRI/SNRI-induced bruxism.<sup>1</sup> There are also reports of antipsychotics being used in the management of the same (Table 1). Oulis et al. reported, for the first time, the utility of aripiprazole (10 mg/d) for the management of SSRI (fluoxetine and escitalopram)-induced bruxism.<sup>3</sup> Aripiprazole is a partial agonist of dopamine receptors (D<sub>2</sub> and D<sub>3</sub>), with 30%–40% intrinsic dopamine agonistic activity.<sup>4</sup> So, our report suggests that aripiprazole at the dose of 2 mg/d may have sufficient dopamine agonistic activity to be effective for the management of venlafaxine-induced bruxism. Aripiprazole blocks about 70% of the postsynaptic D<sub>2</sub> receptors at about 2 mg, and the proportion increases to 90%–94% at the dose of 30 mg.<sup>5</sup> SSRI-induced bruxism is due to the decreased prefrontal dopaminergic tone.<sup>6</sup> It may be that 2 mg/d of aripiprazole leaves adequate postsynaptic dopamine receptors for the available endogenous dopamine to act upon; the endogenous dopamine, together with the partial agonistic activity of aripiprazole, may overall enhance the dopaminergic activity in the prefrontal cortex. This may be the reason for the improvement it produces in SSRI-induced bruxism. At higher dose, aripiprazole may compete with the available

endogenous dopamine and occupy higher percentage of the postsynaptic dopamine receptors. The overall effect may be dopamine antagonism in the prefrontal cortex and inducing bruxism as noted in an earlier case report. Lithium carbonate has been reported to increase serum aripiprazole levels by 43%, and the combination of lithium 1200 mg/d and aripiprazole 15 mg/d was reported to be associated with bruxism and extrapyramidal symptoms in a patient with BPAD.<sup>7,8</sup> It may be due to the dopamine antagonistic activity of the aripiprazole at that dose when given in combination with lithium. In our patient, complete improvement in bruxism had been noted before adding lithium, and there were no extrapyramidal symptoms after adding lithium. The current report also suggests that venlafaxine-induced bruxism is dose-dependent and that, if clinically advisable, dose reduction may be used as a measure to relieve bruxism. This is in line with earlier reports of dose reduction being helpful in the management of SSRI-associated bruxism.<sup>2,9</sup> In other available reports of venlafaxine-induced bruxism, either venlafaxine was stopped or add-on treatment with other agents was considered.<sup>1</sup> Though anecdotal, our report suggests the need for further systematic studies to explore the role of very low dose aripiprazole in the management of antidepressants-induced bruxism.

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## Diagnostic and Therapeutic Challenges due to Psychosis and Catatonia in Non-syndromic Retinitis Pigmentosa: A Case Report

To the editor,

**R**etinitis Pigmentosa (RP) is a group of heterogeneous, inherited retinal disorders characterized by the degeneration of the photoreceptor cells and, in extreme cases, the retinal pigment epithelium. The patients initially experience night blindness and eventually progressive constriction of the visual field, leading to central vision loss or even complete blindness.<sup>1</sup> The prevalence of RP is 1:3000 to 1:7000.<sup>2</sup> RP is classified into three types: syndromic (involving special senses of the nervous system such as hearing), non-syndromic (not affecting other organs or tissues), and systemic (affecting multiple organs). Similar to the other variants, the mode of inheritance of non-syndromic RP include autosomal dominant (15%–25%), autosomal recessive (5%–20%), X-linked (5%–15%), unknown: simplex (40%–50%), and digenic (very rare).<sup>3</sup> Few reports exist regarding the intriguing relationship between syndromic RP (ushers syndrome) and psychiatric illnesses, the most common being schizophrenia with manifestations of delusions and hallucinations.<sup>4</sup> However, whether psychiatric disturbance can co-occur in non-syndromic RP and whether it can have other varied psychopathology has not been studied earlier.

We describe a rare case of a young female with non-syndromic RP presenting with psychotic features, including catatonic symptoms and auditory hallucinations, with a difficult-to-treat episode requiring electroconvulsive therapy (ECT).

## Case Presentation

A 20-year-old female with no past or family history of psychiatric illness presented with an acute onset behavioral disturbance lasting for one month, characterized by irrelevant speech, wandering behavior, hallucinatory behavior (claiming to hear voices, without perceiving their images), agitation, and disturbed sleep and appetite. Her medical history revealed progressive deterioration of vision since childhood. There was no family history of visual impairment. Her enduring visual impairment led to difficulties in assessing her present state perceptual abnormalities. Her blood biochemistry (complete blood counts, renal and liver function tests) and electrocardiogram returned normal. A diagnosis of acute and transient psychotic disorder, based on the *International Classification of Diseases, Tenth Revision*, was made. She scored 39 in the Brief Psychiatric Rating Scale (BPRS).

Ophthalmology opinion was sought and a comprehensive evaluation was done for the visual impairment. The fundi examination revealed retinal pigment clumping, waxy disc pallor, and arteriolar attenuation. Subsequently, the possibility of a syndromic RP was evaluated. The absence of hearing impairment (noted in the clinical examination) ruled out usher syndrome. The lack of ophthalmoplegia, ataxia, dysphagia, and cardiac conduction deficits eliminated the possibility of Kearns–Sayre syndrome. Absence of steatorrhea and peripheral neuropathy and normal peripheral blood smear ruled out abetalipoproteinemia. A normal sexual and psychological development ruled out Bardet–Biedl syndrome.

She was started on T. Risperidone 2 mg/d and the dose was gradually titrated to 6 mg/day. Risperidone was chosen

primarily because, as an atypical anti-psychotic, it was safer than the typical antipsychotics (especially, chlorpromazine and thioridazine), which have an increased propensity to cause ocular side effects such as retinopathy.<sup>5</sup> She was also on T. clonazepam 3mg/day for sedation. Her psychotic symptoms improved significantly within two weeks, as reflected by the BPRS score of 28. While tapering down the dose of clonazepam from 3mg/day to 2 mg/day, she developed catatonic symptoms of excitement, verbigeration, negativism, withdrawal, and perseveration. She scored 11 on Bush Francis Catatonia Rating Scale (BF-CRS). With lorazepam (up to 8 mg/day), she showed minimal improvement in catatonic symptoms, and her oral intake was compromised. ECT was administered, and the catatonia resolved after two sessions (BF-CRS = 0, BPRS = 25). After discharge, lorazepam was tapered and stopped over four weeks. Psychosocial interventions such as supportive counseling and educating on personal safety and the provision for disability benefits were done. The patient was on anti-psychotic prophylaxis for six months, was maintaining well (BPRS = 20), and did not have further relapses.

## Discussion

This report highlights that apart from the syndromic variety of RP, non-syndromic RP also can have psychiatric complications. Additionally, psychosis in non-syndromic RP can present with catatonic symptoms. Psychoses in syndromic RP had mostly presented with hallucinations and delusions, and catatonic symptoms appear to be a rare phenomenon.<sup>2,6</sup> As noted in our patient, a thorough medical history and clinical examination are necessary to eliminate the variants of syndromic RP and to arrive at a diagnosis of non-syndromic RP.