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Case report

Polysomnographic sleep effects of fluoxetine and nefazodone on a seasonal affective disorder patient

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Abstract

This reported person met DSM-III-R criteria for seasonal affective disorder. In successive years she had two treatments with fluoxetine and one with nefazodone. Every year regular polysomnographic monitoring was carried out during the critical initial 8 weeks of treatment. The results indicate that on both occasions fluoxetine decreased the patient's sleep quality. Nefazodone had no sleep disturbing effects and on withdrawal no relapse has been seen for 3 years. © 2002 Published by Elsevier Science B.V.

Keywords: Seasonal affective disorder; Case report; Treatment; Fluoxetine; Nefazodone; Polysomnography

1. Introduction

Although the polysomnographic (PSG) effects of fluoxetine (increased sleep latency and decreased slow wave sleep) and nefazodone (decreased wakefulness and increased REM sleep) in major depression patients are well documented [1–7], to our knowledge there are limited data vis-à-vis seasonal affective disorder (SAD), particularly the contrasting effects in a single patient. The purpose of this report is to highlight the different PSG effects of fluoxetine from those of nefazodone in a single patient with SAD.

2. Case

Ms T was a 44-year-old nurse when she was diagnosed with SAD in 1996. At the time she presented she was experiencing her fourth consecutive winter depression. Her diagnosis of SAD was based on the Structure Clinical Interview for DSM-III-R (SCID) [8]. She scored 38 on the 29-item Hamilton Depression Rating Scale (HDRS-29). She scored 33 on the Beck Depression Inventory (BDI) and 47 on the Center for Epidemiological Studies of Depression Scale (CES-D). These results indicate that Ms T suffered from a severe depression. The score on the Seasonal Patterns Assessment Questionnaire (SPAQ) was 12. A score of 10 or higher on the SPAQ supports a diagnosis of SAD.

3. Method

Ms T started taking fluoxetine 20 mg daily in November 1996 and stopped the medication in January 1997 when she experienced a good antidepressant response. Consecutive two-night sleep studies were done prior to administration of medication (baseline, W0), and by the end of 4 (W4) and 8 weeks (W8) of treatment, respectively. In October 1997, Ms T had a relapse of her depression and repeated the same procedure on fluoxetine (20 mg daily) with PSG recordings. After taking fluoxetine for 8 weeks her mood improved but her sleep disturbance was such that she was unable to continue the medication. Two weeks after discontinuation she started taking nefazodone at doses of 100 mg daily in week 1, 200 mg daily in week 2, 300 mg daily in week 3 and 400 mg daily in weeks 4–8, respectively. Two consecutive overnight PSG recordings were done at baseline, and at the end of weeks 4 and 8 as well. Sleep recordings were carried out and scored by an experienced registered technologist. To avoid the first-night effect, only the second night recordings were analyzed.

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Table 1
Sleep measurement changes induced by fluoxetine and nefazodone[a]

Variable		Week		
		0	4	8
Sleep efficiency (%)	F1	86.8	55.4	46.3
	F2	93.7	64.2	54
	N	65.4	87.2	93.4
Sleep latency (min)	F1	24.5	17.5	100
	F2	10	39	28.5
	N	63.5	14.5	11
REM latency (min)	F1	92	243.5	116
	F2	73	110.5	166
	N	176	67.5	78.5
REM sleep (%)	F1	20.4	2.4	6.7
	F2	20.7	10.9	4
	N	16.4	14.4	27.7
Wake (%)	F1	7.8	36.6	39.3
	F2	3.3	30.2	41
	N	24	9.8	4.1
Slow wave sleep (%)	F1	34.4	30	22.2
	F2	25.2	22.5	14
	N	22.6	29	15.6

[a] F1 = the first occasion of taking fluoxetine; F2 = the second occasion of taking fluoxetine; N = on nefazodone.

4. Results

The sleep disrupting effect of fluoxetine treatment was similar on both occasions: a progressive decrease in sleep efficiency over time. Nefazodone seems to have no disturbing effects on sleep efficiency. During both episodes fluoxetine had a tendency to increase rapid eye movement (REM) latency and wakefulness. It mildly decreased slow wave sleep, but substantially reduced REM sleep. While taking nefazodone her wakefulness during sleep tends to decrease. At W8 on nefazodone her REM sleep increased significantly, but slow wave sleep decreased mildly. On the first fluoxetine treatment occasion her HDRS-29 scores decreased from 38 at W0 to 25 at W4 and 17 at W8, and on the second occasion from 36 at W0 to 19 at W4 and 17 at W8. HDRS-29 scores on nefazodone treatment were 24 at W0, 19 at W4 and 20 at W8. In the following 3-year observation she did not have any relapse of depression (Table 1).

5. Discussion

The sleep quantity and quality of this patient were significantly disturbed by fluoxetine. This finding is consistent with those of other researches [1–6]. Although there was a 1-year interval between the two episodes of fluoxetine consumption, the PSG profiles were very similar on both occasions. Apart from a mild decrease of slow wave sleep at week 8, nefazodone seems to have had no sleep disturbing effect. Different PSG effects of these two medications may be based on different mechanisms of action [2].

A limitation of this case description is that the baseline PSG measurements on nefazodone treatment may have been contaminated by the effects of fluoxetine, which was discontinued for 2 weeks at the time of starting nefazodone.

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