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CASE REPORT

Spontaneous Orgasms During Risperidone Treatment in a Schizophrenic Patient: A Case Report

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We present a case report of a male schizophrenic patient who suffered spontaneous orgasms after receiving risperidone. This is a phenomenon that has been previously described in relation to antidepressive drugs. The strong 5-HT2 antagonism of risperidone may be the mechanism responsible for an indirect enhancement of the dopaminergic transmission. © 1998 John Wiley & Sons, Ltd.

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KEY WORDS — risperidone; schizophrenia; sexual dysfunction; orgasms

INTRODUCTION

Spontaneous orgasms is an unusual condition described during antidepressive treatment with several different drugs: amineptine (Chabrol and Bonnet, 1995), clomipramine (McLean *et al*., 1983; Bertschy *et al*., 1991), fluoxetine (Modell, 1989; Morris, 1991; García-Campayo *et al*., 1995), and trazodone (Purcell and Ghurye, 1995).

Neuroleptic treatment in schizophrenic male patients is associated with frequent sexual dysfunctions (Aizenberg *et al*., 1995a), including impotence and decreased libido. Spontaneous ejaculation, but without sexual arousal, during neuroleptic treatment is a very rare condition that has been communicated with zuclopentixol (Williams and O'Brien, 1994), trifluoperazine, and thiothizene (Keitner and Selub, 1983). During risperidone treatment some genitourinary secondary effects (priapism, ejaculatory dysfunction) have been communicated (Emes and Millson, 1994; Tekell *et al*., 1995; Madhusoodanan and Brenner, 1996), as in other neuroleptic drugs (Patel *et al*., 1996).

CASE REPORT

We present a case of spontaneous orgasms related to risperidone treatment.

Mr A. is a 40 year-old white man suffering from paranoid schizophrenia; he is married and sexually active. He was taking 15 mg/day of trifluoperazine with a partial control of his delusional ideas. During his treatment, the patient has suffered from decreased libido, erectile difficulties, sexual insatisfaction, and a diminution of intercourse with his wife. He presented blunted affect and energy loss, but he did not complain of depressive mood and we decided to change his antipsychotic treatment.

The patient was gradually switched from trifluoperazine to risperidone reaching 6 mg/day. After one week of taking risperidone the patient suffered three episodes of sexual pleasure without any stimulus which he identified as orgasms, stating that he was very surprised and disturbed. He did not present any ejaculation. The patient recovered from the sexual dysfunctions, having complete and satisfactory relations with his wife. His negative symptoms and the delusional ideas also improved and he was less worried about them. After one month of treatment at the same dosage, the patient's sexual function again worsened. Risperidone was progressively decreased to 3 mg/day and sexual function improved again, becoming satisfactory for the patient. No more spontaneous orgasms have been related by this patient and his psychotic symptoms remained stable.

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DISCUSSION

The enhancement of dopaminergic transmissions by drugs in experimental animals and in humans has been related to sexual hyperarousal. Serotonin, as we can see with serotonergic antidepressants, decreases libido and generates erectile and orgasmic dysfunctions (Gitlin, 1994). These effects on sexuality may be due to the inhibition of serotonin upon dopaminergic transmission (Kapur and Remington, 1996).

Risperidone can stimulate sexual behaviour in rats, under acute and chronic administration, at doses of 0·1 mg kg^{-1} (Drago *et al.*, 1997). Cyproheptadine has been used in reversing the anorgasmia induced by SSRI (Aizenberg *et al.*, 1995b), tricyclic antidepressants (Sovner, 1984), MAOI (De Castro, 1985), and flupentixol (Jeffries and Walker, 1987); the 5-HT2 antagonism of cyproheptadine has been proposed as the restoring mechanism of sexual function. This effect may be due to an indirect enhancement of dopaminergic transmission as the one described with ritanserin, a highly selective 5-HT2 antagonist (Ugedo *et al.*, 1989).

Risperidone can also activate dopaminergic transmission (Stathis *et al.*, 1996), despite its D2 antagonism, and by this mechanism exacerbate sexual arousal. Possibly trazodone, with 5-HT2 antagonism properties, acts in the same way.

Controlled studies may be of interest to determine the profile of sexual secondary effects of risperidone versus other neuroleptic drugs.

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