
Selective Serotonin Reuptake Inhibitor–Induced Spermatorrhea in 2 Patients

To the Editor: Antidepressants are known to induce sexual dysfunction, resulting in increased risk of nonadherence to treatment.¹ While problems with ejaculation and orgasm have been reported,¹ to our knowledge, there is no report of antidepressant-induced spermatorrhea (ie, excessive emission of semen without orgasm or erection). Spermatorrhea annoys men of all ages and lowers their quality of life.² Here, we report the first case series of patients with obsessive-compulsive disorder or depression who experienced possible antidepressant-induced spermatorrhea.

Case 1. Mr A, a 33-year-old man with a 1-year history of *DSM-IV* major depressive disorder, visited us in April 2006 due to impaired concentration and depressive mood. From the day he started taking fluvoxamine 200 mg/d, spermatorrhea occurred during urination without pain every day. Following a switch to nortriptyline 75 mg/d, spermatorrhea ceased. However, nortriptyline was not effective for depressive symptoms and was therefore switched to paroxetine 40 mg/d. Following this switch, Mr A's depressive symptoms were diminished, but spermatorrhea reoccurred once per week. Finally, spermatorrhea was stopped by reducing the paroxetine dose to 20 mg/d, without a worsening of depression.

Case 2. Mr B, a 19-year-old man with a 2-year history of *DSM-IV* obsessive-compulsive disorder, visited us in October 2008 due to disease phobia, germ phobia, and hand-washing compulsions. From the day that he started taking fluvoxamine 75 mg/d, he was awakened by the sensation of emission of semen without orgasm or erection every night, which disturbed his sleep. Spermatorrhea stopped when fluvoxamine treatment was discontinued. When fluvoxamine treatment was restarted at 25 mg/d, Mr B experienced a marked reduction in his obsessive and compulsive symptoms, with no recurrence of spermatorrhea.

Neither patient had a history of urologic disorders or current abnormal findings in urologic examinations, including semen and urine tests. Their compliance with medications was regularly

monitored, using pill counts, and found to be good in both cases. In addition, neither patient took any concomitant medication.

The literature on spermatorrhea is scarce, and mechanisms underlying this phenomenon remain unknown.² While serotonin (5-HT) receptors are known to be associated with sexual function, 5-HT has 2 distinct and opposite effects: 5-HT_{1A} and 5-HT_{2C} receptors have stimulating and inhibitory effects, respectively, on sexual function during exposure to receptor agonists.³ The spermatorrhea that we observed in this report might have been caused by an imbalance between contrasting functions of these 2 receptors.

Dose reduction or discontinuation of antidepressants ceased spermatorrhea in both cases, which is comparable to the fact that antidepressant-induced sexual dysfunction is dose-dependent.^{1,4,5} Furthermore, switching from a selective serotonin reuptake inhibitor (SSRI) to a tricyclic antidepressant (TCA) resulted in an improvement of spermatorrhea, and a subsequent switch from the TCA to another SSRI caused this phenomenon in the first case. This finding is consistent with the recent contention that more powerful serotonergic medications may cause more sexual side effects than TCAs.⁵⁻⁷

Given the widespread use of SSRIs for various psychiatric disorders,⁸ more careful attention should be paid to this annoying and potentially serious side effect, irrespective of diagnosis. Further investigations of spermatorrhea are needed to elucidate its mechanisms, effective management, and risk factors.

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