

A rare symptom in posttraumatic stress disorder: Spontaneous ejaculation

Authors' Contribution:
Study Design A
Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
Literature Search F
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Patient: Male, 25
Final Diagnosis: Post Traumatic Stress Disorder
Symptoms: Insomnia • nightmares • spontaneous ejaculation
Medication: Paroxetine
Clinical Procedure: —
Specialty: Psychiatry

Objective: Unusual clinical course
Background: Sexual dysfunction is reported to occur more frequently in posttraumatic stress disorder (PTSD) patients than in the general population. Herein, we present the case of a patient with spontaneous ejaculation that developed when severity of PTSD symptoms increased.
Case Report: Our patient was a 25-year-old single man admitted to a psychiatric polyclinic because of PTSD symptoms and concurrent spontaneous ejaculations. He was diagnosed with PTSD after clinical interviews. Organic pathology to explain spontaneous ejaculations was not detected. Paroxetine treatment was initiated and PTSD symptoms and frequency of spontaneous ejaculations were decreased at the clinical follow-up.
Conclusions: Assessment of the presented case in the light of the literature indicates that his re-experiencing (flashbacks, nightmare) and hyperarousal (symptoms of anxiety specific to PTSD) led to an increase in adrenergic system activation and, consequently, spontaneous ejaculation without sexual stimulus. The effect of Paroxetine in decreasing the frequency of spontaneous erection and ejaculation in the presented case is thought to have occurred via control of PTSD symptoms and their adverse effects on ejaculation. Treatment based on a consideration of PTSD symptoms and autonomic instability might increase the positive outcome rate in such patients.

MeSH Keywords: Paroxetine • Ejaculation • Stress Disorders, Post-Traumatic

Full-text PDF: <http://www.amjcaserep.com/download/index/idArt/889658>

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Background

Sexual dysfunction is reported to occur more frequently in post-traumatic stress disorder (PTSD) patients than in the general population [1–8]. Premature ejaculation, loss of libido, and difficulty maintaining an erection are the most common sexual dysfunctions in PTSD patients [2–4]. Various mechanisms have been proposed to play a role in causing sexual dysfunction in PTSD patients [9]. Numbing of responsiveness and a general lessening of affect that manifest as avoidance behaviors in PTSD patients are thought to be associated with sexual dysfunction that occurs following traumatic life events [3,9]. Physiological arousal caused by sexual activity can initiate memories of trauma and other re-experiencing phenomena [3]. Comorbid physical health problems and psychiatric disorders, such as panic disorder, depressive disorder, social phobia, and generalized anxiety disorder, may cause or exacerbate sexual dysfunction in PTSD patients [8,10].

On the other hand, selective serotonin reuptake inhibitors (SSRIs), commonly used to treat PTSD, can cause sexual dysfunction [11–15]. Moreover, secondary sexual dysfunction can develop in the sexual partners of PTSD patients due to impairment in their sexual compatibility, which can completely disrupt the sexual relationship between PTSD patients and their partners [16]. Additionally, a hyperactive autonomic nervous system that leads to hyperarousal symptoms in PTSD [17,18] has been associated with the sexual dysfunction observed in PTSD patients.

Although cases of spontaneous ejaculation due to a variety of drugs, as well as psychiatric and neurological problems, have been reported, spontaneous ejaculation comorbid with PTSD not treated with drugs has not been reported. Herein, we present the case of a patient with spontaneous ejaculation that developed when severity of PTSD symptoms increased.

Case Report

M.B. was a 25-year-old, single, male, university graduate. While performing mandatory military service, he was wounded in the left arm by gunfire in 2012. He received orthopedic and physical therapy treatment. Psychiatric consultation was requested by the physical therapy clinic based on the patient's request 3 months after the traumatic event. The patient's complaints were insomnia, nightmares, irritability, avoidance of crowds, inability to watch news of violent events, startle response due to sudden noises, and flashbacks of the traumatic event of being wounded by gunfire, which began after the traumatic event. Additionally, he experienced involuntary partial erection and ejaculation 2–3 times per day. Involuntary erection and ejaculation would occur during routine daily activities and sleep

during multiple periods in which the patient re-experienced the traumatic event (as flashbacks and nightmares) and had hyperarousal. The patient did not experience any pleasure while having spontaneous erection and ejaculation, but did experience intense guilt and anxiety. During the psychiatric interview, the patient was restless and exhibited such symptoms of anxiety as sweating and hand tremors. Physical examination revealed no pathology other than limited left elbow movement secondary to his gunfire injury. Neurological examination findings were normal.

Electroencephalography and brain magnetic resonance imaging (MRI) were performed to exclude possible organic pathologies, and the findings were normal. The patient was evaluated by a psychiatrist using the Structured Clinical Interview for Diagnostic and Statistical Manual of Mental Disorders, Axis I Disorders (SCID-I) and was diagnosed with PTSD. Psychometric assessment included the Beck Anxiety Inventory (BAI) (score: 42) and the Beck Depression Inventory (BDI) (score: 15). The patient's Impact of Event Scale (IES) score was 76 (IES-R re-experiencing: 30; IES-R avoidance: 25; IES-R hyperarousal: 21) and his State-Trait Anxiety Inventory (STAI) score was 65 for state anxiety and 70 for trait anxiety. The psychometric findings indicated that the patient had severe anxiety and PTSD. Results of urological examination, including pelvic examination, bulbocavernosus reflexes, and ultrasound imaging, were normal. Routine biochemical and hormonal profiles [adrenocorticotropic hormone: 16.2 pg/ml (0–46), follicle-stimulating hormone: 5.57 IU/L (1.4–18.1), luteinizing hormone: 4.69 IU/ml (1.5–9.3), free T3: 3.53 pg/ml (2.3–4.2), free T4: 1.01 ng/dl (0.89–1.76), thyroid-stimulating hormone: 2.45 microu/ml (0.35–5.5), growth hormone: <0.05 ng/ml (0–1), prolactin: 7.6 ng/ml (2.1–17.7), total testosterone: 522.0 ng/dl (241–877), and release testosterone: 16.27 pg/ml (8.6–54.6)] were normal. Based on these findings, the patient's spontaneous erection and ejaculation complaint were considered to be secondary to psychiatric symptoms.

Paroxetine 20 mg/day was initiated and 2 weeks later there was a partial reduction in the overall complaints. The patient's drug treatment compliance was good and the frequency of spontaneous erection/ejaculation decreased to 1–2 times per week. The paroxetine dose was gradually increased to 60 mg/day and no adverse effects were observed.

Although the patient had partial improvement in the severity of PTSD symptoms, PTSD was determined to be chronic at the 3-month follow-up. The frequency of spontaneous erection/ejaculation was reduced to a mean of once every 2 weeks. Psychometric assessment scores at the 3-month follow-up were as follows: BAI: 33, BDI: 12; IES: 54 (IES-R re-experiencing: 20; IES-R avoidance: 21; IES-R hyperarousal: 13); STAI state anxiety: 48; STAI trait anxiety: 55. These scores indicated that the

disorder was persistent, even though the severity of the symptoms of anxiety and PTSD decreased.

Discussion

Coordination of the autonomic and somatic nervous systems is required for ejaculation to occur [19]. Catecholamines, such as dopamine and noradrenaline, facilitate ejaculation, whereas serotonin inhibits it [20–23]. Increased adrenergic activity in the anterior hypothalamus can cause spontaneous ejaculation by decreasing ejaculatory latency [24]. After ejaculation, an increase in extracellular serotonin in the anterior hypothalamus initiates the refractory period and prevents recurrent ejaculations [20,25]. Premature or spontaneous ejaculation has been reported in cases of increased adrenergic activity (panic attacks, social phobia, and use of adrenergic drugs) [20,21,24,26–29]. Sympathetic hyperactivity and a decrease in serotonin activity have been reported to be possible causes in such cases [20,21,24,27,29].

Plasma and urinary catecholamine levels in PTSD patients are higher than in healthy controls [30,31]. A correlation between dopamine and epinephrine levels, and the severity of PTSD symptoms has been noted [30]. The hyperactive autonomic nervous system in PTSD patients can cause sexual dysfunction, even in patients without any hormonal pathology [17,18,32,33]. On the other hand, hyperarousal associated with erection and ejaculation can cause intrusive phenomena in PTSD patients [30], and dopamine, epinephrine, and norepinephrine are associated with hyperarousal and intrusive symptoms in PTSD. These catecholamines were observed to play a role in the formation of intrusive images during sexual intercourse in PTSD patients [30].

Assessment of the presented case in the light of the literature indicates that his re-experiencing (flashbacks, nightmare) and hyperarousal (symptoms of anxiety specific to PTSD) led to an increase in adrenergic system activation and, consequently, spontaneous ejaculation without sexual stimulus. This conclusion is further supported by the decrease in re-experiencing and hyperarousal scores after starting pharmacological treatment for the symptoms of PTSD, and the concurrent decrease in the frequency of spontaneous erection/ejaculation.

In a rat model of PTSD, trauma-associated symptoms of anxiety caused ejaculatory dysfunction via the gastrin-releasing peptide (GRP) system [34]. The researchers have suggested that symptoms in humans with PTSD might similarly affect the GRP system, resulting in erectile and ejaculatory dysfunction [34]. Adrenergic and GRP system dysfunction might cause the sexual dysfunction observed in PTSD patients. Dysfunction of these 2 systems may play a role in the development of spontaneous

ejaculation triggered by the symptoms of PTSD, as observed in the presented case. Nevertheless, the lack of research on these systems constitutes a limitation for analyzing the presented case; our conclusions about the presented case were based on clinical observation and treatment response.

Clinical improvement in the symptoms of PTSD and a reduction in the frequency of spontaneous erection/ejaculation were observed in the presented case in response to treatment using paroxetine. Pharmaceutically, among the SSRIs, paroxetine is the most potent inhibitor of serotonin reuptake [35]. Paroxetine alone was reported to be effective for treating 3 primary symptom clusters of PTSD (hyperarousal, re-experiencing, and avoidance/numbing), as compared to the SSRIs fluoxetine, sertraline, fluvoxamine, and citalopram; males and females were equally responsive to paroxetine [36].

The effectiveness of paroxetine in treating each of the 3 PTSD symptom clusters is why it was used to treat the presented case. The effectiveness of paroxetine in treating each of these PTSD symptom clusters is thought to be due to alterations in the serotonin receptors located in the fear circuit [37]. Considering that spontaneous ejaculation in the presented case was caused by hyperarousal and re-experiencing his trauma, paroxetine might have reduced the frequency of spontaneous erection and ejaculation by decreasing the severity of hyperarousal and re-experiencing symptoms. When the active role of the adrenergic system in the occurrence of ejaculation and the lack of affinity of paroxetine in the sympathetic activity in the autonomic nervous system are evaluated together, it may be concluded that the paroxetine molecule had no direct effect in the reduction of the frequency of spontaneous ejaculation. Thus, we think that the effectiveness of paroxetine on the symptoms of PTSD might have indirectly reduced the frequency of spontaneous erection and ejaculation in the presented case.

The effect of paroxetine in treating ejaculatory dysfunctions must be considered in this case. Although sertraline, fluoxetine, and citalopram have an ejaculation-delaying effect, that of paroxetine is greater [38]. Stimulation of post-synaptic 5-HT_{1A} receptors decreased ejaculation latency in male rats, whereas 5-HT_{2C} receptor stimulation caused a delay in ejaculation [39]. On the basis of these findings, men with premature or spontaneous ejaculation may have 5-HT_{1A} receptor hypersensitivity and/or 5-HT_{2C} receptor hyposensitivity [40,41]. The ejaculation-delaying effect of paroxetine is thought to be associated with 5-HT_{2C} receptors [38,42]. Additionally, cholinergic receptor blockade and inhibition of nitric oxide synthase are effective for disinhibition of ejaculation [43,44]. Furthermore, the ejaculation-delaying adverse effect of SSRIs increases with chronic use [45]. Paroxetine has a potent cholinergic receptor blockade effect [46] and an inhibitory effect on nitric oxide

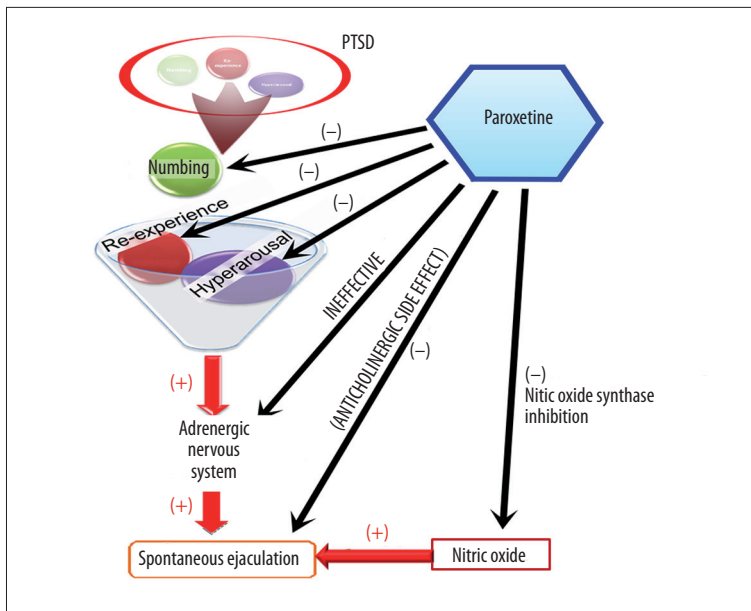


Figure 1. The effects of paroxetine on spontaneous ejaculation.

synthase [35]. It is also possible that these adverse effects of paroxetine on sexual function and ejaculation might have mediated the decrease in the frequency of spontaneous erection and ejaculation in the presented case. Consequently, the role of paroxetine in decreasing the frequency of spontaneous erection and ejaculation in the presented case is thought to have occurred via control of the symptoms of PTSD and its adverse effects on ejaculation (Figure 1).

Conclusions

To the best of our knowledge, this case presentation is the first to describe spontaneous ejaculation secondary to PTSD. We considered the mechanisms reported in the literature that might be involved in spontaneous ejaculation in PTSD. Additionally, the concurrent effect of paroxetine on the symptoms of PTSD

and spontaneous ejaculation were shown. Measurement of the metabolites of plasma, cerebrospinal fluid, corporal catecholamines, and serotonin should help to illuminate the underlying neurophysiology of sexual dysfunctions in PTSD patients. Treatment based on a consideration of PTSD symptoms and autonomic instability might increase the positive outcome rate in such patients.

Acknowledgements

Written consent was obtained from the patient for publication of the case details. No sources of funding were used to assist in the preparation of this report. The authors have no potential conflicts of interest directly relevant to the content of this report. This case report was conducted in accordance with the Declaration of Helsinki [47] and the subject gave written informed consent to participate.

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