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Case report

POSTTRAUMATIC STRESS DISORDER AND COMORBID SEXUAL DYSFUNCTIONS

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INTRODUCTION

The rates of sexual dysfunctions among patients with PTSD are much higher than in the general population. An increasing body of scientific research has confirmed clinically relevant sexual problems (Letourneau et al. 1997, Kotler et al. 2000, Hossain et al. 2013, Yehuda et al. 2015, Tran et al. 2015), among which erectile dysfunction (ED) and premature ejaculation (PE) were the most frequent (Letourneau et al. 1997). It is important to underline that patients, particularly military veterans with PTSD, have an increased risk of sexual dysfunction independent of the use of psychiatric medications (Benjamin et al. 2014).

Considering the utilization of pharmacotherapy, data indicate that over 80% of the veterans treated for PTSD in the USA have been receiving at least one of the psychotropic medications (Bernardy et al. 2012). A drug utilization study conducted in Croatia revealed that the annual frequency of drug use among pharmacologically treated PTSD patients was the highest for anxiolytics (75.83% patients), antidepressants (61.36%), hypnotics (35.68%) and antipsychotics (30.21%) in 2012 (Letica-Crepulja et al. 2015). In this context, it is very important to highlight that a variety of psychotropic medications recommended for the treatment of PTSD can induce sexual function disorders (Clayton & Shen 1998, Labbate 2008). Most practice guidelines for the treatment of PTSD highlight antidepressants as the first-line pharmacotherapeutic agents, particularly selective serotonin reuptake inhibitors (SSRIs) (Ballenger et al. 2000, American Psychiatric Association 2004, National Institute for Clinical Excellence (NICE) 2005, Baldwin et al. 2005, Forbes et al. 2007) and serotonin-norepinephrine reuptake inhibitors (SNRIs) (Bandelow et al. 2008, Benedek et al. 2009, Stein et al. 2009, Department of Veterans Affairs 2010, World Health Organization 2013, Baldwin et al. 2014). Since the introduction of these medications, increasing attention has been given to the side effects, such as sexual dysfunction (Labbate 2008, Corona et al. 2009, Serretti & Chiesa 2011). SSRIs can negatively affect all domains of sexuality (desire-arousal-orgasm-resolution) (Corona et al. 2009). A study of sexual functioning in war veterans with posttraumatic stress disorder conducted in Croatia showed that these patients had less sexual activity, hypoactive sexual desire and more frequent ED compared with healthy volunteers. These problems might be associated with the antidepressant therapy (Antičević & Britvić 2008). Another Croatian study revealed that the population exposed to traumatic event(s) had the same level of sexual functioning (or the same incidence of sexual dysfunction) regardless of the absence or presence of PTSD symptoms and their severity (Arbanas 2010).

The aim of this report was to present a patient with PTSD and comorbid sexual dysfunctions.

CASE REPORT

The Croatian 47-year-old male war veteran was a member of the Special Forces of the Croatian Army during the Homeland War (1991-1995) in Croatia. He participated in all crucial military actions at the western battlefield during the war and was exposed to multiple repetitive potentially traumatic events during combat and incarceration. He experienced the most stressful traumatic event in 1992, when his fellow soldier and he were caught in crossfire. His fellow soldier was shot dead and left bleeding in the fire-swept zone. Unable to help his dying fellow, our patient felt intense fear, helplessness and an irresistible urinary urgency. When he allowed a flow of urine, he was blocked, dry, not even a drop of urine came out of the urethra. After the traumatic exposure, he experienced recurrent and intrusive distressing recollections of the event, tension with vegetative arousal, insomnia and nightmares, startle responses to sudden noises accompanied by urinary urgency, urinary frequency and PE. He did not ask for professional help and was active in the armed forces until the end of the war. After military deployment, he continued his previous professional employment in the police, continually suffering from PTSD-related symptoms, urinary and sexual dysfunction. He got married in 1995, but still has no children.

Subsequently, in 2005, he was referred to the regional centre for PTSD. During the psychiatric and psychological assessment, the clinical diagnosis of PTSD was supported by the Harvard Trauma Questionnaire (HTQ): Croatian Version in the form of a structured interview to determine PTSD and the traumatization level (Allden et al. 1997). The treatment included participation in a psychotherapeutic program in a day hospital during 3 months, which included psychoeducation, group cognitive – behavioral therapy and trauma-focused group treatment in a homogeneous group of war veterans with PTSD and continuous psychopharmacological treatment.

During the subsequent period, he was under outpatient psychiatric care delivered through approximately 4 psychiatric controls per year. Psychopharmacologic treatment included antidepressants with conversions of several different selective serotonin reuptake inhibitors (SSRIs), serotonin-norepinephrine reuptake inhibitor (SNRI)) prescribed regularly and anxiolytics and hypnotics prescribed as needed. In 2011, quetiapine was prescribed with the intention to decrease anxiolytics utilization and because of the expected therapeutic effect on hyperarousal symptoms. However, PE and urinary urgency persisted and urological evaluation revealed no organic basis for the difficulties.

Finally, in 2014, he came to the regular control expressing his concerns about the adverse effects of the therapy. When I encouraged him to talk about his satisfaction with his sexual functioning, he revealed with great discomfort that he had been suffering from PE for years. He explained that he was usually very tense and anxious about his sexual performance because he could not maintain the time taken to ejaculate during vaginal penetration (i.e. the intravaginal ejaculation latency time (IELT)) longer than for a few seconds, and sometimes he ejaculated even before the penetration. His problem was generalized and present even when he masturbated. He masturbated rarely and in a compulsory manner trying to get it done as quickly as possible just to relieve tension. He perceived vegetative hyperarousal as a reminder of his traumatic experiences with subsequent intrusive memories, hypervigilance and even more anxious feeling. He developed strategies to suppress and repress all negative and uncomfortable feelings, thoughts and memories and to avoid all the reminders as much as he could. His sexual desire was decreasing as well as his sexual activity. Due to constraints resulting from his traditional cultural background, he was unwilling to speak about the problems with his wife. Their relationship was stable, but insufficient in in-depth communication about intimate issues, feelings, emotional needs, relational problems and in particular sexuality. Physical intimacy such as kissing, touching, hugging was sparse. The patient was also reluctant to engage in any diagnostic or therapeutic intervention beyond the acceptance of conversion of the drugs. For this reason and in view of the known ejaculation-delaying effect of paroxetine and its scientifically proven therapeutic effects in patients with PTSD, this medication was considered the best therapeutic option and was administered in a targeted dose of 30 mg per day. As a result, the patient was calmer, his sexual functioning improved, ejaculation was delayed, and urinary urgencies became much less frequent than in the previous period. However, in December 2014, problems with erectile functioning began. He did not complain about this until March 2015, when complete loss of erection occurred even during the sleep. In that period, multimodal antidepressant vortioxetine was introduced in the Croatian market and its good characteristics considering the very low incidence of sexual dysfunction during the treatment were argument enough for choosing this medication. The switching was done by cross-tapering with paroxetine during the period of 10 days with a targeted dose of vortioxetine of 10 mg. The patient also used 100mg of quetiapine per day. He experienced an improvement of erectile functioning, but during the first subsequent sexual intercourse, which occurred 20 days after the introduction of the drug, the erection continued after the intercourse ended in the absence of any physical and psychological stimulation. Unfortunately, the priapism was complicated with paraphimosis. The patient found his troubles humiliating and shameful, and giving that the problems occurred during the Easter period, he was reluctant to seek medical help. He stopped taking vortioxetine immediately. Seven days later, the pain was becoming unbearable so he finally went to the hospital. Urgent incision of the prepuce was performed by the urologist because of the incipient necrosis of the glans penis. After the procedure, oral antibiotics and daily sterile dressing were administered. In the meantime, recommended elective circumcision was performed. Several months later, short cognitive-behavioral therapy was conducted with the focus on psychoeducation, overcoming relational obstacles, relaxation and distraction techniques. The patient was not willing to engage in other psychotherapeutic modalities such as sexual therapy and was reluctant to include his wife in the therapy, but he accepted to talk about sexuality and the sexual dysfunction with his wife, especially after the surgical intervention. His wife and he gradually opened to each other expressing their needs and willing to improve their relationship, spending much more time together and becoming more physically intimate. To date, the patient has achieved a substantial improvement in his sexual life with periodic worsenings (lasting several weeks), particularly during the period of the anniversary of the trauma, when he temporarily withdrew himself from the social interactions and expected to be left alone The patient has provided his written informed consent for the publication of this case report.

DISCUSSION

We presented this case to contribute to the discussion on sexual dysfunction among veterans with PTSD. The problem is often overlooked clinically and

underexamined in scientific studies. PTSD and sexual dysfunction following trauma exposure share many of the same neurobiological processes (Yehuda et al. 2015). In PTSD, the sympathetic nervous system (SNS) is in the state of sustained hyperactivity, as evidenced by elevations in the heart rate, blood pressure, skin conductance, and other psychophysiological measures (Sherin & Nemeroff 2011, Yehuda et al. 2015). The optimal level of SNS activation is also required for healthy sexual functioning. Sexual stimuli activate the SNS, increasing the oxygen uptake and blood flow from the heart (Lorenz et al. 2012). The appropriate response of the hypothalamic-pituitary-adrenal (HPA) axis is crucial in the stress response. Although stressors activate the HPA axis, studies in combat veterans with PTSD demonstrate decreases in cortisol concentrations (Resnick et al. 1995, Delahanty et al. 2000, Meewisse et al. 2007, Bremner et al. 2008). The reduced cortisol activity facilitates a prolonged SNS response and increased levels of catecholamines (such as noradrenaline or adrenaline) (Yehuda 2009, Sherin & Nemeroff 2014, Yehuda et al. 2015). Intrusion symptoms of PTSD such as recurrent memories, flashbacks and nightmares are directly related to elevated catecholamines as they are linked to "overconsolidation" of the traumatic memories (Dębiec et al. 2011).

Sexual stimuli generally produce a decrease in cortisol, allowing increment of catecholamines such as noradrenaline and consequent sexual arousal and activity. In patients with PTSD, hyperactivated SNS induce overly high levels of catecholamines producing a generalized fear response that is sexually inhibitory (Geracioti et al. 2001). Serotonin is a monoamine neurotransmitter which is included in the modulation of the HPA axis and noradrenaline responses during exposure to stress. Decreased serotonergic activity compromises anxiolytic effects, increases vigilance, startle, impulsivity, and memory intrusions (Sherin & Nemeroff 2011). This is the neurobiological basis for treatment of core symptoms of PTSD with SSRI, which are repeatedly confirmed by numerous scientific studies as a first-line pharmacotherapeutic treatment option. Among other medicines from this pharmacological group, paroxetine has the most prominent ejaculation-delaying effect (Segraves 2006), caused by its impact on serotonergic receptors, cholinergic receptor blockade effect and inhibition of nitric oxide synthase (Waldinger et al. 1998, Waldinger 2002, Waldinger & Olivier 2005, Rowland et al. 2010)

As a consequence of the processes described above, it is probable that our patient got stuck in the interwoven neurophysiological pathways of PTSD and sexual dysfunctions. The physiological arousal required in sexual activity was not accompanied by inhibition of fear and stress response, which could be the reason why every sexual arousal was perceived as threat and danger, with symptoms of tension, anxiety, hypervigilance and intrusion, resulting in the urge to overcome and resolve this state with early or premature ejaculation. The applied combined treatment had a limited and unsatisfactory effect on PTSD symptoms so premature ejaculation persisted. Paroxetine with its potent effect on sexual functioning could have had a transitory positive delaying effect on ejaculation, but adverse effects occurred very soon, manifested in complete erectile dysfunction. Vortioxetine, another modulator of serotonergic function, was administered, particularly owing to its favorable tolerability profile with regard to low sexual dysfunction. Regrettably, the improvement in the erectile function was complicated with priapism and paraphimosis, but this serious condition was gradually overcome by the partners' willingness to interact more closely with each other, with the resulting improvement of sexual functioning and reduction of PTSD symptoms.

CONCLUSION

Our case report confirms that PTSD is a complex set of multidimensional domains (Jakovljević et al. 2012). Although sexual dysfunction is not a specific symptom of PTSD, it is a frequent comorbid disorder which requires a transdisciplinary comprehensive treatment approach. As sexuality is one of the crucial domains of the quality of human life further research on the comorbidity and treatment of sexual dysfunction and PTSD is warranted.

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Contribution of individual authors:

Marina Letica-Crepulja: developed the study concept and design;

Marina Letica-Crepulja, Tanja Grahovac-Juretić, Sanja Katalinić, Tomislav Lesica, Ika Rončević-Gržeta & Tanja Frančišković: performed acquisition of data, data analysis and interpretation;

Marina Letica-Crepulja: drafted the manuscript;

Marina Letica-Crepulja, Tanja Grahovac-Juretić, Sanja Katalinić, Tomislav Lesica, Ika Rončević-Gržeta & Tanja Frančišković: made the critical revision of the manuscript and final approval of the version to be published.

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