Lisdexamfetamine Dimesylate (Vyvanse) for the Treatment of Neurogenic Anejaculation

American Journal of Men's Health
2017, Vol. 11(3) 618–619
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DOI: 10.1177/1557988316658640
journals.sagepub.com/home/JMH
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Dear Editor:

We report the first successful pharmacologic treatment of neurogenic anejaculation with lisdexamfetamine dimesylate (Vyvanse) in a 22 year-old Caucasian male with a history of pediatric pelvic neuroblastoma. At 2 years of age, the patient was found to have a pelvic mass abutting the sacrum and iliac vessels. Surgical excision required dissection along the sacral prominence and sacral nerve roots. He did not undergo chemotherapy or radiation as part of his treatment course for neuroblastoma. The patient was referred to our fertility clinic for a lifelong history of anejaculation and aspermia with normal sensation of climax. The patient described a single small volume ejaculation several years prior following administration of lisdexamfetamine dimesylate (Vyvanse). The patient denied problems with libido, erectile dysfunction, and orgasm. Physical exam was unremarkable and revealed normal testicular volume of 20 cc bilaterally. He underwent hormonal evaluation and was found to have normal serum total testosterone of 720 ng/dL, follicle-stimulating hormone 5.8 mIU/mL, and luteinizing hormone 3.8 mIU/mL. A postejaculate urinalysis did not reveal sperm. A pelvic magnetic resonance imaging study was unremarkable. Given his history, we elected to try a short course of intermittent dosing of lisdexamfetamine dimesylate (Vyvanse) 60 mg 2 hours prior to masturbation. With the first use of the medication he produced an antegrade ejaculate. Following a short trial of lisdexamfetamine dimesylate, he returned to clinic for a semen analysis, which demonstrated low volume asthenoteratospermia (Table 1). He reported no adverse events, but did note a subjective temporary decrease in his erectile rigidity, which reversed after the drug was metabolized. This case is the first reported use of lisdexamfetamine dimesylate (Vyvanse) for the treatment of neurogenic anejaculation.

Anejaculation is a rare cause of male factor infertility and can result in significant psychoemotional distress. Etiologies of neurogenic anejaculation include spinal cord injury, low abdominal or pelvic surgery such as retroperitoneal lymph node dissection, diabetes mellitus, and other diseases causing peripheral neuropathy, myelodysplasia, multiple sclerosis, and stroke or traumatic brain injury.

Table 1. Semen Analysis.

Test	Value
Volume	0.7 mL
Sperm concentration	47×10^6/mL
Total sperm count	32.9×10^6
Percent motility	33%
Total motile count	10.9×10^6
Kruger strict morphology	1%

Treatment approaches include pharmacologic strategies, penile vibratory stimulation, and electroejaculation, often tried in that order.

Off-label pharmacologic treatments have been attempted based on knowledge of anatomic and physiologic considerations. Emission is under neural control originating from the thoracolumbar spine at the T10-L2 level. Sympathetic efferent fibers form the lumbar sympathetic trunk ganglia then travel posterior to the inferior vena cava in the interaortocaval region and continue inferiorly and coalesce to form the superior hypogastric plexus anterior to L5 and the sacrum. Ultimately, postganglionic fibers travel to their target organs including the seminal vesicles, bladder neck, prostate, and vasa deferentia to combine to mediate sympathetic control during the emission phase (Safarinejad, 2009).

While the exact mechanisms of neurotransmitter control over ejaculation have not been fully elucidated, dopaminergic pathways have frequently been associated with ejaculatory function. Parkinson's disease patients treated with L-DOPA have reported hypersexuality with more frequent masturbation, sexual hallucinations, and

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increased nocturnal erections. Patients treated with dopaminergic antagonists for schizophrenia may report ejaculatory delay (Barazani, Stahl, Nagler, & Stember, 2012; Ohl, Quallich, Sonksen, Brackett, & Lynne, 2008). Pharmacologic therapy for non–spinal cord injury patients has centered on the off-label use of α-agonist drugs including pseudoephedrine, phenylephrine, imipramine, ephedrine, milodrine, and midodrine (Kamischke & Nieschlag, 2002; Safarinejad, 2009). While pharmacologic treatment does not always produce normal ejaculation, it may allow for partial antegrade ejaculation or retrograde ejaculation.

Lisdexamfetamine dimesylate (Vyvanse) is a medication commonly prescribed to children and adolescents with attention deficit hyperactivity disorder. Lisdexamfetamine dimesylate is a prodrug that is converted by the liver to the active dextroamphetamine (a noncatecholamine, sympathomimetic amine), which causes release of dopamine and norepinephrine from the presynaptic nerve terminals and blocks catecholamine re-uptake by competitive inhibition (Banaschewski et al., 2014). In theory, this release of dopamine and norepinephrine should potentiate the sympathetic receptors mediating seminal emission. The drug is generally well tolerated with a minimal side effect profile, and its treatment effectiveness for attention deficit hyperactivity disorder and tolerability has been validated in double blinded, placebo-controlled studies (Banaschewski et al., 2014).

In summary, our case experience and literature review suggest that lisdexamfetamine dimesylate (Vyvanse) may be a viable off-label pharmacologic treatment option for the treatment of neurogenic anejaculation. Further studies are required to confirm these results.

Declaration of Conflicting Interests

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Funding

The author(s) received no financial support for the research, authorship, and/or publication of this article.

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