

Cabergoline in the Treatment of Male Orgasmic Disorder—A Retrospective Pilot Analysis



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ABSTRACT

Introduction: Male orgasmic disorder is common, with few treatment options. Cabergoline is a dopamine agonist that acts centrally to normalize serum prolactin that could improve orgasmic dysfunction.

Aims: To determine whether cabergoline increases the potential for orgasm in men with orgasmic disorder.

Methods: A retrospective chart review of men treated in a single andrology clinic for delayed orgasm or anorgasmia in a pilot study using cabergoline 0.5 mg twice weekly was performed. Duration of treatment and response were noted. Medical records were examined for other factors including history of prostatectomy and concomitant androgen supplementation.

Main Outcome Measures: Subjective improvement in orgasmic function resulting from cabergoline treatment.

Results: Of 131 men treated with cabergoline for orgasmic disorder, 87 (66.4%) reported subjective improvement in orgasm and 44 (33.6%) reported no change in orgasm. Duration of therapy ($P = .03$) and concomitant testosterone therapy ($P = .02$) were associated with a significant positive response to cabergoline treatment. No differences were found between injectable and non-injectable testosterone formulations ($P = .90$), and neither age ($P = .90$) nor prior prostatectomy ($P = .41$) influenced the outcome of cabergoline treatment. Serum testosterone levels before ($P = .26$) and after ($P = .81$) treatment were not significantly different in responders vs non-responders.

Conclusion: Cabergoline is a potentially effective and easy-to-administer treatment for male orgasmic disorder, the efficacy of which appears to be independent of patient age or orgasmic disorder etiology. Prospective randomized trials are needed to determine the true role of cabergoline in the treatment of this disorder.

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Key Words: Anorgasmia; Male Orgasmic Disorder; Orgasm; Cabergoline

INTRODUCTION

Male orgasmic disorder is defined as the persistent or frequent absence of orgasm during normal sexual arousal and activity. The prevalence of delayed orgasm or anorgasmia in the general

population of men is approximately 8%,¹ and known causes include medical conditions such as diabetic neuropathy or prolactinoma, hypogonadism, psychological disorders, medications, and genitourinary procedures including prostatectomy.^{1–5}

Men undergoing radical prostatectomy (RP) are at risk for sexual dysfunction, most commonly manifesting as erectile dysfunction. However, patients undergoing RP also are at significant risk for developing orgasmic disorder. One study reported anorgasmia in 39.7% of patients after RP, with an additional 38.1% reporting less satisfying orgasms.⁶ Another study observed decreased orgasm intensity in 37% of patients after RP and anorgasmia in an additional 37%.⁷ Because sexual dysfunction after RP has been shown to affect quality of life negatively, especially in younger patients, treatment options should be considered.⁸

Although the definitive etiologies of male orgasmic disorders are unknown, prolactin is believed to influence the likelihood of

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orgasm in men; high prolactin levels have been associated with delayed orgasm, and low levels have been associated with premature ejaculation.⁹ Prolactin levels in the lowest part of the normal range have been associated with premature ejaculation in the general population and in men presenting for sexual dysfunction.^{10,11} Thus, orgasmic disorders likely represent a continuum of symptoms linked to hormone levels. As part of the normal male sexual response, a prolactin surge occurs after orgasm and lasts approximately 1 hour, resulting in a decrease in erectile and ejaculatory potential during this refractory period.^{3,4,12,13} Inhibition of prolactin could, by similar mechanisms, aid with the resolution of delayed or absent orgasm.^{1,14}

Cabergoline is a dopamine agonist with a long half-life (63–69 hours) and has high affinity for the D₂ receptor.¹⁴ The drug is used for treatment of Parkinson's disease and hyperprolactinemia,^{14,15} normalization of serum prolactin and sexual drive in hyperprolactinemic men,¹⁶ and effective treatment of psychogenic erectile dysfunction in otherwise healthy men.¹⁴ Side effects are rarely reported but include gastrointestinal upset, headache, and valvulopathy.¹⁴ The efficacy of cabergoline in the treatment of healthy men with orgasmic disorder has not been evaluated, although it might be an effective treatment for this condition because of its inhibitory effect on prolactin, thereby increasing the potential for orgasm.

AIMS

The aim of this study was to explore a potential role for cabergoline in the treatment of male orgasmic disorder and to identify factors that could modify therapeutic efficacy.

METHODS

Patient Selection

After obtaining institutional review board approval, retrospective chart review was performed for men treated for delayed orgasm or anorgasmia using cabergoline 0.5 mg twice weekly during an off-label pilot study from 2010 through 2013 in a single andrology clinic at an academic medical institution staffed by a single attending physician. All patients were informed of the off-label use of cabergoline, its mechanism of action, and associated potential adverse effects and provided verbal consent for treatment. Men were excluded if they had a history of cabergoline use unrelated to orgasmic dysfunction or for lack of follow-up while on cabergoline. Duration of treatment; subjective quality of orgasm defined using a self-reported three-point scale consisting of (i) no orgasm, (ii) diminished orgasm, or (iii) satisfactory orgasm before and after treatment; possible medical causes of anorgasmia; and history of genitourinary surgeries were determined. History, type, and duration of prior androgen supplementation also were recorded. Men treated for hypogonadism were on concomitant testosterone therapy. Hypogonadism was diagnosed using the presence of clinical symptoms and serum testosterone levels lower than 300 ng/dL.

Testosterone therapy was in the form of transdermal gels or intramuscular injections, with dosage adjusted based on clinical effect and serum testosterone levels.

Serum testosterone, free testosterone, estradiol, SHBG, follicle-stimulating hormone, luteinizing hormone, and prolactin levels were determined before and after cabergoline treatment as part of routine patient care (Supplementary Table 1). All samples were analyzed in an on-site clinical laboratory at our institution using enzyme-linked immunoassay on a single Beckman Access 2 (Beckman Coulter, Brea, CA, USA) analysis platform. Patients presented for follow up every 3 to 6 months.

Data Analysis

Continuous variable data were analyzed with a linear regression multivariate model using IBM SPSS 22 (IBM Corp, Armonk, NY, USA) and Excel 14.3.9 (Microsoft Corp, Redmond, WA, USA). Dependent variables and categorical variables were compared using two-tailed Student t-test and χ^2 test.

MAIN OUTCOME MEASURES

The primary outcome of the study was the change in subjective orgasmic response of men with orgasmic disorder treated using cabergoline. Secondary outcomes included serum testosterone and prolactin levels as a function of response to cabergoline and variables that influenced therapeutic efficacy of cabergoline.

RESULTS

In total, 166 men on cabergoline therapy were identified in a single andrology practice from 2010 through 2013. Of these, 17 were excluded for treatment unrelated to delayed orgasm or anorgasmia (eg, hyperprolactinemia). An additional 18 patients were excluded for lack of follow-up while on cabergoline. The characteristics of the remaining 131 patients are listed in Table 1. Pre- and post-treatment hormone data are listed in Table 2. The median age (interquartile range [IQR]) within the cohort was 61 years (50–69). Notably, 23 men (17.6%) with orgasmic disorder had undergone prior prostatectomy and 86 (65.6%) were treated with concomitant testosterone therapy.

The median duration (IQR) of cabergoline treatment was 9.8 months (5.4–13.5). Of the 131 men treated with cabergoline, 88 (66.4%) reported subjective improvement in orgasm and 44 (33.6%) reported no change in orgasm. Of the 88 men with improvement in orgasm, 59 (45.0%) had a return to normal orgasm after therapy, with the remaining 28 (21.4%) showing improvement without complete return to baseline orgasmic function. The median duration (IQR) of therapy for non-responders and responders to cabergoline was 7.3 months (5.1–12.5) and 10.3 months (6.2–14.5), respectively ($P = 0.04$). There was no difference in treatment outcomes for men presenting with anorgasmia vs delayed orgasm.

Table 1. Patient Demographics

	All patients	Cabergoline responders	Cabergoline non-responders	<i>P</i> value*
Patients, n (%)	131 (100)	87 (66.4)	44 (33.6)	
Age (y), median (IQR)	61 (50–69)	61 (50–68)	61 (51.5–69.25)	.92
Treatment duration (mo), median (IQR)	9.8 (5.4–13.5)	10.3 (6.2–14.5)	7.3 (5.1–12.5)	.04
Prior prostatectomy, n (%)	23 (17.6)	17 (19.5)	4 (9.1)	.41
Testosterone formulation, n (%)	87 (66.4)	51 (58.6)	36 (81.8)	.90
Injectable	38 (29.0)	22 (25.3)	16 (36.4)	
Non-injectable	49 (37.4)	29 (33.3)	20 (45.5)	

IQR = interquartile range.

*Comparison between cabergoline responders and non-responders.

Of the 87 men undergoing concomitant testosterone therapy during cabergoline treatment, 51 were responders and 36 were non-responders ($P = .02$) to cabergoline, accounting for 58.6% of all responders and 81.8% of all non-responders. Median serum testosterone levels before (414.0 ng/dL, IQR = 302.3–629.3) and after (506.5 ng/dL, IQR = 329.5–922.5) treatment with cabergoline were available in 29 men, including 19 responders and 10 non-responders. The median testosterone levels in responders before (415.0 ng/dL, IQR = 303.0–690.0) and after (564.5 ng/dL, IQR = 330.5–891.8) treatment and in non-responders before (367.0 ng/dL, IQR = 303.0–612.0) and after (496.0 ng/dL, IQR = 335.8–891.8) treatment were not significantly different before ($P = .26$) or after ($P = .81$) treatment. A significant difference was found between prolactin levels before and after treatment in responders ($P < .0001$) but not in non-responders ($P = .56$).

Univariate and multivariate analyses showed that duration of cabergoline therapy and patients being on testosterone therapy increased the likelihood of response to cabergoline (Table 3). In contrast, age ($P = .997$), history of prostatectomy ($P = .157$), hormone levels before and after the survey, psychotropic drug use other than cabergoline, and prolactin levels were not associated with improvement in orgasm.

DISCUSSION

Several studies examining the endocrine response to sexual arousal and orgasm by Exton et al provide a foundation for the efficacy of cabergoline in the setting of male orgasmic

dysfunction. Initially, they showed that sexual arousal without orgasm resulted in increased blood pressure and plasma norepinephrine but without concomitant increases in other catecholamines or in prolactin.¹⁷ Subsequently, they continuously measured serum prolactin and catecholamine levels during intercourse in men and women and observed that prolactin levels increased after orgasm and remained elevated for 1 hour.¹⁸ Then, they showed that masturbation-induced orgasm had the same effect on plasma prolactin levels,¹⁹ leading to the theory that prolactin could contribute to the refractory period after orgasm in which arousal and repeated orgasm are more difficult. Intriguingly, in men who report short or absent refractory periods, a prolactin surge might not occur after orgasm.¹²

Previous efforts to treat male orgasmic dysfunction have focused on delayed or absent orgasm caused by selective serotonin reuptake inhibitors or other psychotropic medications. Early efforts used amantadine, an indirect dopamine agonist, which has been shown to stimulate sexual activity in rats but requires pre-coital dosing in humans to achieve effect.³ Other reports or small case series have described the use of pseudoephedrine, bupropion, buspirone, and yohimbine, but there is a paucity of data demonstrating the efficacy of these treatments.³

A recent report described resolution of idiopathic anorgasmia in an 82-year-old man using oxytocin.² Prior studies have indicated that oxytocin levels increase during arousal and peak during orgasm.²⁰ However, the 2- to 3-minute half-life of oxytocin necessitates intranasal administration during intercourse at the point when orgasm is desired, a significant inconvenience to the patient.²

Table 2. Serum Testosterone and Prolactin Levels Before and After Cabergoline Treatment*

	All patients	Cabergoline responders	Cabergoline non-responders	<i>P</i> value†
Testosterone (ng/dL)				
Before cabergoline	414.0 (302.3–629.3)	415.0 (303.0–690.0)	367.0 (303.0–612.0)	.26
After cabergoline	506.5 (329.5–922.5)	564.5 (330.5–891.8)	496.0 (335.8–891.8)	.81
Prolactin (ng/dL)				
Before cabergoline	6.2 (5.0–8.5)	7.2 (6.0–9.7)	5.5 (4.6–6.6)	.04
After cabergoline	1.1 (0.4–7.6)	0.6 (0.3–6.9)	3.6 (0.6–8.3)	.24

*All values are presented as median (interquartile range).

†Comparison between cabergoline responders and non-responders.

Table 3. Factors Associated with Improvement in Orgasm

	β	95% CI	P value
Univariate analysis			
Age	0.000	−0.026 to 0.027	.997
Duration of cabergoline therapy	0.002	0.000–0.004	.042
Before prolactin (ng/mL)	−0.004	−0.115 to 0.106	.937
Prolactin (ng/mL)	0.079	−0.043 to 0.202	.205
Total testosterone (ng/dL)	0.000	−0.001 to 0.001	.810
Free testosterone (ng/dL)	−0.007	−0.039 to 0.250	.659
Estradiol (ng/dL)	0.038	−0.038 to 0.115	.327
On testosterone therapy (reference = none)	−1.261	−2.178 to −0.344	.007
Psychotropic medications (reference = none)	−0.413	−1.178 to 0.353	.290
History of prostatectomy (reference = none)	0.836	−0.323 to 1.995	.157
Multivariate analysis			
Duration of cabergoline therapy	0.002	0.000–0.005	.031
On testosterone therapy (reference = none)	−1.330	−2.265 to −0.396	.005

Cabergoline has been used historically to treat sexual dysfunction, but not male orgasmic disorder, although its mechanism of action would seem reasonable for its use in treatment of men with orgasmic disorder. Several dopamine agonists, including cabergoline, have been shown to improve erectile function and libido in patients with Parkinson disease,²¹ and cabergoline is useful in treating sexual dysfunction in hyperprolactinemic men.¹⁶ Cabergoline also has been used to treat psychogenic erectile dysfunction in young, healthy men.¹⁴ Based on the supporting evidence, we used cabergoline in an off-label pilot study to treat male orgasmic disorder and found significant improvement in orgasmic function in these men using a self-reported un-validated metric.

Uni- and multivariate analyses demonstrated that men on testosterone therapy were more likely to respond to cabergoline treatment, in line with prior findings that orgasmic disorders occur more frequently in hypogonadal men, and that normalization of testosterone levels might ameliorate orgasmic dysfunction.²² Because age and history of prostatectomy do not influence the efficacy of cabergoline, its use might be applicable in all men with orgasmic disorder. In addition to erectile preservation therapy, treatment of orgasmic disorder could be an important future consideration for maintaining sexual function in men after RP, thereby increasing their quality of life.

This study does have limitations that affect its generalizability to all men with orgasmic disorder. First, our study is not placebo controlled or randomized, introducing the possibility that the purported efficacy of cabergoline in this setting might be a placebo effect. Second, our sample is relatively small, limiting the generalizability of the study. Third, the significant rate of coadministration of testosterone could confound the effects of cabergoline, although most of these patients were on testosterone therapy without resolution of orgasmic disorder before administration of cabergoline, and serum testosterone levels did not differ between responders and non-responders. In addition, a larger proportion of non-responders used testosterone compared with responders, and longitudinal testosterone data were not available for the entire

cohort. Fourth, orgasmic symptoms were not assessed using a validated questionnaire, limiting our ability to assess the nature of symptomatic improvement while on cabergoline. We did not have information on patients' social history including relationship status, which could affect outcomes of therapy with cabergoline. Fifth, all patients were seen in a single andrology clinic at a tertiary referral academic medical center and might not be typical of men with orgasmic disorder in the general population. Future, larger, randomized controlled studies could serve to evaluate more rigorously the efficacy of cabergoline in male orgasmic disorder while avoiding these limitations.

CONCLUSIONS

To our knowledge, this is the first report showing the efficacy of cabergoline in treating delayed orgasm and anorgasmia in men, regardless of patient age or whether the etiology is idiopathic or secondary to prostatectomy. Although randomized controlled studies are necessary to clarify the magnitude of the effect of cabergoline in these men, it potentially represents an easy-to-administer treatment option for delayed orgasm or anorgasmia, with minimal side effects.

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Supplemental Table 1. Hormone Data*

	All patients	Cabergoline responders	Cabergoline non-responders
Testosterone (ng/dL)			
Before cabergoline	414.0 (302.3–629.3)	415.0 (303.0–690.0)	367.0 (303.0–612.0)
After cabergoline	506.5 (329.5–922.5)	564.5 (330.5–891.8)	496.0 (335.8–891.8)
Free testosterone (ng/dL)			
Before cabergoline	10.7 (7.2–60.2)	11.47 (7.3–60.2)	9.30 (6.9–27.2)
After cabergoline	13.97 (9.8–62.5)	14.21 (10.4–62.5)	13.58 (8.8–41.5)
Prolactin (ng/mL)			
Before cabergoline	6.2 (5.0–8.5)	7.2 (6.0–9.7)	5.5 (4.6–6.6)
After cabergoline	1.1 (0.4–7.6)	0.6 (0.3–6.9)	3.6 (0.6–8.3)
Estradiol (ng/dL)			
Before cabergoline	4.95 (3.0–83.0)	5.84 (3.0–83.0)	3.31 (3.0–10.0)
After cabergoline	5.71 (4.0–55.0)	6.63 (4.0–55.0)	4.06 (3.0–18.0)
FSH (mIU/mL)			
Before cabergoline	7.04 (3.0–104.0)	6.39 (3.0–72.0)	8.30 (3.0–104.0)
After cabergoline	6.64 (1.5–98.0)	6.99 (3.0–74.0)	6.10 (0.8–98.0)
LH (mIU/mL)			
Before cabergoline	3.59 (2.0–44.0)	3.47 (2.0–44.0)	3.83 (2.0–40.0)
After cabergoline	3.69 (0.7–51.0)	3.68 (1.0–41.0)	3.69 (0.5–51.0)
SHBG (nmol/L)			
Before cabergoline	36.61 (34.0–94.0)	36.01 (34.0–89.0)	37.70 (34.0–94.0)
After cabergoline	35.60 (32.0–84.0)	35.18 (32.0–84.0)	36.35 (34.0–84.0)
DHEA-S (ng/mL)			
Before cabergoline	1,396.96 (1,054.0–7,753.0)	1,524.13 (1,194.0–7,753.0)	1,156.25 (878.5–3,741.0)
After cabergoline	1,296.36 (824.0–8,552.0)	1,374.63 (824.0–8,552.0)	1,181.75 (840.0–6,422.0)

DHEA-S = dehydroepiandrosterone sulfate; FSH = follicle-stimulating hormone; LH = luteinizing hormone.

*All values are presented as median (interquartile range).