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CASE REPORT

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Obstructive sleep apnea as a potential contributor to stuttering priapism: a case series

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Abstract

Background: The etiologies and management of patients with stuttering priapism remain somewhat perplexing, with most cases being idiopathic, however the underlying molecular basis for some causes, such as sickle cell disease, is believed to be due to the depletion of nitric oxide and resulting dysfunction of cyclic guanosine monophosphate (cGMP)-induced vasodilation of corporal smooth muscle. We propose a potential mechanism for stuttering priapism in patients with obstructive sleep apnea (OSA) and report on patients with OSA and success (or lack thereof) in their management at our institution. **Cases**: A brief query of the institution's electronic medical record was performed from 01 January 2023 to 01 January 2024 through the use of an existing institutional review board approval, and a total of three patients were identified who were treated for stuttering/recurrent priapism with a pre-existing diagnosis of OSA or with high suspicion of OSA. Some of these patients noted a reduction in priapism episodes with improved control of their OSA through means such as continuous positive airway pressure (CPAP) machines. **Conclusions**: Obstructive sleep apnea may be a possible risk factor for stuttering priapism and thus should be considered by the clinician. It shares a similar mechanism to that of sickle cell disease's role in priapism: nitric oxide depletion and PDE5 (phosphodiesterase 5) downregulation resulting in intermittent, unregulated smooth muscle relaxation through non-nitric oxide pathways; thus, this should be evaluated by the clinician.

Keywords

Priapism; Recurrent; Stuttering; Nitric oxide; Erection; Erectile dysfunction; Sleep apnea

1. Introduction

Priapism is a urologic emergency characterized by a partial or full erection lasting longer than 4 hours. If left untreated, priapism can result in long-term erectile dysfunction. There are three kinds of priapism: nonischemic (high flow or arterial), ischemic (low flow or venous), and stuttering (intermittent) [1]. Ischemic priapism accounts for 95% of all cases and has numerous contributing etiologies, including alcohol and drug use, perineal trauma, and/or sickle cell disease (SCD) [1]. However, the majority of cases are idiopathic. In contrast, nonischemic priapism is uncommon and its etiology is mostly attributed to either blunt or penetrating trauma that results in injury to the cavernous artery or its branches within the corpora [2]. Although more rare, nonischemic priapism can result from the conversion of ischemic to nonischemic following aggressive treatment with either aspiration with alpha-adrenergic injections or surgical shunting [3, 4]. Stuttering priapism, which describes a pattern of recurrent priapism, is typically associated with men with SCD and can be precipitated by dehydration, fever, and cold exposure [5]. Stuttering priapism should also be differentiated from sleep-related painful erections (SRPE) in which patients develop recurrent, painful erections during sleep. However, erections during daytime hours are normal, painless, and last less than 4 hours, thus distinguishing this from the aforementioned priapism varieties [6]. In all types of priapism, the pathophysiology involves alterations in the normal process of tumescence and/or detumescence.

During normal penile erections, the relaxation and contraction of smooth muscle are critical for tumescence and detumescence, respectively [7]. This process is regulated by the cyclic intracellular second messenger cyclic guanosine monophosphate (cGMP) which is generated by guanylyl cyclase within smooth muscle cells. Guanylyl cyclase requires nitric oxide (NO), which is released in penile smooth muscle cells upon sexual stimulation [8]. cGMP results in smooth muscle relaxation, thereby resulting in tumescence. cGMP is catabolized by phosphodiesterase 5 (PDE5), which is an enzyme found primarily in the corpus cavernosum smooth muscle [9]. The action of PDE5 contributes to detumescence. However, the release of nitric oxide, a process necessary for tumescence, can be impaired through various means. This includes states of

chronic illness (vascular disease, diabetes, hypertension) or in states of acute illness (hypoxemia, febrile illness). Hypoxemia, for example, is commonly noted in patients with SCD due to erythrocyte hemolysis, thereby lowering oxygen-carrying capacity. Additionally, elevated hemolytic activity liberates hemoglobin from erythrocytes, which binds directly to NO and thus decreases its bioavailability. Initially, this appears paradoxical given that patients with SCD have decreased NO, and yet SCD is a known risk factor for priapism with up to 40% of these individuals suffering from priapism [10]. However, mice with severely deficient NO production also experience increased rates of priapism. This is attributed to significant downregulation of PDE5 because of chronically low levels of NO. In turn, this results in episodic uncontrolled cGMP-induced vasodilation of penile smooth muscle, as there is insufficient PDE5 to process cGMP at a sufficient rate. Although typically produced via NO-dependent pathways, cGMP can also be produced by guanylyl cyclase in NO-independent pathways. This may explain, in part, why long-term PDE5 inhibitor use can be a preventative strategy for recurrent idiopathic priapism in individuals with and without SCD [11].

Similar to patients with SCD, those with obstructive sleep apnea (OSA) experience hypoxemia due to repetitive upper airway obstruction during sleep [12]. Sleep apnea is defined as $\geq$5 apnea/hypopnea events per hour. This is also commonly referred to as the apnea-hypoapnea index (AHI) [13]. Based on this cutoff of AHI $\geq$5, the overall population prevalence ranges from 9–38%. Other studies, which evaluated global prevalence, estimate that 900 million adults suffer from OSA [14]. However, the prevalence increases with risk factors, such as male gender, obesity, and increasing age. Prevalence reaches as high as 90% in some elderly male groups within the United States. The prevalence of the disorder has increased over time, with more recent epidemiological studies indicating prevalences >30% in the general population. The gold standard of diagnosis is polysomnography (or sleep study) which can be done under supervision at a facility or at home; however, there do exist screening questionnaires which can facilitate clinical evaluation in patients who may lack easy access to a sleep study [15].

One of the most widely used and validated tool is the Stop-Bang questionnaire, which is named as acronym for the eight questions it poses (Snoring, Tired, Observed during Sleep, Blood pressure, Age, Neck circumference, and Gender) [16]. A systematic review demonstrated that the Stop-Bang questionnaire had an 81–97.5% sensitivity for diagnosing OSA with an AHI $\geq$5 [17]. Sleep apnea is a common and potentially significant disorder which significantly impacts one's quality of life, causing fatigue, irritability, depression, and headache. Furthermore, OSA is a risk factor for multiple conditions, such as coronary artery disease, type 2 diabetes, stroke, and cerebrovascular diseases.

Decreased NO is one hypothesis for the increased risk of a variety of cardiovascular conditions in OSA. Oxidative stress due to multiple and repetitive hypoxemic episodes in OSA induces apoptosis and dysfunction of endothelial cells, which are responsible for production and regulation of NO [18]. Furthermore, OSA impairs and down-regulates NO synthase directly through decreased oxygen availability [19]. OSA severity appears to be negatively correlated with overall NO levels. Furthermore, obese patients (body mass index >30) with OSA tend to have even lower levels of NO regardless of OSA severity [20]. Interestingly, the administration of continuous positive airway pressure (CPAP), which is a treatment for OSA, does appear to improve or possibly normalize NO within these patients [21–23]. Although erectile dysfunction is highly prevalent in patients with OSA, as those with high AHI tend to be more likely to have erectile dysfunction, there may be a subset of patients with OSA with good erectile dysfunction who are at higher risk for priapism [24, 25]. This subset of patients is likely younger in age with fewer pre-existing comorbidities. However, no information is available on AHI severity in relation with priapism. However, there exists evidence that patients with SCD who experienced more desaturation episodes at night were at a higher risk of priapism in a small case-control study [7]. Therefore, it is hypothesized that there is a common link between OSA and idiopathic stuttering priapism in patients without sickle cell anemia. Furthermore, it is hypothesized that these patients would see improvement or resolution of their priapism episodes through primary treatment of their sleep apnea.

2. Methods

A retrospective, observational case series of patients was conducted with recurrent priapism with OSA (or highly suspected OSA) at a large, tertiary-care institution. A brief query of the institution's electronic medical record was performed from 01 January 2023 to 01 January 2024, after obtaining an institutional review board (IRB) approval. Any patients with more than 1 episode of priapism that required treatment at our institution were included for review. Patients were excluded if we could not verify episodes of priapism (*i.e.*, the individual only reported them and/or were treated at other institutions). Variables of interest included known diagnosis of OSA, CPAP compliance, testosterone levels (if available), diagnosis of SCD, prior treatment attempts for stuttering priapism (including medical and surgical), and current treatment protocol. As this is a descriptive case series, no formal sample size calculation was performed.

3. Results

3.1 Case 1

A 51-year-old Latino male with history of hypertension (HTN), type 2 diabetes, hyperthyroidism, and OSA was evaluated in the office for recurrent idiopathic priapism. Initially, his history of priapism started in 2013 as an adverse reaction to trazodone. Since then, despite discontinuation of trazodone, the patient has experienced recurrent episodes of priapism at various intervals (occasionally going 6 months without an episode while at other times having several episodes within a 1-month period). Most episodes ranged from 4 hours to upwards of 12 hours. He required multiple emergency room visits that necessitated drainage with concomitant use of intracavernosal phenylephrine and required hospitalization once for a distal shunt conducted within the operating room.

The patient's sickle cell hemoglobin testing was negative. The patient was then eventually placed on ketoconazole for his recurrent priapism after exhausting other options. This was successful for 1 year, but he then discontinued the medication and developed episodes of priapism again. The patient was then placed on a trial of bicalutamide, but he only intermittently used it due to adverse side effects. During this entire duration, the patient could not comply with his CPAP treatment due tolerability issues. He had a repeat sleep study which demonstrated severe sleep apnea. The patient was urged to use a CPAP machine or be fitted for a new one, but he has deferred this. Unfortunately, the patient continues to suffer from stuttering priapism despite most recent testosterone value indicative of hypogonadism (133 ng/dL). He continues to present through the emergency room every 2–3 months for drainage.

3.2 Case 2

A 37-year-old black male with history of HTN and stroke was seen in the office for recent episodes of priapism lasting between 4- and 6-hours, requiring drainage with administration of phenylephrine in the emergency room. His lab test results were notable for a testosterone of 344 ng/dL and negative sickle cell hemoglobin test. Of note, however, the patient reported a long (20+ year) history of poor sleep, loud snoring, headaches after waking, and that other people had observed he has stopped breathing during sleep. The patient was referred to a pulmonologist who performed a screening questionnaire for OSA called Stop-Bang, which resulted in a score of 4 points (intermediate risk for moderate to severe OSA). The patient was then scheduled for and is currently pending a formal sleep study. At the time of last clinic visit, he deferred any medical treatment options for his priapism episodes (such as bicalutamide, low dose sildenafil, *etc.*) and opted to pursue evaluation and treatment of likely OSA.

3.3 Case 3

A 42-year-old black male with history of asthma, generalized anxiety disorder, and OSA was referred for evaluation of recurrent, frequent episodes of ischemic priapism. These episodes occurred every 3–4 days and typically lasted 4–5 hours prior to presentation. They all required drainage with administration of phenylephrine. Of note, the patient had a similar situation 15 years previously which required a distal penile shunt for resolution. The patient's testosterone was 123 and his sickle cell hemoglobin electrophoresis test was negative. The patient had 5 episodes of ischemic priapism over the month preceding his 1st clinic appointment and all of them started during the night while the patient was asleep, thus bringing up a concern for possible SRPE. He denied any inciting agents or new medications. He did admit that he stopped use of his CPAP machine due to the mask being uncomfortable and he did also endorse significant snoring and daytime drowsiness. During his office visit, the decision to initiate bicalutamide was made in addition to increase CPAP compliance. The patient also contacted his pulmonologist to make an appointment for a better fitting CPAP mask. At his 3- and 6-month follow-up appointments, the patient had no further episodes of priapism. Use of bicalutamide gradually tapered during that time period, initially starting with 50 mg per day to 50 mg every other day, and ending with 50 mg once every 7 days. He had also been using CPAP every night consistently during that interval after being re-fitted to a new mask. The patient has not had any further episodes of priapism since initiation of this treatment protocol.

4. Discussion

This report describes three patients with recurrent priapism and with diagnosed or highly suspected sleep apnea. In one of three patients, his priapism no longer recurred after a combination of improved CPAP compliance and gradual taper of bicalutamide. Interestingly, two of three patients were hypogonadal (with testosterone <150) and the 3rd patient was low-normal (344) and yet all suffered from priapism. Given that there is low NO both in patients with sickle cell disease and in those with OSA, it is hypothesized that this results in extremely down-regulated PDE5. Thus, through alternative non-nitric oxide pathways, there can be episodic uncontrolled cGMP-induced vasodilation of penile smooth muscle, thereby resulting in prolonged erection time (Fig. 1). The potential impact of sleep apnea on recurrent priapism should be considered given the high prevalence of OSA. Yet, sleep apnea, despite its impact on both quality of life and co-morbid conditions, remains underdiagnosed in many groups [26]. This may be a potential gap that both the patient and clinician fail to take account of. The Stop-Bang questionnaire could be administered to patients suspected of having sleep apnea or in those with recurrent priapism as an initial screening tool given its high sensitivity.

Given that individuals with OSA have low NO levels and it is theorized that this paradoxically increases their risk of priapism, part of management of these patients should aim to improve or normalize NO levels. Although use of CPAP is associated with improvement or normalization of nitric oxide levels in patients with OSA, it may take up to 12 weeks to act. Thus, patients may also be initiated on antiandrogen therapy consisting of bicalutamide which has been shown to be successful in treatment of refractory priapism [27–29].

There are limitations within this study. Follow-up times for these patients are all less than 1 year, thus long-term results are yet unknown. Second, only 3 patients are included in this study, thereby limiting generalizability to a broader population. As this was a descriptive case series, no formal sample size calculations were performed. Additionally, these patients are from New Jersey's largest public hospital system. Therefore, there may be issues with compliance with appointments, medications, and obtaining sub-specialist care. Patients also may lack the resources, health literacy, or support systems for long-term focused care (such as obtaining a sleep study). Regardless, further research should be focused on this potential link between OSA and priapism given its potential shared molecular mechanism which would mimic that of recurrent priapism within SCD.

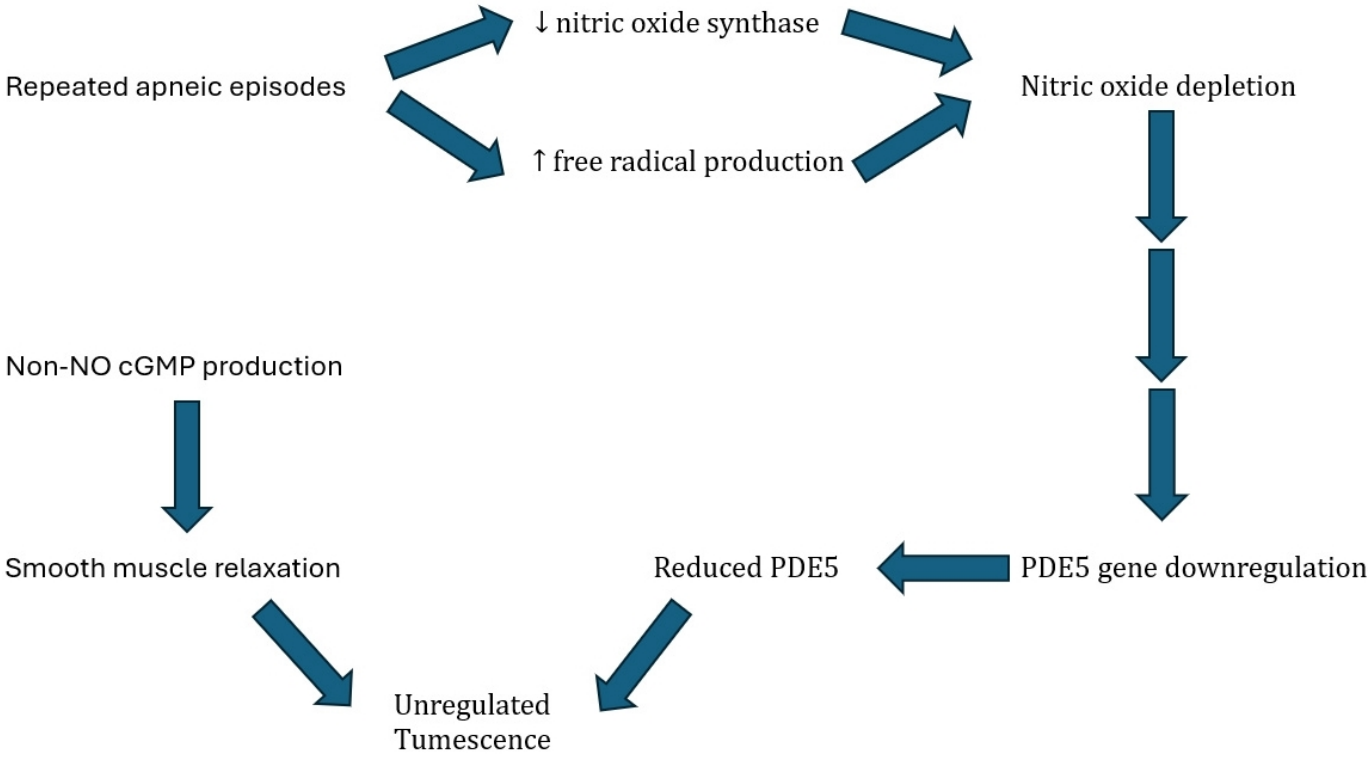

FIGURE 1. Proposed pathway of unregulated tumescence. NO: nitric oxide; PDE5: phosphodiesterase 5; cGMP: cyclic guanosine monophosphate. ↓: decreased activity; ↑: increased activity.

5. Conclusions

Obstructive sleep apnea may be a possible risk factor for stuttering priapism and thus should be considered by the clinician. This is particularly important given the prevalence of obstructive sleep apnea and how many individuals are relatively unaware of their condition or its severity. It shares a similar mechanism to that of sickle cell disease's role in priapism: nitric oxide depletion and PDE5 downregulation resulting in intermittent, unregulated smooth muscle relaxation through non-nitric oxide pathways and thus, this should be evaluated by the clinician.

ABBREVIATIONS

OSA, obstructive sleep apnea; NO, nitric oxide; SCD, sickle cell disease; SRPE, sleep-related painful erection; PDE5, phosphodiesterase 5; CPAP, continuous positive airway pressure; AHI, apnea-hypoapnea index; HTN, hypertension; cGMP, cyclic guanosine monophosphate; IRB, institutional review board.

AVAILABILITY OF DATA AND MATERIALS

The data utilized in this project was obtained from a single institution's electronic medical record. All relevant data was included in the body of this manuscript. There was no statistical analysis conducted.

AUTHOR CONTRIBUTIONS

AP—designed the research study; performed the research and data collection; and wrote the manuscript. KJ, MP and EG—all performed data collection and assisted in writing the manuscript. AA—designed the research study. All authors contributed to editorial changes in the manuscript. All authors read and approved the final manuscript.

ETHICS APPROVAL AND CONSENT TO PARTICIPATE

This project received approval from the institutional review board of University Hospital Newark, NJ. (Study ID: Pro2023001466). All patients consented to participate.

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CONFLICT OF INTEREST

The authors declare no conflict of interest.

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