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Treating stuttering priapism

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Stuttering priapism is an uncommon recurrent form of ischaemic priapism consisting of episodes of unwanted, painful erections that typically last for <3 h. It occurs repeatedly with intervening periods of detumescence. If these episodes are not treated, it may evolve into a classic ischaemic priapism and eventually lead to irreversible corporal fibrosis with permanent erectile dysfunction. A comprehensive literature search was conducted in August 2010 using the PubMed database, MEDLINE and generic search engines. The search terms used to source information on this topic were, stuttering priapism (44 hits) and recurrent priapism (161 hits). Although there are numerous publications on this topic the majority of them are small trials and case reports. We identified 117 case reports, 28 reviews, 37 anecdotal reports, 22 small size clinical trials and one in vitro work. Our understanding of the underlying pathophysiology of stuttering priapism has improved in recent years. Further multicentre randomized clinical trials are required to evaluate the efficacy of different treatment options and to define safe and effective management strategies for patients with low-flow recurrent priapism.

What's known on the subject? and What does the study add?
Stuttering priapism is a form of ischemic priapism which mainly affects sickle cell patients. Despite recent advances in understanding the aetiology and pathophysiology of this condition, no credible evidence-based management strategy has been produced to this date.

In this mini review we have attempted to summarize the published articles on this topic systematically. The main aim was to present the treatment options available and analyse their indications and efficacies.

KEYWORDS

stuttering priapism, recurrent priapism, mechanism, aetiology, treatment

INTRODUCTION

By conventional definition, priapism is a pathological condition of erectile tissue tumescence characterized by prolonged erection without any sexual stimulation and excitement [1,2]. The penis is recognized as being the affected body organ, although priapism of the clitoris has also been reported in the medical literature [3]. A time interval of 4 h has frequently been cited as a qualifying criterion since, on a practical basis, pathological consequences are associated with priapism extending beyond this time limit [2]; however, presentations of shorter durations, and conversely those lasting days or months that may not overtly result in erectile tissue damage, are identifiable as representing priapism.

A classification commonly used to differentiate clinical presentation of priapism divides priapism into ischaemic (low-flow) and non-ischaemic (high-flow) priapism. They have different pathological consequences [4]. The division also has practical consequences with respect to overall clinical management.

In 1914 Hinman [5] first recognized that some recurrent forms of priapism are suffered for limited durations compared with isolated episodes. He called those recurrent forms acute transitory attacks.

The more familiar term stuttering priapism was devised by Emond *et al.* [6] in reference to the frequent recurrent episodes of priapism observed in patients with sickle-cell disease. Such priapism, which progresses in severity, may be considered to represent a subtype of ischaemic priapism [2].

The underlying pathophysiology of priapism is still somewhat unclear, but it is believed that the molecular mechanisms underlying priapism are similar to those seen in non-pharmaceutical-induced ischaemic priapism [7].The clinical presentation of stuttering priapism is similar to ischaemic priapism but it is typically self-limited, lasting less than 3 h. It is particularly common among patients with sickle-cell disease and, if it remains untreated, can cause severe structural and physiological effects on erectile apparatus [2].

EPIDEMIOLOGY

Estimates of incidence rates of priapism depend on the population group being studied. This matter applies particularly to populations in which sickle-cell disease (a major risk factor for priapism) is highly prevalent. In other adult populations, idiopathic and drug-induced priapism seem to be the commonest aetiologies [8]. Typically there is a bimodal peak of incidence, between 5 and 10 years in children and 20 to 50 years in adults [9].

Epidemiological studies of stuttering priapism are severely lacking. The majority of studies in this area have been conducted in patients with sickle-cell disease. Cohort studies involving populations with sickle-cell disease show that these populations

represent ≈ 28% of all cases of priapism; 42% of adults and 64% of children with sickle-cell disease will eventually develop it. Although high-flow priapism in patients with sickle-cell disease has been reported, the majority of cases are of the low-flow type [10–12].

Adeyoju *et al.* [13] conducted an international multicentre (five centres) study in the UK and Nigeria. They investigated the incidence rate of priapism in patients with sickle-cell disease. In this study 35% of patients reported a history of priapism and of these, 72% had a history of stuttering priapism.

AETIOLOGY AND PHYSIOPATHOLOGY

Stuttering priapism shares its aetiologies with ischaemic priapism. It has been linked with a host of disease states, and a number of other clinical contexts have risk association for developing the disorder. The most common aetiologies associated with stuttering priapism include haematological dyscrasias (mainly sickle-cell disease), idiopathic, and rarely, neurological disorders.

In evaluating the origins of idiopathic priapism, Hinman [4] proposed that vascular stasis in the penis and decreased venous outflow from it were the primary circumstances that mechanically or physically interfered with detumescence. Hinman [14] further reasoned that the deoxygenated blood (with decreased oxygen tension and increased carbon dioxide tension) associated with ischaemia combined with venous congestion, which increases blood viscosity, contributes to the deformity of erythrocytes locally in the penis (particularly in patients with sickle-cell disease) causing veno-occlusive erythrocytes.

The mechanism of ischaemic priapism is the interaction between delicate integrated smooth muscle contraction/relaxation and erectile tissue homeostasis. It is thought that molecular mechanisms underlying stuttering priapism are similar to those implicated in non-pharmaceutical-induced ischaemic priapism, and that the tonically deficient endothelial nitric oxide in the penis can cause the downregulation of cGMP-specific protein kinase I, phosphodiesterase 5 (PDE-5) and Rho A)/Rho-Kinase. In such circumstances, with the smooth muscle cell tone running at a reduced set point, under the influence of sexual or unrelated stimulation, the smooth muscle cells will over-respond with a prolonged erection. With suitable management, detumescence will occur, however, the low set point will remain and as a result this form of priapism occurs repeatedly [7].

HAEMATOLOGICAL DYSCRASIAS

Priapism secondary to sickle-cell disease is reported to account for 42% of adult cases and 64% of paediatric cases. Sickle-cell disease ranks as the most frequent basis for priapism in the paediatric age group (≤18 years of age) [10,15].

Other haemoglobinopathies, e.g. thalassaemia, thrombophilia and haemoglobin Olmsted, have also been associated with priapism. Haematological malignancies, including leukaemia and multiple myeloma, have also been identified for their risk association with priapism. Other thrombotic risk associations with priapism have also been described. These include asplenism, erythropoietin use and haemodialysis with heparin administration and withdrawal of warfarin which are believed to represent rebound hypercoagulable disease states. Total parenteral nutrition, particularly that containing 20% fat emulsions, has been associated with priapism [16].

IDIOPATHIC PRIAPISM

Priapism occurring without any discernible cause is idiopathic. This form of stuttering priapism is considered to be the most common form in adults [8,17]. Although the pathophysiological mechanism of this form of priapism remains unclear, prolonged or painful presentation tends to fit an ischaemic priapism model.

CLINICAL COURSE AND TREATMENT OF STUTTERING PRIAPISM

Recurrent forms of ischaemic priapism follow a distinct natural course in that they occur repeatedly, with intervening periods of detumescence. Patients with haematological abnormalities, such as sickle-cell disease and those with idiopathic presentations, commonly fit this clinical pattern. The frequency can range from several times per day to once every few months, which can be disabling [16]. The episodes appear to bear no relationship to other vaso-occlusive crises that are characteristic of sickle-cell disease [18,19].

The process of corporal fibrosis starts 4 h after the beginning of symptoms; however Kulmala and Tamella [20] show that within 24 h most cases respond to aspiration and α-adrenergic drugs with no consequent corporal fibrosis. Beyond 24 h patients usually do not respond to medication and develop varying degrees of intracavernosal fibrosis.

Stuttering priapism,by definition, is a self-limiting condition usually lasting <3 h. However, a proportion of patients with stuttering priapism develop an episode of full-blown prolonged ischaemic priapism, requiring immediate intervention, e.g. corporal aspiration, aspiration with irrigation and α-receptor agonist penile injection or even surgical shunt procedures for refractory cases [2,9].

The goal of the management of a patient with stuttering priapism is the prevention of future episodes, while the management of each episode should follow the specific treatment recommendations for acute ischaemic priapism as briefly described above. This can be achieved by hormonal manipulation, increasing corpus cavernosum smooth muscle tone, or surgical intervention.

HORMONAL MANIPULATION

The aim of hormonal manipulation for stuttering priapism is to suppress circulating testosterone levels by downregulating the pituitary gland (GnRH agonists or antagonists), inhibiting feedback (diethylstilbestrol), blocking androgen receptors (antiandrogens) and reducing adrenal and testicular testosterone production (ketoconazole) [18,21–23]. Finasteride has also been tested in one study (35 patients with sickle-cell disease) and shown to be effective in reducing the number of recurrent episodes considerably [24].

It has been reported that libido and frequency of nocturnal erections can be

suppressed by reducing testosterone levels to 10% of normal physiological levels [25]. There is minimal information regarding the efficacy and safety of most of these agents and none has been investigated using controlled study designs. Hormonal agents, specifically GnRH agonists, appear to be effective and, while they reduce libido, most patients are still able to engage in sexual activity [2].

It may well be the case that if stuttering priapism is successfully managed for a period of time (weeks to months), the condition will not recur and the patient can be withdrawn from treatment eventually. The duration of treatment with hormonal agents varies from weeks to years depending on the type of agent. It has been reported that a 2-week treatment course with stilbestrol (5 mg daily) has been effective in treating stuttering priapism [18,26]. Other agents such as GnRH analogues (leuprolide acetate 7.5 mg monthly) and antiandrogens (bicultamide 50 mg daily, flutamide 125–250 mg three times a day) have been successfully tried for longer periods (2 months to 2 years) [21,22,27,28]. All these agents have been shown to be effective only in small case series, in both idiopathic stuttering priapism and patients with sickle-cell disease who have this condition.

Hormonal agents have a contraceptive effect and interfere with normal sexual maturation. In addition, they may interfere with the timing of epiphyseal plate closure. These agents are therefore contraindicated in children who have not completed their growth and sexual maturation or in men trying to conceive [2].

INCREASING CORPUS CAVERNOSUM SMOOTH MUSCLE TONE

Pseudoephedrine (most commonly used as an oral decongestant) is often used as a first-line treatment in stuttering priapism. The effect of pseudoephedrine on corporal smooth muscle has yet to be investigated, although it is likely that smooth muscle contraction is mediated via α-adrenergic receptors [29].

Digoxin inhibits the smooth muscle membrane Na-K-ATPase which prevents the efflux of Ca^{2+} from the smooth muscle cell and increases the intracellular Ca^{2+}. Smooth muscle contraction occurs, which ultimately leads to penile detumescence. A small *in vivo* double-blind placebo-controlled study using six human subjects showed no significant effect of digoxin on testosterone, oestrogen and luteinizing hormone plasma levels but subjects reported a decrease in sexual desire and reduction in penile rigidity with a dose of 0.25–0.5 mg/day. The same study group obtained the same results in a multicentre study but with fewer side effects [30,31].

Terbutaline is a β2-adrenergic agonist. The underlying mechanism of action of terbutaline is unclear, although it has been postulated that the drug has α-adrenergic effects and may also alter permeability of the cavernosal tissue which ultimately leads to an increase in plasma flow within the sinusoids [33]. The efficacy of terbutaline in treating priapism has been investigated in two randomized controlled trials and found to be successful in 36–42% of cases [32,33].

Etilefrine (α-agonist) has been successfully used in patients with sickle-cell disease, both in oral and intracavernosal forms. Oral administration (50–100 mg daily) is used for preventing stuttering priapism, while the intracavernosal form is used in acute episodes [34].

Phosphodiesetrase type 5 (PDE-5) inhibitors in low doses have been successfully tried in a series of men with sickle-cell-associated and idiopathic stuttering priapism. More intriguing, all of these cases were unresponsive to other currently available management options. Thus a low dose PDE-5 inhibitor therapy (sildenafil 25 mg daily then tadalafil 5 mg three times weekly) has become a paradoxical treatment for priapism (i.e. medication that is normally used for erectile dysfunction) [35,36].

Gabapentin is a drug with anticonvulsant, antinociceptive and anxiolytic properties, widely used as an analgesic and antiepileptic agent with an unknown mechanism of action. Although the molecular targets of gabapentin remain unknown, the inhibition of Ca^{2+} efflux from muscle cells in the corpora, with a consequent inhibition of smooth muscle relaxation, may explain the effectiveness of gabapentin in the management of refractory priapism. Another study showed that gabapentin treatment in rats significantly reduced testosterone and FSH levels, which might be another mechanism of gabapentin in treating priapism [37].

Several studies have shown that early management at home with self-intracavernosal injection of sympathomimetics by the patient can be an effective strategy to avoid hospitalization for stuttering priapism. This method of management is not preferred over systemic therapies because priapism in such cases is being treated rather than prevented. Nonetheless it could be an option for patients who cannot be treated with hormonal therapy. Patients should be counselled regarding the injection site, dose and potential side effects [27,38–40].

Several studies report the successful treatment of stuttering priapism with hydralazine, hydroxyurea, baclofen and procyclidine [29,40–43].

For priapism related to sickle-cell disease, conventionally offered medical therapies including analgesia, hydration, oxygenation, alkalization and even transfusion may be performed, but these interventions should not delay intracavernous treatment if prolonged periods of ischaemia have occurred [16].

SURGICAL TREATMENT

Surgical intervention is indicated for stuttering priapism if conservative treatments fail. The aim is to create a shunt between the corpus cavernosum and glans penis, corpus spongiosum or a vein so that the obstructed veno-occlusive mechanism is bypassed. This can be achieved by either proximal or distal shunts.

The creation of a distal shunt involves a transglanular to corpus cavernosal scalpel or needle-core biopsy, using the Winter [44] or Ebbehoj [45] techniques. This is the first reasonable approach for refractory cases. A unilateral shunt is often effective. Bilateral shunts are used only if necessary (usually apparent after 10 min). The El-Ghorab procedure (excision of tunica albuginea tip) is a more aggressive open surgical cavernosal shunt and is indicated if the Winter shunt fails [2].

The Quackels [46] or Sacher [47] proximal shunts are cavernosal-spongiosum shunts

(unilateral or bilateral). These involve creating a window between the respective corporal bodies. A Grayhack shunt is a cavernosal-saphenous vein shunt (rarely necessary or indicated) [48]. Such shunts are rarely effective if a more distal shunt has already failed because thrombosis of the corpora is usually already present [2].

The success rate for various surgical shunt procedures is around 75% [2]. In most cases, shunts will close with time. However, long-term patency of the shunt may lead to erectile dysfunction [49].

Some investigators have also advocated immediate penile prosthesis surgery for particularly prolonged ischaemic priapism presentations when complete erectile dysfunction is considered to be an inevitable result [50,51].

CONCLUSION

Stuttering priapism is an underinvestigated and poorly managed urological problem. Epidemiological data is severely lacking and despite the recent progress in understanding the pathophysiology of stuttering priapism, no universal therapeutic strategy for this rare but potentially very serious condition has been produced to this date. The majority of recommended treatments have been tested in small case series so their true efficacy is unknown. Because of the rarity of stuttering priapism, multicentre studies are required in order to recruit sufficient number of patients for conducting randomized placebo-controlled trials. There is also a need to evaluate the long-term effects of currently available treatments on patients' sexual performance and health-related quality of life.

CONFLICT OF INTEREST

None declared.

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Abbreviation: PDE-5, phosphodiesterase 5.