Priapism

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Key Points:

1. Ischemic priapism is an emergency that requires prompt management.
2. History and physical examination along with corporal blood gas and/or color Doppler ultrasound can distinguish ischemic and non-ischemic priapism (Table 2).
3. Corporal aspiration/irrigation and injection of a sympathomimetic agent should be the initial primary management for acute ischemic priapism (Figure 1).
4. With failure of non-surgical management or prolonged ischemic priapism (>36 hours), a surgical shunt is warranted (Table 3).
5. Non-ischemic priapism is not a medical emergency and conservative, non-invasive treatment is recommended as most cases resolve spontaneously.

Key words

priapism, ischemic priapism, low flow priapism, non-ischemic priapism, high flow priapism, recurrent priapism, stuttering priapism, intermittent priapism, prolonged erection

1. Introduction

Priapism is the persistence of penile erection in the absence of sexual arousal.[1] The name is derived from the Greco-Roman god Priapus, a fertility deity who was always depicted with a continuously erect phallus.[2] Priapism was first recognized in the Western medical literature in 1616 AD.[3] Despite this long history, it remains a poorly understood and researched clinical problem.

Priapism is divided into two variants, ischemic priapism (IP, formerly known as low-flow or veno-occlusive priapism) and non-ischemic (NIP, formerly known as high-flow or arterial priapism).[4] Distinguishing **ischemic** from non-ischemic priapism is critical, as management differs markedly. “Stuttering” priapism is a term frequently used to describe recurrent episodes of IP and is now often referred to as recurrent ischemic priapism (RIP). RIP classically is associated with Sickle Cell Disease (SCD) or trait; young boys, adolescents, and adult men may experience morning erections that persist for several hours. In some longitudinal series of SCD-related priapism the duration and frequency of ‘stuttering episodes’ is associated with episodes of ischemic priapism requiring medical

or surgical interventions. These episodes may be self-limited but can be very painful and may over time lead to corporal tissue damage. Approximately 25% of men with IP have a recurrence within 1 year.[5]

2. Risk Factors and Pathophysiology of Priapism

Normal erection is the result of increased cavernous arterial inflow and a reduction in penile outflow by cavernous venous occlusion. IP results from failure of the veno-occlusive mechanism (valve mechanism) of penile erection to reverse following orgasm. Persistent venous occlusion has the effect of trapping blood in the cavernous sinusoids. While restriction of outflow is desirable during normal penile erection, failure to evacuate blood (and allow the introduction of oxygenated blood) leads to time dependent changes in corporal environment characterized by decreasing oxygenation, hypercarbia and acidosis.

Control of the corporal veno-occulsive mechanism is physiologically dependent upon the ability of cavernosal smooth muscle contraction. With progressive ischemia the cavernous smooth muscle is paralyzed and cannot contract. Recent evidence has implicated dysregulation of the nitrergic and related molecular pathways in the pathogenesis of IP.[6]

IP is conceptualized as a compartment syndrome of the penis. The ischemia and acidosis that occurs with stagnation of blood within the corporal bodies leads to tissue dysfunction and ultimately tissue death (very much like a myocardial infarction). Microscopic structural changes occur as early as 8 hours. Corporal tissue biopsies obtained after 12 hours of IP show evidence of interstitial edema. At 24 hours post-onset biopsies reveal endothelial cell destruction and thrombosis. Biopsies at 48 hours or more typically show smooth muscle necrosis and fibroblast proliferation.[7,8,9]

Conditions associated with IP are listed in **Table 1**.

Table 1: Causes of Priapism	
Idiopathic	
Hematologic	• Sickle Cell Disease or Trait • Thalassemia • Multiple Myeloma • Hyperalimentation/Total Parenteral Nutrition • Glucose 6-Phosphate Dehydrogenase Deficiency • Hemodialysis • Leukemia
Drugs	• Erectogenic Prescription Drugs • Herbal Therapies • Antihypertensives • Neuroleptics • Antidepressants • Anticoagulants • Methylphenidate/Atomoxetine[10]
Neoplastic/Malignant	• Penis • Urethra • Bladder • Prostate • Rectal • Metastatic Cancer to pelvis
Neurologic	• Syphilis • Spinal Cord Injury • Cauda Equina Syndrome • Spinal Cord Stenosis/Disc Herniation
Trauma	• Pelvic Trauma • Perineal Trauma • Penile Trauma (including iatrogenic)

Adapted from Huang et al 2009

Blood dyscrasias are an important cause of priapism. SCD, the most common inherited multi-system blood disorder, is a common cause of IP. SCD can impact individuals of any ethnic group, but disproportionately affects individuals of African descent. Nearly one out of 12 African-Americans carry a sickle cell gene. About 40% of all men with SCD experience at least one episode of IP in their lifetime. IP in this population was long thought to result from trapping of deformed red blood cells in the corporal bodies. These deformed blood cells were thought to lead to “sludging” of blood. However, further research has elucidated that reduced nitric oxide (NO)/cGMP bioavailability in penile tissue secondary to decreased eNOS is the principal cause for IP in SCD. [11,12] Patients with SCD and priapism tend to have exhibit greater degrees of hemolysis compared to men with SCD without priapism.[13] Dysregulation of PDE5 expression and the calcium sensitizing RhoaA/Rho Kinase pathway under hypoxic conditions [14,15,16] may further exacerbate IP by preventing breakdown of cGMP and preventing arterial constriction. Disorders of oxygenation during sleep may also play a role in SCD-associated priapism. [17] The mechanism of priapism associated with other hematologic conditions is poorly understood but likely related to increased blood viscosity and hypercoagulability. With the recent coronavirus disease 2019 (COVID-19) pandemic, several case reports have suggested an association between priapism and COVID-19, given the unifying mechanism of thromboembolic disease.[18] Although this association may likely be confounded by other risk factors, further research is needed to understand the risk of priapism with COVID-19.

The most common cause of priapism in the United States is the use of erectogenic drugs, particularly injectable agents and urethral suppositories. [19,20] Priapism has been reported after use of PDE5i but is rare except in cases where other predisposing factors (illicit drugs, blood dyscrasia, etc) are also present.[21] Among users of injectable erectogenic drugs, episodes of IP were higher when prescribed by non-urologic medical providers. [22] IP has also been linked to use of psychotropic drugs (e.g. trazodone, clozapine, ziprasidone, risperidone, quetiapine, methylphenidate, carbamazepine),[23,24,25,26,27,28] recreational drugs (cocaine),[29] ephedrine,[29] and certain herbal medications.[30,31,] Neurologic causes of priapism may be related to the disruption of the autonomic nervous system and penile vascular tone.[32]

Less common causes of IP include primary and metastatic pelvic/penile malignancies,[33,34,35] alpha blockers,[36] black widow spider venom,[37] low molecular weight heparin,[38] propofol,[3352] disseminated intravascular coagulation,[39] tertiary syphillis,[40] prostate abscess,[41] spinal cord lesions,[42,43] and possibly lumbar sympathetic nerve blockade.[44] NIP is the result of unregulated inflow into the corporal bodies, usually the result of a cavernous arteriolar-sinusoidal fistula resulting from trauma to either the penis or perineum. NIP has been reported in neonates as a result of birth canal trauma to the perineum but has also been seen in the absence of identifiable perineal or penile trauma.[45] NIP is NOT a true emergency. In NIP blood within the corporal bodies is oxygenated and as such tissue damage from ischemia does not occur. However, urgent evaluation should still proceed to ensure the prolonged erection is not ischemic in nature. The natural history of NIP is persistence of partial erection for weeks to months. During this time patients are likely to retain the ability to obtain sexually stimulated erections.

3. Evaluation of Priapism

Table 2. Corporal Blood Gas and Doppler Values Based on Priapism Etiology

	pH	pO2 (mmHg)	pCO2 (mmHg)	Cavernosal Artery Blood Flow Velocity
Ischemic Priapism	<7.25	<30	>60	Zero or Minimal
Non-Ischemic Priapism	7.4	>90	<40	High or Normal
Normal Arterial Blood	7.35	40	50	Normal

The most important step in management of the man who presents with prolonged penile erection is characterizing the priapism episode as either IP or NIP, as prognosis and management differ markedly. [1]

Time is of the essence in management of IP. Men with IP lasting 12 hours or less are very likely to maintain the capacity for normal erectile function after resolution.[46] Men with medication induced priapism of 12-24 hours may experience preservation of erectile function in about 92% of cases[47] although the odds of preservation may be lower in priapism of 12-24 hours not related to medical therapy for erectile dysfunction (ED) or in cases of recurrent priapism. IP lasting longer than 48 hours has a very high rate of long term ED. [46] Even if hope for recovery of erections is low, management to reverse the pathologic erection will relieve pain and shorten the course of the disease process. Un-reversed ischemic priapism may take weeks to resolve.

A focused history and physical exam are the initial steps to differentiating the etiology of priapism. Important components of the history include the duration of the erection, penile pain associated with the erection, and prior history of priapism. Recent history of trauma to the genitals, perineum, or pelvic region should be documented. The patient should also be asked about use of erectogenic drugs, recreational drugs, and prescription drugs known to be associated with IP. Penile injections for the management of ED are more commonly associated with prolonged erection (1 – 4 hours) but may result in true IP, especially when patients first begin home dosing, or supplement penile injectables with oral phosphodiesterase type 5 inhibitors or recreation drugs like cocaine. Selective inhibitors of phosophodiesterase type 5 (PDE5i) are rarely associated with IP. PDE5i-induced priapism accounts for less than 3% of all drug-induced priapisms. [48] However, men with other risk factors for IP appear to be at slightly increased risk of IP when using PDE5i.[49] Any history of blood dyscrasia or advanced pelvic malignancy should be elicited.

Non-ischemic priapism is generally associated with penile or perineal trauma and is usually painless and associated with less than a fully rigid erection. There are case reports where NIP has been diagnosed by color Doppler examination in men who have been treated for IP with penile shunts. IP is thought to convert to NIP in some cases by iatrogenic laceration of the cavernosal artery during attempts at corporal aspiration or surgical shunting.

In all cases of suspected IP presenting to Emergency Departments, a urine drug screen should be ordered to document illicit drug use and a CBC ordered to detect occult hematologic malignancy (leukemia, multiple myeloma). In patients where concern exists about SCD or thalassemia, a serum electrophoresis is warranted. If Sickle Cell prep is positive an outpatient hematology consultation should be obtained.

Emergency diagnostics to define the type of priapism include: (i) corporal blood gas analysis and (ii) color Doppler ultrasound of the penis (CDU).[50] A summary of corporal blood gas analysis and CDU findings in priapism can be found in **Table 2**. Dark, acidotic, hypoxic and hypercapnic blood is a classic finding in IP. In NIP, the corporal blood gas is typically consistent with arterial blood. CDU examinations should include scanning the pendulous shaft as well as the perineum in boys and men

who give history of straddle injury. The injury may be remote to the presentation of NIP.

Color Doppler ultrasound (CDU) is a real time test to ascertain corporal blood flow before or after interventions (aspiration, corporal irrigation, corporal shunting).[51] Peak Systolic Velocity in the corporal bodies tends to be very high (> 50 cm/sec) in NIP. Doppler examination of a normal erection shows peak systolic velocities > 35 cm/sec and absence or reversal of diastolic flows. These wave forms may be evident in IP at the base of the penile shaft or in the perineum, but as time progresses in IP no flow is noted in the distal cavernous arteries.[52] CDU may reveal a fistula as a cause of NIP.[50] Magnetic Resonance Imaging (MRI) of the penis has also been suggested as a means to evaluate corporal tissue integrity (document thrombosis) and blood flow.[53] For patients with suspected malignant priapism, MRI is useful for evaluating for a penile mass.[54] CDU may also be useful for monitoring response to treatment of IP.[55]

4. Epidemiology of Ischemic Priapism

The national incidence of priapism is 5.34 per 100,000 males per year.[56] Interesting, the number of emergency department visits increase during the summer versus winter months. Approximately 13% of ER visits for priapism lead to hospital admission for further management. The estimated annual cost for priapism care in the U.S. was estimated at over $123 million dollars, with the majority of that amount driven by those patients who were admitted for in-patient care.[57]

5. Treatment of Ischemic Priapism

It is important to have frank discussion with the patient. The natural history of untreated IP is corporal fibrosis and ED. Depending on the duration of IP, interventions may be successful in reversing the painful erection, but not in assuring future potency. Careful documentation of this discussion is essential.

There are three goals in the management of ischemic priapism: detumescence, preservation of potency, and prevention of future occurrences.

5.1 Initial Conservative Management

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5.2 Corporal Aspiration and Irrigation

Corporal irrigation/aspiration is accomplished by placement of a large bore needle into one of both corpora cavernosa.[1] The approach is facilitated by first performing a dorsal nerve block in the awake patient (lidocaine or a mixture of lidocaine/bupivacaine). Needle placement in the awake patient is

typically lateral (3 or 9 o'clock) at the base of the penile shaft. In the anesthetized patient large bore needle/s (16 or 14 gauge) can be place transglanular (needle placed though the glans). [58] A butterfly needle or angiocatheter are commonly used (19 or 18 gauge) in awake patients. For patient comfort, the needle should be placed and left *in situ* rather than placed and withdrawn multiple times, for serial aspiration or aspiration/irrigation. Connections between the corpora cavernosa should facilitate removal of old blood from the contralateral corporal body. Old blood may be aspirated via the needle using a 30-60 cc Luer Lock syringe; after aspiration, irrigation with sterile saline (chilled saline if available) may be of utility in flushing out clotted blood.

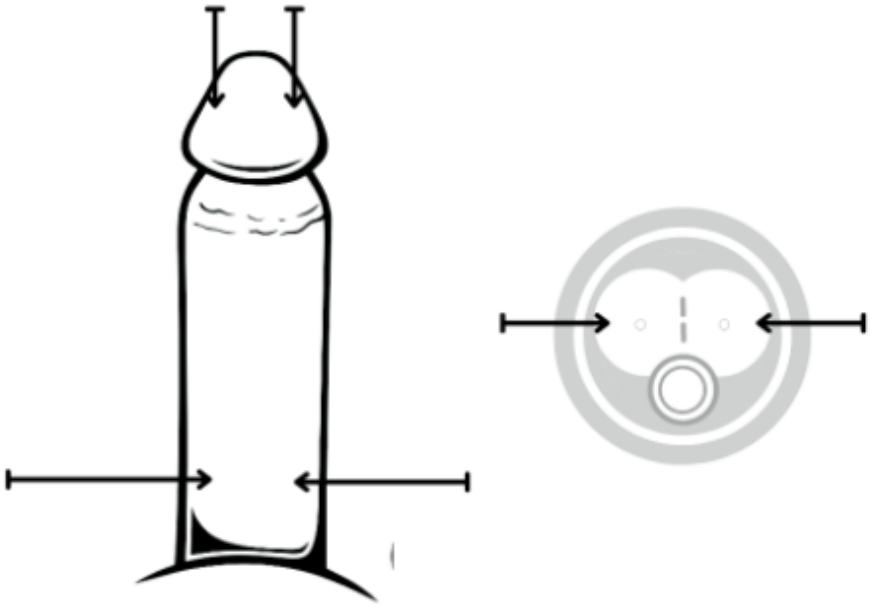

Figure 1: Needle placement for corporal aspiration and irrigation may be performed laterally at the base of the penis at 3 or 9 o'clock or transglanular. (Courtesy of Darshan Patel)

5.3 Alpha Adrenergic Agent Injection

The first step in pharmacologic reversal of a prolonged erection or reversal of IP is aspiration of de-oxygenated, hypercarbic, acidotic blood. With priapism secondary to the use of vasoactive substances (such as alprostadil), intracavernosal injection (not subcutaneous) of a sympathomimetic drug can be attempted as a first step with a small bore needle (27 gauge) prior to formal aspiration/irrigation. Keeping the base of the penis compressed between the thumb and index finger of the surgeon's non-dominant hand to prevent refilling of the penile shaft, the penis should be aspirated until it is flaccid. At this point the alpha-adrenergic can be administered. The penis will refill with fresh blood. Serial aspiration before adrenergic injection may be needed to 're-activate' corporal smooth muscle.

Phenylephrine is the agent of choice in IP due to its pure alpha agonist profile.[59] A phenylephrine dose between 100-500 mcg/ml in saline in 1 ml aliquots should be administered via a hollow-bore needle placed into the corpora cavernosa for purposes of corporal aspiration/irrigation.[1] The patient should be monitored using an automated sphygmomanometer. The side-effects of alpha-adrenergics are hypertension and reflex bradycardia. For men with heart disease and children, cardiac monitoring should be considered and lower doses of phenylephrine should be used. The penis should be examined and palpated to assess for detumescence. The half-life of intravenous phenylephrine is approximately 5 minutes. If the penis remains fully erect after 3 to 5 minutes, repeat aspiration and injection of similar dosage of phenylephrine can be considered.[1]

High dose protocols for priapism have been reported in a small series with doses of phenylephrine up to 12 mg.[60] While generally safe,[60] potential side effects of sympathomimetics include angina and headache.[61] Severe complications including subarachnoid hemorrhage[62] and myocardial infarction with cardigeonic shock.[63] The use of monoamine oxidase inhibitors (MAOI) for depression represent a significant concern for alpha adrenergic agonist use as they are usually monoamines and MAOI leads to prolonged hypertensive activity of these agents. Stroke has been reported in patients receiving up to 2 mg of phenylephrine, and deaths have been reported because of improper concentrations of phenylephrine. Phenylephrine solution is typically available in 10 mg/ml vials therefore it is imperative that the correct dose is prepared for intracavernosal injection. Preparation of the phenylephrine by pharmacy can help reduce the risk of miscalculation and overdose.

5.4 Surgical Shunts

Many cases of IP of 36 hours duration or less can be managed non-surgically.[60] If non-surgical methods fail, surgical intervention is the next step. The duration of priapism, prior history of priapism, and large doses of phenylephrine (>950 mcg) have been identified as predictors for the need of a surgical shunt. [64,65] More recently, machine learning models incorporating multiple clinical factors have been used to predict the probability of a shunt for IP.[66]

A variety of eponymous procedures have been developed for the management of refractory IP. The fundamental goal of all such procedures is to create a shunt between the corpora cavernosa bodies and a venous structure (most commonly the glans or corpus spongiosum) to facilitate drainage. Examples are highlighted in **Table 3** and **Figure 2**. All procedures have purported success but results have not been well replicated in follow-up studies. Generally, success rates for distal shunt procedures range from 66-74% with a 25% rate of long-term ED. Success rates for proximal procedures are reported as high as 76% with a 50% rate of long term ED.[4] Rates of success and complications are difficult to compare across procedures due to confounding variables and general lack of long-term data.[67] Contemporary management of IP refractory to pharmacologic reversal is based on caverno-glanular shunting. Proximal shunts have significantly higher risks related to urethral injury (corpus cavernosum to spongiosum shunting) or pulmonary embolism following saphenous or deep dorsal vein shunting[68] and have largely fallen out of favor in the acute management of IP refractory to pharmacological reversal or distal surgical shunt. There is little

evidence to suggest benefit of a proximal shunt of any kind in a patient with persistent IP after distal shunting per the **AUA guidelines**.[1]

Table 3. Surgical Shunts for Ischemic Priapism

	Description	References
Percutaneous Shunts		
Winters	Biopsy Needle passed in a transglanular fashion into corpora multiple times	Winter. 1978 J Urol 119:227
Ebbehoj	11 blade scalpel passed in a transglanular fashion into distal corpora with 90 degree rotation	Ebbehoj. Scand J Plast Reconstruct Surg 1974;8:241
"T-Shunt"	10 blade scalpel passed in a transglanular fashion into distal corpora with 90 degree rotation +/- corporal tunneling with a dilator	Zacharakis. J Urol 2014 191:164 Brant W. J Urol 2009;181:1699
Distal Shunts		
Al-Ghorab	Glans incision and anastomosis of corporal tips to glanular spongiosum	Lue TF. JSM 2006 3:749
"Corporal Snake"	Scalpel passed in transglanular fashion into the distal corpora with subsequent corporal tunneling through the incision with a dilator	Segal. J Urol 2013 189:1025 Burnett AL. JSM 2009;6:1171
Proximal Shunts		

Sacher (bilateral) or Quackel (unilateral)	Proximal corpora cavernosa to corpus spongiosum	Sacher. J Urol 1972 108:97 Quackels. Acta Urol Belg 1964;32:5
Venous Shunts		
Barry	Dorsal penile vein to corpus cavernosum	Barry J. Urol 1976 Dec;116:754-6
Grayhack	Saphenous vein to corpus cavernosum	GRAYHACK JT, MCCULLOUGH W, O'CONOR VJ Jr, TRIPPEL O. VENOUS BYPASS TO CONTROL PRIAPISM. Invest Urol. 1964;1:509-513.

Two contemporary surgical approaches have described to manage refractory IP without the need for proximal incisions by combining distal shunting with corporal tunneling. The first of these approaches is known as the T shunt (or Lue procedure) and involves passage of a 10 blade scalpel into the distal corporal body by a transglanular approach. The tips of the corporal bodies are usually easy to palpated through the glans in the rigid erection. The scalpel is inserted parallel to, but away from the urethra. The blade pierces skin, glans and underlying tip of corpus cavernosum. Before the blade is withdrawn the cutting edge is rotated 90 degrees away from the urethra and then pulled out (creating a T shaped opening). The clotted blood can be expressed out of the incision. The procedure may be repeated on the opposite corpora to express clotted blood.[9,69] Another novel procedure for refractory IP is known as the “Corporal Snake” (named after the practice of “snaking a drain” to resolve home plumbing issues). The approach is essentially a modification of the transglunular incision procedures but involves passage of a Hegar dilator through the glans incisions into one or both corporal bodies. [9,69] The dilator (usually a Hegar) may be passed down one or both corpora, proximally enough to relieve thrombosed and coagulatred blood from the intracorporeal space. Again the expressing/milking procedure should be performed; success is marked by the evidence of bright oxygenated blood and detumescence. Absorbable suture is placed at the level of the skin incision leaving a shunt between the corpora and glans

More recently, penoscrotal decompression has been described for the treatment of prolonged IP as a salvage or alternative to surgical corporoglanudular approaches. In this approach, a penoscrotal incision is made and exposure of the bilateral corporal bodies is obtained similar to that of placement of a penile prosthesis through a penoscrotal approach. A corporotomy is made and a suction tip is used to gently dilate to the proximal and distal extent of the corpora to evacuate the corpora of dark clotted blood. This is typically repeated on the contralateral side. Once detumescence is achieved the corpora are closed. An advantage of this approach is that the distal tunica albuginea are not violated and a subsequent penile implant may have a lower risk of distal erosion due to the preservation of the tunica albuginea. This approach has been described in a small multi-institutional cohort, with a 100% success rate for bilateral penoscrotal decompression. [70,71]

In a patient with acute IP with persistent erection after a shunting procedure, a corporal blood gas or penile CDU should be performed to help define the penile hemodynamic parameters.[1] This can help guide subsequent surgical interventions such as placement of penile prosthesis. This is especially important for a patient who is being referred for surgical consultation for refractory IP and was initially treated by another clinician at the referring institution.

Risks of surgical shunts for IP include infection/abscess formation, failure to resolve compartment syndrome, and development of NIP from surgical disruption of the cavernous arteries. Penile edema may persist after resolution of compartment syndrome. CDU is the most reliable means to verify restoration of corporal blood flow and should absolutely be performed if there are concerns about persistent IP. Measurement of intracorporal pressure (should be < 40 mm Hg) and/or corporal blood gas may be of some use, however, resolution of acidosis and hypercarbia may take several hours so

immediate results may not reflect appropriate restoration of blood flow. [1]

5.5 Immediate Penile Implant Surgery

In cases of severe priapism of long duration some experts have recommended consideration of immediate placement of penile prosthesis.[72,73] The AUA guidelines recommend consideration of penile prosthesis in patient with IP >36 hours or refractory IP after prior shunt procedures. The advantages of immediate prosthetic placement include decreased risk of penile length loss and avoiding dense corporal fibrosis which may complicate delayed placement of penile prosthetics following resolution of IP. Immediate prosthesis placement should only be contemplated when there is high risk of refractory post-priapism ED (episodes lasting at least 24 hours). Biopsy of the corpora cavernosa and/or pre-procedural T2-weighted gadolinium-enhanced MRI to confirm necrosis of the corporal tissue has been used to justify prosthetic placement. [74]

In the setting of recent distal shunt procedures, immediate placement of penile implants is complicated by higher risks of urethral injury, erosion, and infection[75]. Reasons for surgical re-operation after immediate penile prosthesis include urethral injury (0-20%), device infection (6-7%), and device erosion (0-6%).[76,77] After recent distal shunt for IP, malleable prosthesis is recommended if there is penile edema and bruising. To reduce the risk of distal erosion, especially with distal shunts that weaken the distal corpora (i.e. El-Ghorab, T-shunt), a non-absorbable suture can be placed through the core of a malleable implant to secure it to the corporotomy. [75] In men with severe ED after distal shunt or immediate placement of a malleable implant after distal shunt, the optimal timing for inflatable penile prosthesis if desired is unknown. Anecdotally, we would recommend a period of at least 3 months to allow healing of the distal corpora prior to consideration for inflatable penile prosthesis.

6. Management of Recurrent Ischemic Priapism

RIP should be distinguished from sleep related painful erections that cause frequent night-time awakenings from penile pain due to prolonged but not ischemic nocturnal erections. Management of RIP is challenging due to the broad spectrum of symptoms (number, frequency, length of prolonged erections). Common medications, drugs, and over the counter supplements potentially associated with priapism should be discontinued. [1] Consideration of medical management of RIP after the acute episode of IP is addressed often requires a detailed informed decision between the patient and provider (**Table 3**). This is often best achieved in the outpatient setting.

Patients with SCD or trait make a large proportion of patients presenting with RIP. Hydroxyurea is often recommended for RIP in SCD, based on small case series.[78,79] Chronic exchange transfusions may be beneficial for sickle cell crises as well as RIP, however are associated with certain risks and may be offered in consultation with the patient's hematologist if indicated.[80] The medical therapies described below for idiopathic RIP may also be needed for the management of RIP in men with SCD.

For men with RIP outside the context of SCD (idiopathic RIP), a tiered approach for medical management should be employed, prioritizing agents with demonstrated efficacy and fewer side

effects. As with acute episodes of IP, adrenergic agents have been used for RIP. Reliable patients may be instructed in self-injection of intracavernosal phenylephrine for RIP (typically to end a prolonged morning erection); careful instruction on technique and warnings about the potential for serious vascular events during injection should be provided. [81] Oral adrenergic agonists such as ephredrine, pseudoephridine, etilefrine, terbutaline have been explored for management of RIP however efficacy is limited. [82]

Other medications such as baclofen (muscle relaxant) and gabapentin (analgesic/anti-convulsant) have also been explored for RIP. Baclofen is a γ-aminobutyric acid derivative has been used to prevent reflexogenic erections in men with neurological disorders and muscle spasticity, although the relationship between reflexogenic erections and RIP is not well understood. [83] Gabapentin has an unknown mechanism of action but can cause smooth muscle relaxation. In a small series, it was effective for RIP. [84]

Multiple case series have described the use of low dose, scheduled phosphodiesterase inhibitors-5 (PDE5) (such as sildenafil or tadalafil) for RIP. This paradoxical effect is achieved through tachyphylaxis resulting in upregulation of PDE5 expression in the penis with an end result of decreased vasodilation in corporal circulation. This regimen was first described in a series of four patients of which three had SCD.[85] Scheduled sildenafil alleviated priapism recurrence in these individuals. In another series, five out of seven patients had resolution of priapism recurrences with daily 5 mg tadalafil. In the most contemporary and largest series (n=24), there was a fourfold decrease in the number of RIP-related emergency department visits with regimented PDE5 therapy.[86] Additionally, 22 of the 24 patients reported improvement in their priapism outcomes (number, frequency, duration, etc).[86] PDE5i are generally well tolerated and compared to other medical therapies do not worsen normal sexual function. Patients are usually counseled to take the medication mid-morning while at work.

Hormonal therapies have proven efficacy for RIP, however have an unfavorable side effect profile and are only for adults with RIP refractory to other medical therapies and after informed discussion. These therapies create a hypogonadal state to reduce the androgen mediated component of penile erection. Peripheral androgen ablation is preferred to central androgen ablation. Ketoconazole is the most studied anti-androgren for RIP. Oral ketoconazole (200-400 mg once to three times daily) with corticosteroid supplementation (prednisone 5 mg daily) [87] has been shown to markedly reduce nocturnal erections and reduce recurrent priapism episodes. In the largest study (n=17), using a tampered regimen (200 mg TID ketoconazole for 2 weeks with 5 mg prednisone followed by 200 mg ketoconazole nightly for 6 months), 16 patients had resolution of RIP episodes and all 17 had a reduction in the number of RIP-related emergency department visits. [88] If ketoconazole is selected it is important to include supplementation with corticosteroids due to adrenal suppression. Patients should be regularly monitored for hepatotoxicity while on ketoconazole. Low dose treatment with a 5-alpha-reductase inhibitor medication has been reported as effective in a series of adult men with recurrent priapism. [89,90] The mechanism of action for these medications for RIP is unclear. Other peripheral acting anti-androgens (bicalutamide, flutamide, etc.) have also been described in small

cohorts for RIP, but their efficacy and durability for RIP is unknown. [91,92] Central androgen abalation (such as leuprolide acetate) should seldom be used for refractory RIP and only after informed discussion with the patient about short and long-term side effects. [4]

Table 4: Medical Management of Idiopathic Recurrent Ischemic Priapism

Medication	Class	Side effects	Efficacy (>50% or <50%)
Sildenafil 25 mg daily[86]	Nitrergic	Headaches, nasal congestion, flushing	>50%
Tadalafil 5 mg daily[93]	Nitrergic	Headaches, nasal congestion, flushing	>50%
Pseudoephedrine 60 mg q6hrs[94]	Adrenergic Agonist	Headaches, Palpitations, dizziness	<50%
Ketoconazole 200 mg BID/TID[88]	Peripheral androgen ablation	Fatigue, anorexia, decreased libido, Hepatotoxicity	>50%
Finasteride 1-5 mg daily[89]	Peripheral anti-androgen	decreased libido, Gynecomastia, fatigue	<50%
Dutasteride 0.5 mg daily tapered[95]	Peripheral anti-androgen	decreased libido, Gynecomastia, fatigue	<50%
Intramuscular Leuprolide Acetate[96]	Central Androgen ablation	Decreased libido, hot flashes, gynecomastria, fatigue, osteopenia, loss of muscle mass, worsening lower urinary tract symptoms	>50%

Routine follow-up is advisable for all medical therapies for RIP to assess for treatment efficacy, side effects, and the number of acute episodes of IP. [61] Most patients will likely have recurrent episodes of acute IP that will require emergent management to prevent ischemic damage, while being treated with medical therapies for RIP. Other therapies have been described for the medical management of RIP (i.e. digoxin), however their efficacy is unclear and can have serious side effects.

7. Epidemiology of Non-Ischemic Priapism

It is only since 1960 that NIP has been identified as a pathological entity distinct from IP.[97,98] Etiology of NIP can be classified as traumatic, neurogenic, or post-shunting. The classic history is a man who experiences a straddle injury and later (typically several days to weeks) develops a painless, semi-tumescent and prolonged penile erection. Post-spinal cord injury priapism is most often non-ischemic in nature.[99] Other less common causes of NIP include cavernosal laceration from intracorporal injection for ED, [98] surgical shunts for IP,[100] penile revascularization for vasculogenic ED,[101] penile surgery for Peyronie's Disease,[102] and penile tattooing.[103]

CDU can help confirm the diagnosis by identifying the anatomical site of a sinusoidal fistula which is most often seen as a blush.[104,105] Pelvic and pudendal angiography is no better at identifying a fistula compared to CDU[106] but may be considered since definitive therapy may be offered at the time of diagnosis.

8. Treatment of Non-Ischemic Priapism

Both the AUA and EAU recommend conservative, non-operative, management for the treatment of NIP as spontaneous resolution has been documented in up to 62% of patients.[4,67] Conservative management (perineal compression following acute straddle injury) has a very high success rate in pediatric patients with NIP.[107,108] Conservative measures (such as a cold-pack or compression of the perineum) are used to hasten cavernosal artery vasospasm and fistula closure through clot formation. Should these measures fail, the decision on whether to pursue further treatment is dependent in large part upon patient distress. In some cases, NIP may be of minimal bother to the patient. In the situation where there is no pain and no ED during sexual activity some patients may opt to defer management. A longitudinal study of men with NIP indicated that observation is a viable option for some men. [109] However, approximately 30% of patients with NIP may go on to develop ED. [110]

The standard management for NIP is super-selective angioembolization to occlude the arteriovenous fistula.[111,112,113] This may be accomplished with gelfoam or autologous blood clot; coils should not be initially utilized (although they may be required where temporary occlusive agents fail to resolve the NIP completely) as they are more likely to lead to permanent ED due to migration into the cavernosal artery proper.[114] Success in resolution of NIP can be expected in 73-89% of angioembolization cases; repeat treatment rates are high.[113,115,116,117] Potential side effects of penile angioembolization include ED with an estimated post-procedure incidence of 20-25%.[113,115,116,117] Rare complications include perineal abscess, gluteal ischemia, and penile gangrene.[112,118]

Due to the risk of ED with angioembolization, a number of alternative management strategies have been reported. Androgen blockade has been used in a small series of patients; the proposed mechanism is by reducing corporal blood flow and permitting spontaneous healing of the AV fistula. [119] Surgical ligation of the fistula may also be considered;[120] this requires careful surgical planning and intraoperative Doppler ultrasound to verify location of the fistula.[107] This approach should be considered only by experienced surgeons and after a pseudocapsule amenable to surgical ligation has developed.[107]

9. Clinical Care Pathway

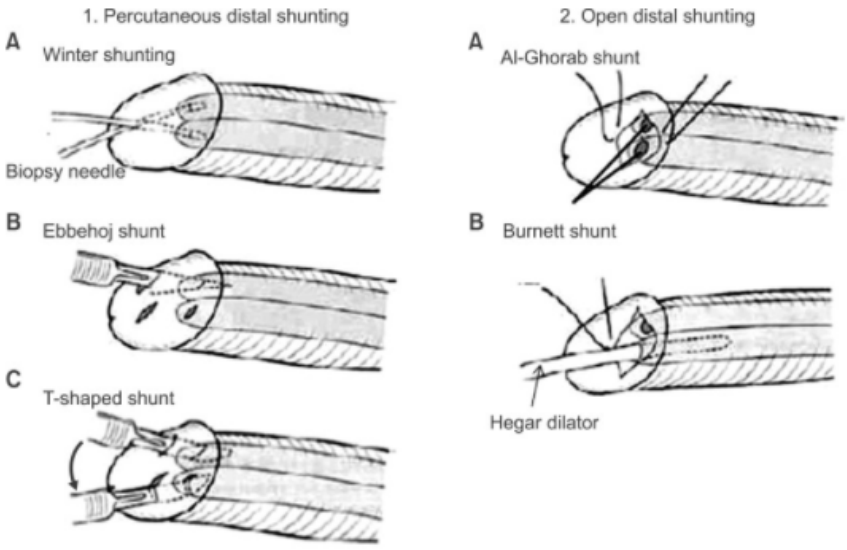

Figure 2: Suggested Clinical Care Pathway

A suggested clinical care pathway is presented in **Figure 2**.

10. Abbreviations

CDU: Color Doppler Ultrasound
cGMP: Cyclic Guanosine Monophosphate
EAU: European Association of Urology
ED: Erectile Dysfunction
IP: Ischemic Priapism
LHRH: Luteinizing Hormone Releasing Hormone
MRI: Magnetic Resonance Imaging
NIP: Non-ischemic Priapism
NO: Nitric Oxide
PDE5: Phosphodiesterase Type 5
PDE5i: Phosphodiesterase Type 5 Inhibitor
RIP: Recurrent Ischemic Priapism

ROS: Reactive Oxygen Species
SCD: Sickle Cell Disease

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