ORIGINAL ARTICLE

Ischemic priapism in South-East Nigeria: Presentation, management challenges, and aftermath issues

FO Ugwumba, HC Ekwedigwe¹, KN Echetabu², AD Okoh², I Nnabugwu, ES Ugwuidu³

Department of Surgery, Urology Unit, Faculty of Medical Sciences, University of Nigeria and University of Nigeria Teaching Hospital, Enugu, Enugu State, ²Department of Surgery, Urology Unit, University of Nigeria Teaching Hospital, Enugu State, ³Department of Surgery, Urology Unit, Federal Teaching Hospital, Abakaliki, Ebonyi State, Nigeria

Abstract

Context: Ischemic priapism is the more common variety of priapism and often presents late. Outcome is largely dependent on the duration of ischemia.

Aims: To determine the etiology, presentation, management, and outcome of ischemic priapism.

Settings and Design: Retrospective analysis of consecutive cases presenting to three hospitals offering specialist urological services in South-East Nigeria from January 2000 to December 2010.

Patients and Methods: Fifteen patients were assessed for clinical data and outcome.

Statistical Analysis Used: The data were analyzed descriptively and inferentially using Statistical Package for Social Sciences (SPSS version 16, SPSS Inc., Chicago IL, USA) with P < 0.05.

Results: Mean age was 30.5 years (standard deviation [SD] =1.63), range: 14–79 years. Onset to presentation interval ranged from 6 h to 28 days. Eight patients (53.3%) had sickle cell disease (SCD). Four patients (26.7%) had unidentified causes. The 8 SCD patients had stuttering priapism on several occasions previously. Six patients (40%) had taken oral herbal medications as treatment prior to presentation. Initial resuscitative measures were intravenous hydration, aspiration, and irrigation with normal saline in 13 patients. Glanulo-cavernous shunt (Al-Ghorab) was performed in all the patients. Detumescence was immediate in 14 and delayed in 1 patient. Three patients had transient recurrence of tumescence, while one had to be reshunted. Erectile dysfunction (ED) occurred in 7 patients (46.7%). Occurrence of ED increased significantly in patients presenting 24 h after onset of symptoms ([*P* = 0.032] Fishers exact test). Mean duration of follow-up was 21.9 weeks (SD = 4.1), range: 3–156 weeks.

Conclusions: Low flow priapism is common in our environment, and approximately half will occur in SCD patients who have had stuttering priapism previously. Timely diagnosis and treatment will reduce the probability of severe ED. In our experience, the Al-Ghorab shunt provides rapid relief. Enlightenment is vital in reducing ischemia time. Emphasis on preventive measures in SCD patients is vital.

Key words: Ischemic priapism, low flow, nonischemic priapism, stuttering priapism

Date of Acceptance: 04-Sep-2015

Address for correspondence:

Dr. FO Ugwumba,

Department of Surgery, UNTH, Enugu State, Nigeria.

E-mail: fredugwumba@gmail.com



This is an open access article distributed under the terms of the Creative Commons Attribution-NonCommercial-ShareAlike 3.0 License, which allows others to remix, tweak, and build upon the work non-commercially, as long as the author is credited and the new creations are licensed under the identical terms.

For reprints contact: reprints@medknow.com

How to cite this article: Ugwumba FO, Ekwedigwe HC, Echetabu KN, Okoh AD, Nnabugwu I, Ugwuidu ES. Ischemic priapism in South-East Nigeria: Presentation, management challenges, and aftermath issues. Niger J Clin Pract 2016;19:207-11.

Introduction

Priapism is defined as a persistent penile erection that continues for >4 h beyond or is unrelated to sexual stimulation. This condition has three varieties; ischemic, nonischemic, and recurrent ischemic (stuttering) priapism. [2]

The ischemic variety is due to venous obstruction. This results in painful hypoxia and is a urological emergency.^[1,2] In the nonischemic type, trauma results in an arteriovenous fistula between the cavernous artery and lacunar spaces, which bypasses the high resistance helicine arteries,^[3] leading to a persistent erection with good oxygenation.^[4,5]

Ischemic priapism, the more common type, has an incidence of between 0.3 and 1.5/100,000 men/year. [6]

In Nigeria, sickle cell disease (SCD) accounts for a large proportion of cases, [7,8] while the use of intracavernosal injection (ICI) of vasoactive substances accounts for many cases in Western climes. [9]

Previous studies show that in developing countries late presentation occasioned by ignorance, patronage of faith healers, and herbalists is the norm.^[7,8] Other possible contributory factors may be physician ignorance and weaknesses in the referral system.^[8] These factors probably act in synergy to worsen outcome.

This study sought to determine the etiology, presentation, management, and treatment outcomes for ischemic priapism in our environment. This would provide a basis for comparison with previous studies to determine any changes in disease profile that may have occurred over time. Findings may also help the development of local management protocols that reflect the peculiar challenges that are encountered locally.

Patients and Methods

Between January 2000 and December 2010, 27 patients managed for priapism at three hospitals in South-East Nigeria were identified through search of admission and theater registers. Of these, 15 case notes with complete records were retrieved and evaluated using a proforma.

Age	J I		Procedures Before	Procedure	Alternative/	Erectile	Degree of erectile	<24 h	>24 h
			presentation		herbal treatment	dysfunction	- -		
14	Ischemic	19 h	IV fluid	Al-Ghorab shunt	No	No	N/A	Yes	Nil
			Aspiration + saline irrigation						
15	Ischemic	3 days	IV fluid	Al-Ghorab shunt	No	No	N/A	Nil	Yes
32	Ischemic	9 days	IV fluid	Al-Ghorab shunt	Yes	Yes	Absent	Nil	Yes
28	Ischemic	8 h	IV fluid	Al-Ghorab shunt	No	Yes	Poor	Yes	Nil
			Aspiration + saline irrigation + adrenaline						
79	Ischemic	28 days	IV fluid	Al-Ghorab	Yes	Yes	Absent	Nil	Yes
			Aspiration + saline irrigation	shunt + bilateral orchidectomy					
22	Ischemic	6 h	IV fluid	Al-Ghorab shunt	No	No	N/A	Yes	Nil
			$Aspiration + saline\ irrigation$						
54	Ischemic	8 days	IV fluid	Al-Ghorab shunt	No	Yes	Absent	Nil	Yes
30	Ischemic	37 h	IV fluid	Al-Ghorab shunt	No	No	N/A	Nil	Yes
			$Aspiration + saline\ irrigation$						
24	Ischemic	20 h	IV fluid	Al-Ghorab shunt	Yes	No	N/A	Yes	Nil
			$Aspiration + saline \ irrigation \\$						
20	Ischemic	11 h	IV fluid	Al-Ghorab shunt	Yes	No	N/A	Yes	Nil
			$Aspiration + saline \ irrigation \\$						
32	Ischemic	40 h	IV fluid	Al-Ghorab shunt	No	Yes	Poor	Nil	Yes
			$Aspiration + saline\ irrigation$						
23	Ischemic	21 h	IV fluid	Al-Ghorab shunt	No	No	N/A	Yes	Nil
			$Aspiration + saline\ irrigation$						
31	Ischemic	22 h	IV fluid	Al-Ghorab shunt	Yes	No	N/A	Yes	Nil
			$Aspiration + saline\ irrigation$						
26	Ischemic	42 h	IV fluid	Al-Ghorab shunt	Yes	Yes	Absent	Nil	Yes
			Aspiration + saline irrigation						
28	Ischemic	72 h	IV fluid	Al-Ghorab shunt	No	Yes	Absent	Nil	Yes
			Aspiration + saline irrigation						

N/A=Not Not applicable; IV=Intravenous

Information retrieved included biodata, clinical features, onset to presentation interval, and type of priapism.

Others were investigations performed, nonoperative care, operative details, prior use of herbs/alternative therapy, complications observed, and duration of follow-up.

Research Ethics Committee approval was obtained. The data were analyzed descriptively and inferentially using Statistical Package for Social Sciences (SPSS version 16, SPSS Inc. Chicago IL, USA) with P < 0.05.

Results

In the study period, 27 patients were seen, but 12 had missing or incomplete records and were excluded leaving 15 evaluable patients. Mean age was 30.5 years (standard deviation [SD] = 1.63), range: 14–79 years. Ten patients (66.7%) were under 30-year-old. Nine patients (60%) were students, 5 (33.3%) were workers, and 1 patient (6.7%) was retired.

All had ischemic priapism presenting with persistent painful penile erections of durations ranging from 6 h to 28 days [Table 1]. Regarding comorbidity, 8 patients (53.3%) had SCD, 1 (6.7%) each had psychiatric disorder and was on treatment with thioridazine, postappendectomy, and

Table 2: Onset - Presentation interval							
Time	Number of patients	Percentage					
<6 h	0	0					
6-12 h	3 patients	20					
12-24 h	4 patients	26.6					
24-48 h	3 patients	20					
3 days	2 patients	13.3					
8 days	1 patient	6.7					
9 days	1 patient	6.7					
28 days	1 patient	6.7					



Figure 1: Priapismic penis: Immediate preoperatively

carcinoma of the prostate. Four patients (26.7%) had unidentified causes. The 8 SCD patients all had stuttering priapism previously.

Six patients (40%) had taken oral herbal medications before presentation. Initial resuscitation consisted of intravenous fluid and aspiration/irrigation with normal saline in 13 patients. Aspiration and irrigation were carried out on the lateral mid-shaft of the penis at 3 o'clock or 9 o'clock using a 19 gauge butterfly needle. Aspiration is then done with a 10 or 20 ml syringe with intermittent injection of normal saline. At conclusion of the procedure, the puncture site is compressed for up to 2 min to reduce hematoma formation. One patient had ICI of dilute adrenaline in addition to aspiration/irrigation.

Glanulo-cavernous shunt (Al-Ghorab) was performed in all the patients. After adequate anesthesia and skin preparation and draping, a transverse incision on the dorsum of the glans penis about 2 cm distal to the corona is carried down to the distal end of the corpus cavernosum. A circular corporotomy is done excising a small disc of tunica albuginea and cavernous tissue deep to it. Dark viscid blood is then expressed until red cavernosal arterial blood is seen and detumescence occurs.

Detumescence was immediate in 14 and delayed in 1 patient. Three patients had recurrence of tumescence that responded to manual expression of blood from the shunt site, while one had shunt revision.

Onset to presentation interval was 6 h to 28 days, with only 20% of patients presenting within 12 h of onset of priapism. Erectile dysfunction (ED), defined in this study as persistently poor or absent erections commencing after the episode of priapism which was self-reported or noted on follow-up questioning, was noted in 7 patients (46.7%). Occurrence of ED increased significantly in patients presenting 24 h after onset of symptoms (P = 0.032) Fishers exact test).



Figure 2: Postshunt photograph: Note detumescence after evacuating sludge via shunt site

Mean duration of follow-up was 21.9 weeks (SD = 4.1), range: 3-156 weeks.

Discussion

All patients presented with ischemic priapism. This is similar to earlier findings, $^{[7,8]}$ this trend was noted to be associated with SCD disease. $^{[10]}$

Fifty-three percent (53.3%) of patients in this study had SCD and was similar to the findings of other workers in sub-Saharan Africa, ranging from 20% to 91%.^[7,8,11] All the SCD patients had a history of stuttering priapism. History also showed poor compliance with SCD clinic visits and routine drugs.

Mean age was 30.5 years (SD = 1.63), which is similar to the previous findings. ^[8,9] This is in sharp contrast to the findings of Earle *et al.*, ^[9] who noted a mean age of 52 years in Australia where the majority of patients developed priapism after ICIs for ED. No patient in our series had ICI as a cause and this probably reflects the low usage of these agents in our environment.

Other etiologies observed were the use of antipsychotic medication (thioridazine) in 1 patient. This had been noted by Earle *et al.*^[9] in Australia, the mechanism being its alpha-adrenergic blocking properties on the corpora cavernosa, which inhibit the sympathetic impulses for detumescence.^[12]

Care should be taken in administering such agents, especially when used in combination with lithium or selective serotonin reuptake inhibitors.^[13] Other known etiologies include chronic myeloid leukemia^[14] and neurological disorders,^[15] but these were not seen in this study.

Carcinoma of the prostate was noted as a cause in one patient and this had been reported by other workers from Africa, Asia, and Europe. [16-18] This usually results secondary to metastatic obstruction within the corpora cavernosa. [18] This patient had bilateral orchidectomy in addition to shunting.

One patient developed postappendectomy priapism and this has been observed by others. [19] Proposed etiology is thought to be due to thrombotic obstruction of venous outflow. [19]

In 4 patients (26.45%), no etiology could be identified.

Onset to presentation interval was noted in our series to be 6 h to 28 days (mean 96.7 h), with only 20% of patients presenting within the 1st 12 h and none presenting earlier than 6 h [Table 2].

Generally, all patients presented late and this has been noted elsewhere. [7,8,11] Reasons adduced for this late presentation include embarrassment, poverty, ignorance of health

personnel on the need for early referral, and patronage of faith healers who often delay presentation to hospital. [7,8]

In this study, 40% of our patients had previously presented to traditional herbalists and alternative practitioners and received medication and only presented to the hospital when these measures failed. Similar pattern of health-seeking behavior has been observed in other developing countries.^[20]

This delay in presentation in low flow priapism is thought to worsen the prognosis and is probably contributory to the high complication rate observed and requires consistent public enlightenment to reduce its occurrence.

Diagnosis was made clinically in all cases, and was based on history of persistent painful penile erection and aspiration of dark viscid blood from the corpora cavernosa. [21]

Current guidelines for the management of ischemic priapism recommend initial corporeal aspiration and injection of sympathomimetics. ^[22] In the cases of ischemic priapism of SCD origin, hydration, oxygenation, and systemic alkalinization can be done. When conservative management fails or where presentation occurs later, the surgical options are to be considered. They include a variety of shunts among the corpus cavernosum to the glans penis, proximal corpus spongiosum, or the saphenous vein. These serve to provide an alternative drainage route for the obstructed venous mechanism. Caverno-glandular shunt of Al-Ghorab was used in all our patients and resulted in immediate detumescence in all the cases [Figures 1 and 2]. This is similar to the findings of others, ^[7,8,23] who noted similar results.

Detumescence was immediate in all patients. Three patients had recurrence of tumescence and all were instructed to squeeze the penis intermittently to manually express blood from the shunt fistula site. This resulted in resolution in 2 cases with 1 reshunted. This phenomenon of rebound tumescence has been observed in other studies. [8,23] Intermittent squeezing of the penis is recommended to ensure shunt patency and prevent recurrence of priapism. [24]

Aspiration and irrigation with normal saline were done in 13 cases. These measures failed to resolve the priapism episode in all attempted cases. A single patient, in addition, received intracavernous injection of adrenaline, which was discontinued later on due to systemic side effects.

Local application of ice pack was used in one patient unsuccessfully. It has been reported that this may worsen the stasis by inducing vasoconstriction in the cavernous tissue and is not useful in ischemic priapism.^[7]

The principal complication seen was ED in 7 patients (46.6%). Occurrence of ED increased significantly

in patients presenting 24 h after onset of symptoms ([P = 0.032] Fishers exact test).

Given that irreversible corporal damage ensues after 6 h of ischemia, [25] it is not surprising that a larger proportion of ED was seen in those presenting later.

With an associated risk of ED of 35–44%, [2,7,11,26] and the grave psychosocial impact if severe, it is essential that early diagnosis and correct treatment has to be carried out.

Five patients (33.3%) had severe ED unresponsive to PDE5 inhibitors. This leaves penile prosthesis insertion as the only remedy to facilitate penetrative sexual intercourse. This has been advocated by other workers in an acute setting before the onset of dense fibrosis that makes insertion difficult and may result in loss of penile length.^[26,27]

Previous studies have recommended an adequate follow-up period as about 58 weeks. [24] Duration of follow-up was 21 weeks (SD = 4.1). We believe that this observed period may be suboptimal in assessing the full range of delayed sequelae of this condition.

Limitations of this study include its retrospective nature, the challenge of medical records storage, and retrieval that led to the exclusion of 8 patients with absent or incomplete charts. Relatively short mean duration of follow-up was also noted.

Conclusion

Low-flow priapism is common in our environment, and approximately half will occur in SCD patients who have had stuttering priapism previously. Irrespective of the predisposing cause, rapid diagnosis and treatment will probably reduce the probability of severe ED. Distal shunting using the Al-Ghorab technique results in rapid detumescence in most cases. Public enlightenment is desired in reducing the prolonged ischemia times seen as a result of delayed presentation. Reemphasizing the need for sustained care for SCD patients with observation of preventive measures would be useful. Further study of this condition of a prospective nature is recommended.

Financial support and sponsorship Nil.

Conflicts of interest

There are no conflicts of interest.

References

 Broderick GA, Kadioglu A, Bivalacqua TJ, Ghanem H, Nehra A, Shamloul R. Priapism: Pathogenesis, epidemiology, and management. J Sex Med 2010;7:476-500.

- Montague DK, Jarow J, Broderick GA, Dmochowski RR, Heaton JP, Lue TF, et al. American Urological Association guideline on the management of priapism. J Urol 2003;170:1318-24.
- Yuan J, Desouza R, Westney OL, Wang R. Insights of priapism mechanism and rationale treatment for recurrent priapism. Asian J Androl 2008;10:88-101.
- Hakim LS, Kulaksizoglu H, Mulligan R, Greenfield A, Goldstein I. Evolving concepts in the diagnosis and treatment of arterial high flow priapism. J Urol 1996;155:541-8.
- Brock G, Breza J, Lue TF, Tanagho EA. High flow priapism: A spectrum of disease. J Urol 1993;150:968-71.
- Muneer A, Minhas S, Freeman A, Kumar P, Ralph DJ. Investigating the effects of high-dose phenylephrine in the management of prolonged ischaemic priapism. | Sex Med 2008;5:2152-9.
- Badmus TA, Adediran IA, Adesunkanmi AR, Katung IA. Priapism in southwestern Nigeria. East Afr Med J 2003;80:518-24.
- 8. Aghaji AE. Priapism in adult Nigerians. BJU Int 2000;85:493-5.
- Earle CM, Stuckey BG, Ching HL, Wisniewski ZS. The incidence and management of priapism in Western Australia: A 16 year audit. Int J Impot Res 2003;15:272-6.
- Adeyoju AB, Olujohungbe AB, Morris J, Yardumian A, Bareford D, Akenova A, et al. Priapism in sickle-cell disease; incidence, risk factors and complications - An international multicentre study. BJU Int 2002;90:898-902.
- Kassogué A, Coulibaly M, Ouattara Z, Diarra A, Tembely A, El Fassi MJ, et al. Clinical and therapeutic aspects of priapism at CHU Gabriel Touré: Study of 36 cases. Pan Afr Med J 2014;17:286.
- 12. Compton MT, Miller AH. Priapism associated with conventional and atypical antipsychotic medications: A review. | Clin Psychiatry 2001;62:362-6.
- Sood S, James W, Bailon MJ. Priapism associated with atypical antipsychotic medications: A review. Int Clin Psychopharmacol 2008;23:9-17.
- I4. Villegas Osorio JF, Corchuelo Maíllo C, Cuevas Palomino A, Medina López RA. Ischaemic priapism as a presentation of chronic myeloid leukaemia. Arch Esp Urol 2014:67:708-11.
- Chen WL, Tsai WC, Tsao YT. Valsalva maneuver-induced priapism: A hidden culprit. J Sex Med 2009;6:1181-4.
- He D, Zeng J, Li X, Wu K, Wu D, He H, et al. Priapism as the initial manifestation of a penile and lower limb cutaneous metastasis of prostate adenocarcinoma with low serum PSA level. J Androl 2012;33:1160-4.
- Sallami S, Ben Rhouma S, Horchani A. Priapism secondary to involvement of corpora cavernosa by locally advanced prostate cancer. Tunis Med 2012;90:411-2.
- Kitley CA, Mosier AD, Keylock J, Nguyen D. Malignant priapism secondary to adenocarcinoma of the prostate. BMJ Case Reports 2010; [doi:10.1136/ bcr.07.2009.2135].
- Sandler G, Soundappan SS, Cass D. Appendicitis and low-flow priapism in children. J Pediatr Surg 2008;43:2091-5.
- Maden C, McKendrick S, Grace R. Alternative medicine use at Vila Central Hospital Vanuatu: A survey of the use of 'custom medicine' in patients and staff. Trop Doct 2003;33:22-4.
- Kadioglu A, Sanli O, Celtik M, Cakan M, Taskapu H, Akman T. Practical management of patients with priapism. EAU EBU Update Ser 2006;4:150-60.
- Salonia A, Eardley I, Giuliano F, Hatzichristou D, Moncada I, Vardi Y, et al. European Association of Urology guidelines on priapism. Eur Urol 2014;65:480-9.
- 23. Lawani J, Aken' Ova YA, Shittu OB. Priapism: An appraisal of surgical treatment. Afr J Med Med Sci 1999;28:21-3.
- Brant WO, Garcia MM, Bella AJ, Chi T, Lue TF. T-shaped shunt and intracavernous tunneling for prolonged ischemic priapism. J Urol 2009;181:1699-705.
- Ralph DJ, Garaffa G, Muneer A, Freeman A, Rees R, Christopher AN, et al. The immediate insertion of a penile prosthesis for acute ischaemic priapism. Eur Urol 2009;56:1033-8.
- Hinman F. Priapism: Report of cases and a clinical study of the literature with reference to its pathogenesis and surgical treatment. Ann Surg 1914;60:689-716.
- Zacharakis E, Garaffa G, Raheem AA, Christopher AN, Muneer A, Ralph DJ.
 Penile prosthesis insertion in patients with refractory ischaemic priapism:
 Early vs delayed implantation. BJU Int 2014;114:576-81.