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Original Article

The Pattern of Penile Gangrene in Sokoto, Nigeria

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ABSTRACT

Objective: To examine the presentation and outcome of management of penile gangrene.
Patients and Methods: The health records of all patients admitted for penile gangrene in Usmanu Danfodiyo University Teaching Hospital, Sokoto, Nigeria between January 1994 and December 2003 were reviewed
Results: The patient group consisted of 16 patients aged between 6 and 62 years (mean: 42.5 years), of whom 7 (44%) presented in an advanced stage of gangrene. The main etiological factors were urethral stricture in 8 (50%) patients, associated with diabetes mellitus in 2 (13%), impacted urethral calculus in 3 (19%), circumcision in 2 (13%), priapism in sickle cell disease in 2 (13%) and trauma in 1 (6%). All patients were initially treated by suprapubic catheterization, broad-spectrum antibiotics, intravenous fluids and wound debridement. Surgical treatment included total penectomy in 3 (19%), phalloplasty in 3 (19%), neomeatoplasty in 5 (31%) and urethroplasty in 5 (31%) patients. Following treatment, 8 (50%) patients had a functional entire penis, while 5 (31%) had a functional penile stump and 3 (19%) no penis. Erectile dysfunction was found in 3 (19%) patients.
Conclusion: Penile gangrene has a unique pattern of causes and presentation. The main cause in our series was urethral stricture. The risk of penile loss is high. Early presentation, aggressive antibiotic therapy and urinary diversion can prevent or minimize penile loss. Some patients with partial penile loss require surgical reconstruction of the urethra and penis.

Keywords : Penile gangrene, Fournier's gangrene, necrotizing fasciitis, phalloplasty, urethroplasty, neomeatoplasty

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INTRODUCTION

Male genital gangrene is commonly called Fournier's gangrene. The infection starts and spreads from a focus in the perineum, scrotum or penis. In 1883, Jean Fournier first described idiopathic, fulminating, life-threatening male genital necrotizing fasciitis which was characterized by a high mortality before the advent of antibiotics[1]. With increasing knowledge of etiology and pathology and the possibility of aggressive antibiotic management, the current picture is that of improved survival[2-6]. We herein report our experience in the management of penile gangrene resulting from various etiological conditions.

PATIENTS AND METHODS

In this retrospective study, we reviewed the records of patients with gangrene affecting the penis treated at the Surgery Department

Table 1: Etiology of penile gangrene in 16 patients.

Etiology	Number of patients	%
Urethral stricture	8	50%
Impacted urethral calculus	3	19%
Circumcision	2	13%
Priapism (sickle-cell disease)	2	13%
Diabetes mellitus	2	13%
Trauma	1	6 %

Table 2: *Clinicopathological patterns of penile gangrene*

Clinicopathological pattern	No. of patients	%
• Urethral stricture, periurethral abscess, spreading gangrene, early presentation, limited tissue loss.	7	44%
• Urethral stricture, spreading gangrene, delayed presentation, extensive tissue loss	4	25%
• Penile trauma, circumcision, ischemic necrosis, spreading wound infection, gangrene, distal loss.	3	19%
• Priapism, ESDA, vascular thrombosis, ischemic necrosis, spreading infection, gangrene.	2	13%

of Usmanu Danfodiyo University Teaching Hospital, Sokoto, Nigeria, between January 1994 and December 2003.

The information obtained included age, demographics, predisposing conditions, etiology, presenting features, duration of symptoms, treatment, surgical procedures, post-operative course, complications, morbidity and mortality.

RESULTS

Sixteen patients aged between 6 and 62 years (mean age 42.5 years) were reviewed. Fifteen patients (94%) were from a low socioeconomic background. Nine (56%) patients presented in the early phase of the acute gangrenous process, while the remaining 7 presented after extensive penile necrosis. All the patients presented with gangrene localized to the penis; the scrotum was not significantly affected. The most common etiology was urethral stricture (Table 1). In 4 (25%) patients there was leakage of urine through two or three necrotic sites in the penile shaft ("watering-can penis"). On presentation 11 (69%) patients gave a history of 3-8 years of poor urine stream and straining to void. Eight (50%) patients had been on some form of treatment for urethral stricture, and 3 (19%) for periurethral abscess. The blood urea level was elevated above 10 mmol/L in 9 (56%) patients. Following catheterization the urea level subsequently fell below 10 mmol/L in all patients. In 2 patients with elevated fasting blood sugar levels (above 20 mmol/L) on admission insulin therapy was started. Ten patients (63%) were screened for HIV and all tested negative.

The 8 (50%) patients with urethral stricture and the 3 (19%) with impacted urethral calculus (they also had untreated urethral strictures) presented in the same manner. They had observed a focal area of pain, swelling and inflammation in the penis earlier on which soon turned black and spread rapidly. On admission, they were very febrile and voided with difficulty. Four of those with urethral strictures suffered from urinary retention. The affected penile skin rapidly broke down as the infection spread. Three (19%) patients with distal urethral strictures had gangrene of the glans. Three (19%) other

Table 3: Penile loss and surgical repair in 16 patients with penile gangrene

Penile loss	No. of Patients	%	Repair Procedure
No penile loss	5	31%	Urethroplasty
Loss of spongiosum and urethra	3	19%	Phalloplasty
Loss of glans penis	5	31%	Neomeatoplasty
Total penile loss	3	19%	Penectomy

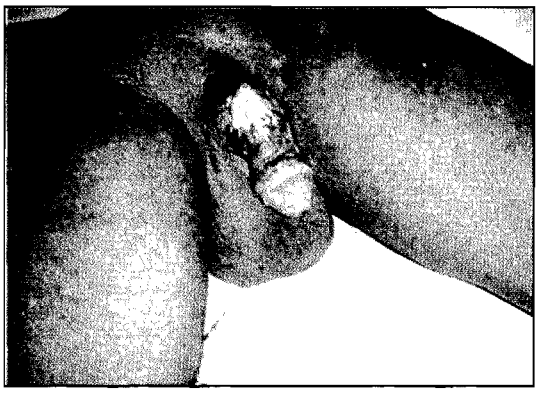

Fig. 1: Early penile gangrene; localised skin loss (Type 1)

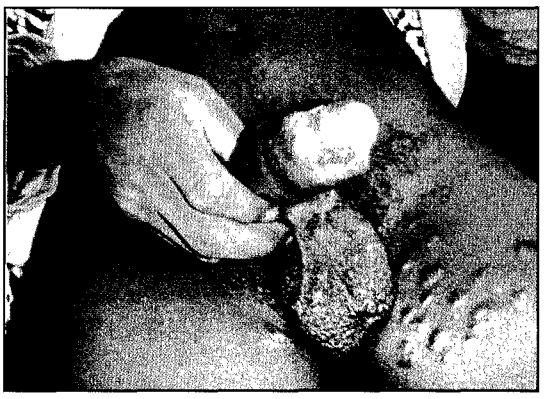

Fig. 2: Distal penile gangrene (Type II), glanular loss

patients with distal bulbar urethral strictures eventually lost the corpus spongiosum and penile urethra.

In 7 (44%) patients who presented early, the gangrenous process was aborted; there was localized penile skin loss in 2 (13%), and little or no tissue loss in 5 (31%) patients.

Infection of the wound due to trauma and circumcision caused distal penile loss in 3 (19%) patients. In 2 (13%) patients there had been excessive excision of the sub-coronal skin and injury to the glans during circumcision. Subsequently, the coronal wound was infected, became gangrenous and the glans was lost. One (6%) patient had fallen from a tree onto a metal fence and had ruptured his penis and scrotum.

Two (13%) patients with sickle-cell disease presented with about ten days of priapism and refused any surgical intervention that did not guarantee potency. Priapism did not subside despite 8 days of conservative treatment and some 12 days later the penis rapidly turned black and wet gangrene set in, resulting in total penile loss.

All patients were initially treated with intravenous fluids, broad spectrum systemic antibiotics (suitable combinations of ampicillin, cloxacillin, gentamycin, ofloxacin, ciprofloxacin and metronidazole), wound debridement, antiseptic baths and suprapubic catheterization. The clinical response to antibiotics was uniformly satisfactory. Microbiological wound cultures revealed a wide range of bacteria including *Escherichia coli, Proteus mirabilis, Staphylococcus aureus,* hemolytic *Streptococcus* and *Pseudomonas*. Most patients had infections with mixed organisms and varying antibiotic sensitivity. The approach to surgical treatment was cautious debridement. Antibiotics administered successfully arrested the gangrenous process within a few days, and the necrotic tissue sloughed away leaving the uninvolved tissues to granulate and heal.

Five patients (31%) were treated for distal penile gangrene; over a period of 5 years they

Fig. 3: Loss of ventral penile corpus spongiosum and urethra (Type III)

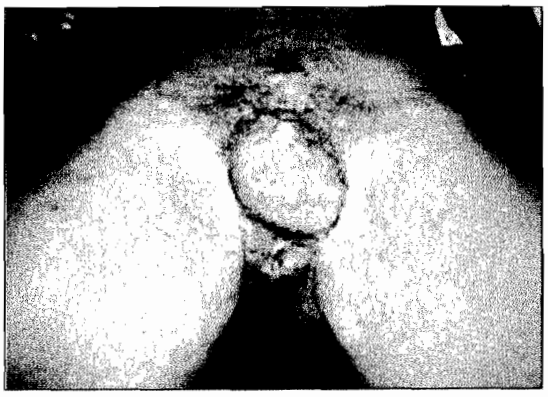

Fig. 4: Total penile gangrene (Type IV)

presented with various degrees of scarring and stenosis of the urethral neomeatus which required neomeatoplasty. In 3 patients (19%) an impacted urethral calculus was identified and extracted during wound debridement. In 5 patients (31%), the acute penile infection completely resolved and the penis was relatively preserved. They were among those who presented early. The clinicopathological patterns demonstrated by these patients are shown in Table 2. Combined retrograde and micturating cystourethrography was used to define the strictures for repair.

Eleven patients (69%) sustained various degrees of penile tissue loss. The pattern of penile loss and the reconstructive surgical procedures are summarised in Table 3. Fig. 1- 4 are illustrative examples of early localized, distal, ventral and total penile gangrene respectively. A summary of the results of treatment is presented in Table 4. Hospitalization ranged from 33 to 65 days, with an average of 48 days. No mortality was encountered in this series.

DISCUSSION

Fournier's gangrene currently encompasses all necrotizing subcutaneous infections of the male external genitalia, both of known and unknown etiology[2-6]. There are three primary types of male genital gangrene: penile gangrene[1-3,10], scrotal gangrene[3,4,11,12] and perineal gangrene[5,6,13,14]. Untreated, any of them can spread beyond the perineum[5,14]. Generally, the lesion is classified according to the extent of spread from the primary site at presentation. There are reports of cases where the infection spread along the facial planes into the anterior abdominal wall and the axilla. When the penis is involved in gangrene the penis has its own focus of infection.

This report focuses on gangrene originating from and localized to the penis. Our patient group consisted of apparently healthy, young and old patients, in contrast to other reports of penile gangrene concentrating on the elderly and debilitated with terminal chronic renal failure and end-stage diabetic atherosclerosis[7-9]. Most of our patients were of low socioeconomic background as has also been observed in many other reports of Fournier's gangrene[2-5].

We were able to identify the etiology and associated predisposing conditions in all the patients. This is also the case with other reports on penile gangrene. However, idiopathic cases still abound in many reports of Fournier's gangrene[1-4]. The most common etiology in our series was urethral stricture, while the most common reported etiologies of Fournier's gangrene are perianal abscess, urethral stricture and scrotal abscess, in descending order of frequency[2-6]. Fournier initially described idiopathic penile gangrene as sometimes associated with idiopathic scrotal gangrene[1]. In the literature, the involvement of the penis in Fournier's

Table 4: Results of treatment of 16 patients with penile gangrene

Result	No. of patients	%
Total penile loss	3	19%
Functional penile stump	5	31%
Functional entire penis	8	50%
Erectile dysfunction	3	19%

Table 5: Penile involvement in Fournier's gangrene from different centers.

Authors	No. of Patients	Penis affected	%
Olsofka et al., USA, 1999[6]	14	5	38.0%
Ekwere, Nigeria, 1999[4]	16	1	6.3%
Corman et al., USA, 1999[2]	23	8	34.8%
Okeke, Nigeria, 2000[3]	26	1	4.0%
Hodonou et al., Benin, 2000[14]	32	10	31.3%
Yang and Wu, Taiwan, 2001[13]	8	0	0
Daali et al., Morocco, 2002[5]	60	30	50.0%

gangrene has been reported to range between 0% and 50% (Table 5). The scrotum is almost invariably involved in Fournier's gangrene. The penis (skin and corpora) and the testes are usually spared[3-6,13]. However in our report and others on penile gangrene, only erythema was noticeable on the contiguous scrotum; there was no gangrene of the scrotum.

In 69% of our patients, the gangrene originated from the urethra (urethral stricture and impacted urethral calculus), while in the remaining patients it originated from the penis (circumcision, priapism, trauma). In other reports the local etiology was found in the vasculature of the penis[7-9]. Corman et al.[2] and Hodonou et al.[14] suggest that when the penis (skin or corpora) is affected in genital gangrene, the etiology of that gangrene may be found in the penis or urethra.

Several studies seem to recognize that genital gangrene begins from a focus of infection, which may be perianal, perineal, scrotal or periurethral[2-6,13]. Periurethral abscess results from extravasation of infected urine, caused by stasis and high-pressure voiding in the presence of urethral obstruction[14]. Bacteria may also enter through a minor abrasion or scratch in the scrotum or perineum or from a perianal abscess[2-5]. The spread of the gangrenous process is promoted by some local and systemic factors[2-6,13]. Systemic conditions such as immunosuppression, diabetes and chronic renal failure which cause atherosclerosis, peripheral angiopathy and calciphylaxis have been implicated[7-9,15].

Two of our patients presented with priapism in sickle cell (HbSS) disease. Priapism is a known complication of HbSS disease, and may even be the initial complaint[16]. Penile gangrene is a rare complication of untreated priapism, but has been reported following aspiration and other invasive procedures for treating priapism[17,18]. Gangrene may be associated with compression dressing, local infection and use of a urethral catheter.

In our series, 50% of patients suffered various degrees of penile loss. The type of penile loss was determined by several factors, including the extent of the spread of

the gangrene, the primary site of the infection (glans or penis), and underlying etiology (systemic or local angiopathy, vasculitis and vascular thrombosis).

- Type I: The gangrenous process affected the penile shaft only, and the patient presented early. With aggressive treatment only a localized portion of penile skin was lost.
- Type II: The glans penis was affected and lost. This may be caused by distal urethral stricture, denuding circumcision and end-stage chronic renal failure[7-9]. Gangrene of the glans penis may also be caused by trauma, diabetes mellitus and, rarely, as a complication of staged hypospadias repair[2,4,18].
- Type III: Extensive gangrene was observed because of late presentation. The ventral compartment (the corpus spongiosum and penile urethra), in some cases also the ventral part of the glans, was lost. This was seen in patients with distal bulbar strictures, some of whom had had multiple urethrocutaneous fistulae in the past.
- Type IV: The entire penis was gangrenous at presentation and was totally lost due to penectomy or autoamputation. We found this type in patients with bulbar urethral strictures, especially those with watering-can scrotum, impacted urethral calculus and priapism. Autoamputation of the penis has been reported in HIV patients[18]. Gangrene following penile self-injection of cocaine has also been reported[19]. Diabetes mellitus and long standing azotemia in our patients might also have contributed to the cause of gangrene.

It has been suggested that the common etiopathology in male external genital gangrene is synergistic bacterial infection taking advantage of the vasculitis and thrombosis of the bulbourethral and scrotal arteries caused by the primary infection[3,6,11]. The bacteria we cultured were mixed flora, similar to those in other reports[1,2-6]. The characteristic spread and rapid progression of the gangrenous process are in part attributed to vascular thrombosis and ischemia of the tissues[6,11,20].

The association of the penis and the scrotum in gangrene (peno-scrotal gangrene) is rare in Nigeria, as illustrated in Table 5. The site of the primary infection often points to the specific etiology[2]. In penile gangrene the etiology and the primary infection are in the penis.

In conclusion, penile gangrene has a unique pattern of causes and presentation. Successful treatment depends on early presentation, aggressive broad-spectrum antibiotics, urinary diversion and judicious supportive measures. The longer the delay in treatment, the higher the risk of partial or total penile loss. Partial penile loss may require surgical reconstruction to restore urethral and penile function.

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