FOURNIER'S GANGRENE: INFECTIVE GANGRENE OF THE GENITALIA

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Abstract

We revisited, a lesion popularly referred to as Fournier's scrotal gangrene, a lesion which dates back to pre-antibiotic era, in order to update ourselves of recent knowledge of its aetiology, features, clinical course and management by comparing our recent data with earlier ones. A case presentation was followed by analyses of recent Lagos University Teaching Hospital experience and practice. Twenty seven patients' records provided adequate data for evaluation of personal data, aetiology, pathogenesis, presentation and management style in our centre. All patients were adults, average of 41 years (range 20-62 years). The clinical course was insidious and was preceded by a prodromal phase of fever, chills and scrotal pain. Infective agents, mainly of gram negative types were isolated in all wounds except in one patient who had been commenced on antibiotics before hospital admission. Precipitating factors consisting of traumatic scrotal skin, infection in neighbouring or contiguous organs, debilitating state of diabetes mellitus, systemic infection and immuno-depression by HIV, were evident in all. The patients' clinical states and wounds improved with systemic metronidazole, and this suggested that anaerobes, particularly bacteroides, played a significant role in its causation. Skin grafting of post scrotal gangrene wound had been abandoned in our centre and was not utilised in any patient. In weeks, scrotal skin far superior in quality to any we had fashioned in the past reformed on simple home treatment of saline scrotal sitz bath. In the light of present day data, we suggest that "Infective Gangrene of the Genitalia" seem a more appropriate term than "Fournier's Gangrene."

Key words: Fournier's gangrene, infective, genitalia.

Introduction

The original descriptions of scrotal gangrene by the Frenchman Fournier was vivid in details[1, 2]. The gangrene was localised to the scrotum. The onset was rapid and its course was a galloping one. It was a disease of adults and was attendant with a large number of deaths. About 20-30% patients died, the early reports documented[3]. Above all, the cause was idiopathic.

Does the scrotal gangrene we encounter today conform with these initial accounts of hurricane-like clinical features, no causative agents and rapid death? Present day literature reports seem to differ and our experience at Lagos University Teaching Hospital in recent times suggest that things have changed for the better.

The grand-round presentation, of this curious disease of the past, was to update ourselves on new facts concerning this disease by sharing current Lagos University Teaching Hospital experience on the causation, predisposing factors, clinical manifestation.

Case Presentation

A-50 year old painter from Imo State but resident in Lagos, was first seen on 28th August, 1997 with complaints of painful scrotal swelling and offensive scrotal discharge, one week prior to presentation. He inadvertently swallowed a fish bone which got stuck within the wall of his anus. Forceful attempts at removal of the said fish bone resulted to an injury to his perineum. He subsequently developed perineal pain and swelling which extended to his scrotum and penis by the third day. The pain was burning in character and continuous. There was an associated swelling of the scrotum which ulcerated by the fifth day, yielding an offensive, brownish discharge. He was not a known diabetic, neither had he been on any immuno-depressant therapy.

He was married with seven children. He did not drink alcohol, nor smoked cigarette. He was not a known hypertensive and there was no family history of a similar illness.

He was found to be in painful distress, anaemic, febrile but anicteric. His genital examination revealed extensive gangrene of a greater proportion of scrotal skin. This had extended to the proximal two-third of his phallus and was discharging offensive, putrid, brownish pus (Fig 1). The testes and spermatic cords were not involved. Rectal examination, revealed a moderately enlarged prostate which felt benign clinically. His inguinal lymph nodes were enlarged and tender on both sides. His other systems were essentially normal.

A clinical diagnosis of Fournier's infective necrotising

gangrene of the genitalia and perineum was made and the following investigations and results (Table 1) were carried out. He was admitted into the ward and the following treatments were given: (a) Antibiotic therapy, consisting of gentamycin, 80mg t.i.d. intramuscularly, third generation cephalosporin, 1gm, bd, intravenously and metronidazole 500mg t.i.d. intravenously, (commenced immediately on admission), (b) intravenous fluids to provide water and calories, (c) suprapubic cystostomy to divert his urine, inserted on day 1, because the penis was sore (Fig 1), (d) extensive debridement of scrotum, penis and perineum under general anaesthesia, performed in theatre on day 2. Minor debridement was repeated on several occasions in the ward when necessary, (e) local wound dressing with hydrogen peroxide and aserbine cream after saline sitz bath.

Lagos University Teaching Hospital Experience

Patients whose records provided adequate data on this lesion were recruited into this study. These were patients seen at Lagos University Teaching Hospital between August 1991 and June 1997. Personal data, details of clinical features including rate of progression of lesion, precipitating factors, scrotal and or other wounds, laboratory tests, including blood, urine and wound cultures, treatment provided, including systemic and local wound care, were extracted from the records. The results, where appropriate, were organised into tables.

TABLE 1: INVESTIGATIONS AND RESULTS

Electrolytes	Renal Function	PCV	Wound Swab Culture	Urine Culture	HIV Screening
Na^+ 126meq/L	BUN 9.1mmol/L	24%	Klebsiella pneumoniae	Pseudomonas aerogenosa	Neg. I, II
K^+ 3.2meq/L	Creatine 139µmol/L		Enterococca cloacae	Providencia rettgeri	
Cl^- 98meq/L					
$HCO\bar{3}$ 20meq/L					

TABLE 2A and B: CLINICAL FEATURES

(A)	SYMPTOMS	NO. OF PATIENTS
1.	Itching and swelling	14
2.	Painful swelling	21
3.	Fever and chills	17
4.	Swelling and "black patch" on scrotum	14
5.	Scrotal pain alone	5

(B)	PHYSICAL SIGNS	NO. OF PATIENTS
1.	Scrotal swelling	27
2.	Pitting oedema of scrotum	27
3.	Crepitus of scrotum	20
4.	Blisters	12
5.	Ulceration	20
6.	Skin desquamation	12
7.	Black patches	13
8.	Overt gangrene	17
9.	Foetid odour	27

TABLE 3: PREDISPOSING/PRECIPITATING CAUSES

		NO. OF PATIENTS
1.	Urethral stricture/extravasation/ periurethral abscesses	5
2.	Post instrumentation/operation on genitourinary tract	3
3.	Ischiorectal abscess	4
4.	Fistula in ano	2
5.	External trauma to genitalia	2
6.	Minor abrasions to scrotum from scratching	1
7.	Infected haemorrhoids	1
8.	Uncontrolled diabetic	4
9.	Septicaemia/general debility	3
10.	HIV infection	4
11.	No cause documented	2
		27

TABLE 4: EXTENT OF GANGRENE

		NO. OF PATIENTS
1.	Limited to scrotum	19
2.	Scrotum + ventral surface of penis	4
3.	Scrotum + perineum	4
4.	Scrotum + perineum + penis	2

TABLE 5: BACTERIAL ISOLATES FROM WOUNDS

		NO. OF PATIENTS
1.	Pseudomonas aeroginosa	11
2.	Klebsiella	11
3.	Coagulase positive staphylococus	5
4.	Proteus vulgaris	5
5.	Escherichia coli	7
6.	Streptococcus faecalis	4

Results

Twenty-seven patients were evaluable. They were aged between 20 and 62 years (mean 41 ± SD6 years). The scrotal lesion was preceded by a prodromal phase of fever, chills, malaise, scrotal discomfort which lasted on average for 5.5 days (range 2-9 days). Their hospital stay varied from 20 to 55 days (average 38 days). The onset of scrotal affliction itemised in Table 2A, was insidious but once established, quickly progressed to various features (Table 2B). These climaxed in loss of scrotal skin and exposure of the underlying testes in most (Fig 1). At presentation scrotal oedema was seen in most and a definite line demacating dead from dying scrotal skin when not present on day one, appeared a few days later. Crepitus of the scrotal skin was evident in most. In many, the scrotal skin sloughed before presentation. The predisposing factors seen in this series are detailed in Table 3. The extent of gangrene are illustrated in Table 4. In all, gangrene involved only the scrotum (Fig. 2) in about 70%, while the scrotum and/or penis and perineum in the rest. No microfilaria was reported in the blood films or scrotal skin biopsies in any patient. The patients included 4 men with

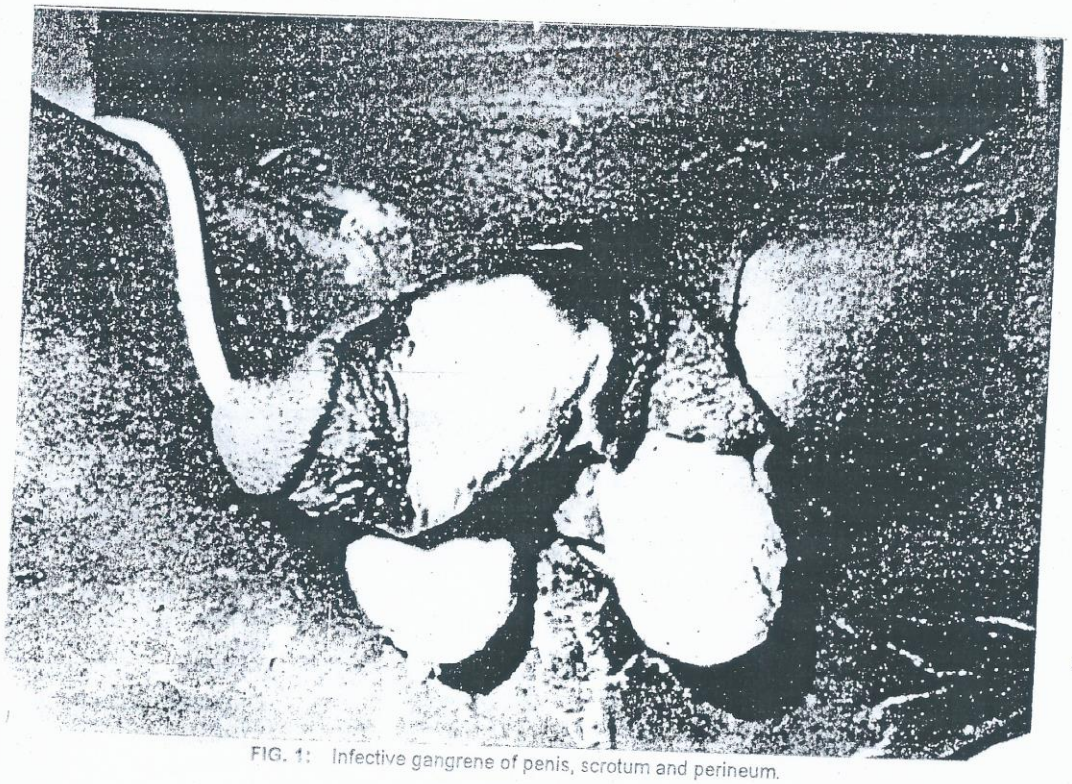

FIG. 1: Infective gangrene of penis, scrotum and perineum.

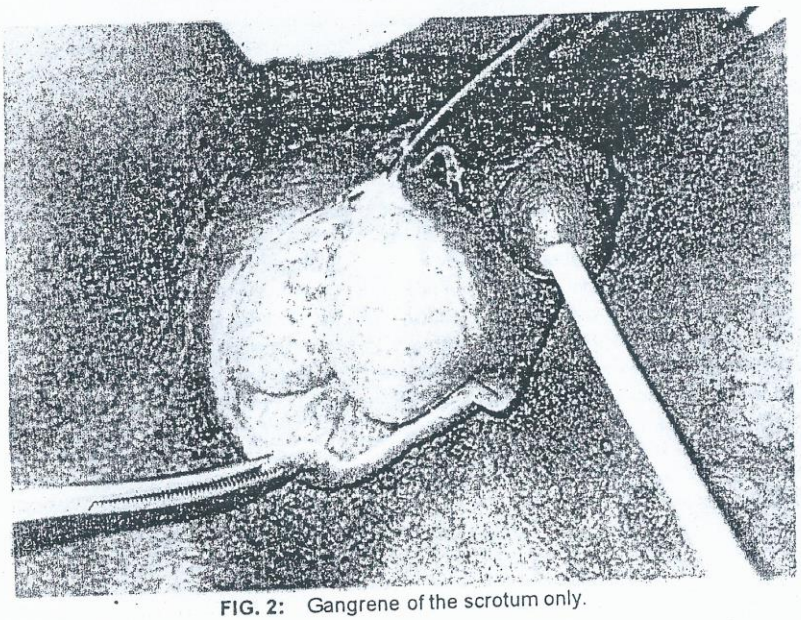

FIG. 2: Gangrene of the scrotum only.

uncontrolled diabetes mellitus and 4 with HIV. Injuries to the scrotum or perineum, both of minor and major nature, were volunteered by 8 patients including the patient in the case report who swallowed a fish-bone that got caught in the anal canal. One patient presented in septic shock with fever, tachycardia, and hypotension. Bacterial isolates from wound swabs are detailed in Table 5. They were sensitive mostly to gentamycin and third generation cephalosporins. No attempt to isolate anaerobes were made. Treatment for anaerobes was based on the reports of others. Blood examination revealed polymorphonuclear leucocytosis in 66%. No patient died in this series.

Discussion

The patients described by Fournier over a century ago, like ours were adults[1, 2]. A few accounts of this lesion in children exist[4, 5]. To our knowledge however, the youngest patient has been an one month old[6]. Elderly men however, are not exempt. Amaku and Ntia[6] described the lesion in a 72 year old[6].

Earlier reports have stressed its idiopathic nature after the manner of Fournier[3, 7]. A lot of theories were soon to follow. The disease was thought to be due to the occlusion of the arterioles to the scrotum (obliterative endarteritis)[8]. This theory was attractive because the disease was initially claimed to affect only the scrotum. Subsequent reports of the involvement of the penis and perineum, organs with different sources of arterial supply, weakened the argument for obliterative endarteritis[9]. Other authors, thought it resulted from antibody/antigen reaction much like the Schwartzman phenomenon[10]. Bacterial isolates from all except one of our patients (Table 5) conform with bacterial pathogens of the colon and those commonly isolated from infected urine (gram negatives). Cultures for anaerobic microbes especially bacteroides, are not routine tests in our centre and were not performed in any patient. However, impressive clinical response that were observed in our study, following systemic metronidazole, a potent anti-bacteroides, and the efficacy of hyperbaric oxygen, reported by others[11, 12], strongly suggest that anaerobes must play a significant role. Indeed, anaerobes mainly bacteroides and clostridia have been isolated by other researchers[13, 14].

Our study suggests that infective agents of either the urine or lower gastrointestinal tract, invade the scrotum and penis, or perineum in susceptible patients and cause subcutaneous gangrene and sloughing of the overlying skin be it that of the scrotum, penis or perineum.

Although scrotal gangrene has been said to occur in an apparently healthy man, our data (Table 3) highlight the aggravating or precipitatory role of devitalised scrotum, or its neighbouring organs and infection in contiguous structures. Four cases in our report with HIV, 4 with uncontrolled diabetes mellitus, and 3 with debilitating ailments stress the contribution of deranged health. It was therefore no surprise that scrotal gangrene was associated with ischiorectal abscess, fistula in ano, and infected haemorrhoids (Table 3) in our series or ruptured appendix[15], distal gastrointestinal sepsis and malignancies[16].

The evolution of gangrene in our patients was less

explosive than earlier studies suggest. The prodromal symptoms, lasting 2-9 days, of fever, scrotal irritation, pain and localised swelling preceeded the development of a black scrotal skin patch, dead and sloughed skin. The extent of gangrene (Table 4) (Fig. 1, 2) in our centre like elsewhere[13] varied and in many of our cases, a clear line, demarcating dead from viable skin was obvious after a few days of onset. In about 70% of our cases (Table 4), gangrene and skin loss was limited to the scrotum, in the rest, penis and or perineum were not spared. In extreme situations, a totally denuded testis was left dangling from its cord (Fig. 1).

We observed that to achieve optimum results, all predisposing factors must be identified and corrected. Four cases of HIV patients in our series, all seen in the last one year, carry obvious lessons and message. We now routinely screen all our patients for HIV.

In our service, we exhibit systemic, third generation cephalosporins and metronidazole from day one. We follow this with urethral catheterization to divert urine in order to assist nursing care. Additional support of intravenous fluids, analgesics, antitetanus regime, and blood transfusion are given as appropriate. We follow this with early, aggressive debridement, excising all dead and dying tissue until healthy, bleeding skin is reached. Local wound treatment with hydrogen peroxide, eusol, desloughing agents and saline sitz bath are applied very generously.

Skin cover for the exposed testes may challenge even the most astute surgeon. In the past, we fashioned like other authors[17, 18] all manners of skin grafts and achieved varying degrees of successes and failures. However, through sheer serendipity, we no longer skin graft post-gangrene scrotal wounds in our practice. Our present approach was informed by a pleasant surprise. One such patient was to have a flap raised from his thigh. His Hb, however, was 7Gm% and the anaesthetist insisted that he be transfused pre-operatively. The patient was a Jehovah's Witness by faith and he refused to be transfused with blood. He subsequently took his discharge against our better judgement. He was therefore consigned to a simple home treatment of scrotal saline sitz bath. When he returned to the surgical outpatient in about 10 weeks, the scrotum had so completely reformed, that it was difficult to distinguish the new scrotum from any original one. In fact, there was a clear median raphe to the new scrotum. The cosmetic result was far superior to the best of our efforts in the past. Ever since, we have ceased to skin graft these scrotal wounds, whatever the extent of skin loss and we have so far not regretted our decision and have had no reasons to revisit this approach. Radical scrotal skin amputation, followed by primary wound closure with good outcome has been reported by Attah[19]. His laudable innovation reduced hospital stay to 10 days. However, when skin loss is great e.g. over 50% (Fig. 1), it is easy to imagine that skin closure may not only be impossible but may put undue tension to the underlying testis and possibly affect its function. In the report from Enugu, a town located in the eastern part of Nigeria and inhabited by the Igbos, Attah[19] considered that the lesion was commoner in them than other races. He treated 13 patients in 4 years. This is comparable to our experience of 27 cases in six years in Lagos, a city of very mixed Nigerian population and this argues against any theory of a disfavoured Igbos. Indeed 9 cases were treated in 9 months in South Africa[14].

Unlike the patients reported from Enugu and Yaounde[13, 19], no micro filaria was not seen in our study. Of course, Attah[19] admitted that filariasis was endemic in his area of practice. Yaounde and Enugu are geographically close and the association between this helminth and gangrene may be pruritus and consequent scratching with fingernails which would readily abrase the skin and create portal for bacterial infection of the scrotum and set-off gangrene.

No patient died in our series. One patient who was admitted in overwhelming gram negative shock quickly rallied upon prompt and adequate antibiotics and metronidazole amongst other supportive therapy. We conclude that the high mortality experienced in the early days was owing to lack of understanding of the role infection played and, of course, the non existence of potent antibiotics and antibacteroides and that this pattern of high death rate should not be seen today.

Question and Answers Session

Question 1: If data presented here strongly support infective process in contrast to what Fournier and early clinicians thought, is it still appropriate to use the term Fournier's Gangrene?

Answer: Many medical giants, like in other fields, who made outstanding contribution to knowledge are immortalised by naming their contributions after them. In that way many others after them may be encouraged to follow in their footstep. To that extent the name Fournier should be retained if only to remind ourselves of our humble beginning. However, there seems to be the need to expand the name to accommodate present day facts.

Attempts to categorise scrotal gangrene into primary where portal to pathogen is obscure and secondary where it is obvious[4] is begging the question. In terms of management, there seems to be no debate that early and effective antibiotics is the pivot of good treatment whatever the categorisation. Without doubt, therefore, infection is behind all cases. It is because of this that we agree that the scope of Fournier's gangrene should be expanded to include infection as its causation. "Infective gangrene of the genitalia" would seem more appropriate today. We there-

fore propose "Fournier's Gangrene: Infective Gangrene of the Genitalia" as a better name to this lesion.

Question 2: One is alarmed by the extent to which the testes are exposed after gangrenous skin sloughs. Is the fertility of these patients compromised?

Answer: Anyone would readily share that anxiety. But if we recall that the testis derives its blood supply direct from the aorta, it is not too surprising that the testes remain red and healthy looking (Fig. 2) in most patients. No change in size or consistency of the testes was noticed in any of our patients. The result of semen examination has been reported from Cameroon[13]. Sperm counts of 20 million/ml or less were recorded in 16 men whose lesions were within 24 weeks. Of these, the sperm counts improved above that level after 24 weeks. It is important to note that in the report, serum albumin was reduced in these men and many of them had polymorphonuclear leucocytosis, features of unhealthy subjects who are not expected to produce semen of normal parameters. That the sperm values rose after 24 weeks suggests that the initial sperm depression may have been the reaction to general body insult and therefore transient.

Question 3: The skin grafting issue is rather unusual. Would you expatiate on your technique? Conservative wound care alone would lead to prolonged hospital stay.

Answer: As we mentioned in our discussion, we earlier[4] in our treatment of the horrifying scrotal skin loss utilised free-skin grafts, rotation flaps from the thigh and so on. It entailed repeated anaesthesia and operating sessions and like anyone familiar with skin grafting, it was subject to varying degrees of failures. Our current practice of allowing nature to re-epithelise the denuded testes, was a child of sheer circumstance and this was detailed in the discussion. It was our hope that the patient would re-think and accept our offer of skin grafting. As it turned out, his scrotal wound healed completely and we were amazed how much better his new scrotum looked. Yes, one of the strong points against conservative scrotal wound management is the long hospital stay involved. In an earlier report of 8 cases of this lesion seen at Lagos University Teaching Hospital in a 10-year period, about 25 years ago, prolonged hospital stay, sometimes as long as 92 days were recorded[6]. The long stay was to allow for complete wound healing whilst the patient was in hospital. In order to achieve early skin cover to the testis and therefore reduce hospital stay, Amaku and Ntia[6] excised one testis. Happily, their patient was 72 years old. Such a risk would be unacceptable in a young man. This emphasises the difficulty of achieving satisfactory surgical skin cover. Prolonged hospital stay, we consider unnecessary, for the treatment while the patient is in hospital, after the acute phase of the disease, is simple local sitz bath. Today, we discharge our patient early to self administered home scrotal saline sitz baths.

Question 4: How early do you recommend debridement of scrotal skin?

Answer: The sooner, the better. We proceed in our centre as soon as theatre space is available, occasionally we start right at admission on the ward and continue in theatre. We must emphasise that the debridement must be very radical, leaving no iota of non-bleeding tissue. This is however, not before we have started the patient off on metronidazole (oral, when possible) 400mg three times daily, gentamycin 80mg (intramuscularly) eight hourly and third generation cephalosporin, 1 gramme twice daily. When the patient is too ill for oral feeds, liberal appropriate intravenous fluids to provide water and calories are used. Antitetanus toxin and toxoid are mandatory.

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