SURGICAL MANAGEMENT OF FOURNIER'S GANGRENE: CLINICAL EXPERIENCE WITH FOUR CASES TREATED AT THE SURGERY CLINIC OF THE CLINICAL HOSPITAL OF TETOVO

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Abstract

Introduction: Fournier's gangrene is a progressive necrotizing fasciitis of the perineal, genital, and perianal region, with high mortality potential if it is not promptly diagnosed and treated. It is characterized by the rapid spread of polymicrobial infection along the fascia, causing vascular thrombosis and necrosis of the soft tissues.
Aim: To present the clinical experience of the Surgery Clinic of the Clinical Hospital of Tetovo in the diagnosis, antibiotic treatment, surgical treatment, and reconstruction of large abdominal wall defects in four patients with Fournier's gangrene.
Materials and methods: This is a retrospective study including four male patients aged 38 to 62 years, treated over several years. Data were collected from operative protocols, clinical records, and laboratory and microbiological analyses.
Results: Two patients on admission presented with skin necrosis in the inguinal region extending to the scrotum, while the other two patients initially presented with subcutaneous purulent collections in the lower abdominal wall, which rapidly evolved into skin necrosis. All patients underwent extensive necrectomy, which left a large abdominal wall defect without skin coverage. Laboratory analyses showed marked leukocytosis, elevated CRP values, and disturbances of several other biochemical parameters. Microbiology identified polymicrobial infection with *MRSA, Escherichia coli, Pseudomonas aeruginosa,* and *Enterococcus spp.* Patients were treated with antibiogram-guided antibiotic therapy, regular dressing changes, VAC therapy, and definitive reconstruction with split-thickness skin autografting. Hospital stay ranged from 10 days to three weeks; all four patients were discharged healed.
Conclusion: Rapid diagnosis, early and aggressive surgical debridement, broad-spectrum antibiotic therapy guided by the antibiogram, negative pressure therapy (VAC), and careful reconstruction of defects with skin autografting are the pillars of success in the management of Fournier's gangrene. Our small but homogeneous experience confirms the value of this algorithm.

Keywords: Fournier's gangrene, necrotizing fasciitis, surgical debridement, MRSA, VAC therapy, skin grafting.

Introduction

Fournier's gangrene (FG) is a specific form of necrotizing fasciitis affecting the skin, subcutaneous tissue, and fascia of the perineal, genital, and perianal region, and very often extending toward the lower abdominal wall, the thighs, or the gluteal area [1–3]. Although the first description of an idiopathic gangrenous process of the genital organs is attributed to Baurienne in 1764, it was the French dermatologist Jean Alfred Fournier who, in 1883, reported five cases of healthy young men with sudden gangrene of the scrotum, thereby making widely known the disease that bears his name [2,3,12]. It is now known that the disease occurs mainly in men over 50 years of age and is associated with a high mortality rate, which in the literature ranges from 20% to over 40%, although certain series report extreme values [1,3,17,19].
The pathogenesis of FG is complex and multifactorial [3,4]. The process is usually initiated by an exogenous or endogenous portal of entry — perianal fistula, perirectal abscesses, urogenital infections, perineal trauma, urological or proctological surgery, urethral catheterization, small scrotal wounds, or injections in the gluteal region [2–4]. Aerobic and anaerobic microorganisms act synergistically: aerobes consume oxygen and create a hypoxic environment, while anaerobes produce heparinase, collagenase, hyaluronidase, and other enzymes that destroy connective tissue [3,6]. The direct consequence is an obliterative endarteritis with thrombosis of the subcutaneous arterioles, which leads to ischemia and

necrosis of the overlying skin. The infection spreads with characteristic rapidity along the fascial planes: Colles' fascia in the perineum, the dartos fascia in the scrotum and penis, and Scarpa's fascia in the lower abdominal wall — a fact that also explains the rapid extension of the process toward the abdomen, as was likewise observed in our patients [3,4,9].

Well-known risk factors include diabetes mellitus (present in 40–60% of cases according to various series), chronic alcohol abuse, obesity, malnutrition, immunosuppressive diseases, malignant neoplasms, end-stage renal disease, HIV infection, parenteral drug use, prolonged corticosteroid therapy, and advanced age [2–4,11,13]. In women, Fournier's gangrene is rarer but more severe, and is often associated with Bartholin's abscesses, infected episiotomies, or gynecological interventions [1,3].

The clinical picture in the early stages can be misleading: local pain disproportionate to objective findings, mild edema, erythema, and a sensation of perineal heaviness [4,5]. Within 24–48 hours, hemorrhagic blisters, subcutaneous crepitation, foul-smelling discharge, dark discoloration of the skin, and the appearance of systemic signs may develop — high temperature, tachycardia, hypotension, and confusion [3,5]. The diagnosis is clinical, but it is supported by laboratory examinations (leukocytosis, neutrophilia, elevated CRP, elevated procalcitonin, hypoalbuminemia, electrolyte disturbances, elevated creatinine, hyperglycemia, coagulopathy, metabolic acidosis) and by imaging methods (ultrasound, computed tomography — CT), which are particularly useful for assessing the extent of the process beneath the skin surface and for identifying gas within the tissues [4,5,11].

Several scoring systems have been proposed for prognostic quantification, the best known being the Fournier's Gangrene Severity Index (FGSI), developed by Laor and colleagues in 1995, which combines the deviations of nine physiological and biochemical parameters (temperature, heart rate, respiratory rate, sodium, potassium, creatinine, hematocrit, leukocytes, and bicarbonate) [7]. An FGSI value above 9 is associated with a high risk of mortality [7,8,13]. Subsequent variants have also been proposed — UFGSI (Uludag) and sFGSI (simplified) — as well as more general instruments such as LRINEC and FGMI [8,14].

Treatment rests on four inseparable pillars: (1) aggressive hemodynamic resuscitation, (2) immediate and extensive debridement of all non-viable tissue — which often requires several repeated interventions, (3) empirical antibiotic therapy with a very broad spectrum that covers gram-positive cocci, gram-negative bacteria, and anaerobes, followed by de-escalation based on the antibiogram, and (4) management of the residual wound, and definitive reconstruction with skin grafting or local and distant flaps [3,4,9–12,15]. Adjuvant treatment with hyperbaric oxygen is debatable but may have value in centers where it is available [11,12]. Fecal diversion with protective colostomy is reserved for cases with active perianal contamination [11,19].

Aim of the study: The aim of this paper is to document the experience of the Surgery Clinic of the Clinical Hospital of Tetovo with four patients treated over several years, focusing on the clinical presentation, the laboratory and microbiological findings, the antibiotic susceptibility profile, the course of treatment, and the final outcomes, and also to discuss these findings in the context of the current literature.

Materials and methods

This is a retrospective, observational, descriptive study including four male patients diagnosed with Fournier's gangrene who were treated at the Surgery Clinic of the Clinical Hospital of Tetovo over several years. The inclusion criteria were a clinical diagnosis confirmed by intraoperative findings (necrosis of the subcutaneous tissue and fascia, characteristic discharge, and tissue planes that slid easily under finger pressure) and the existence of complete medical documentation.

The data collected included: demographic data (age, sex), comorbidities, mode and site of presentation, results of routine laboratory analyses (leukocytes, hemoglobin, hematocrit, platelets, CRP, glycemia, urea, creatinine, transaminases, electrolytes, albumin, coagulogram), antibiogram findings from wound swabs taken at the time of admission, surgical interventions, antibiotic regimens, length of hospital stay, method of wound closure, and final clinical outcome.
Treatment was carried out according to a standardized institutional algorithm: resuscitation with crystalloid solutions, correction of metabolic disturbances, immediate initiation of empirical broad-spectrum antibiotic therapy (usually a combination of a carbapenem or third-generation cephalosporin with an anti-anaerobic agent and a glycopeptide against resistant gram-positive cocci), prompt debridement in the operating theatre with removal of all suspicious tissue, swab collection for culture and antibiogram, daily wound assessments, repeated debridements as needed.

Results

3.1. Demographic characteristics and clinical presentation
All four patients were male, with a mean age of approximately 50 years and a range from 38 to 62 years. The disease presented in two different clinical patterns. The first pattern, observed in two patients, was characterized by extensive skin necrosis already at the time of admission, located in the inguinal region and extending continuously from above downward to the skin of the scrotum. Both of these patients had systemic signs of severe infection: temperature above 38.5°C, tachycardia, severe perineal and inguinal pain, surrounding erythema, a sensation of crepitation on palpation, and fetid discharge from the necrotic area.
The second pattern, observed in the other two patients, had an unusual presentation: the patients were initially referred with purulent collections (subcutaneous abscesses) in the lower abdominal wall, without visible necrosis of the overlying skin at the time of admission. After incision, drainage, and clinical assessment during the first 24–72 hours, the very rapid evolution toward skin necrosis caused the diagnosis to be redefined as Fournier's gangrene with extension to the abdominal wall. This dynamic confirms the spread of the infection along Scarpa's fascia — a well-known anatomical route that connects the perineum with the lower abdominal wall [3,9].
In all four patients, an extensive necrectomy was performed with removal of all non-viable tissue. After the first debridement, a considerable defect of the abdominal wall remained, devoid of skin, involving a large surface area and requiring secondary coverage. In two of the patients, one or two additional debridements were required during the following days because of further areas of necrosis, while in the other two patients a single radical debridement was sufficient.

Table 1. Clinical characteristics of the four patients

Patient	Age	Initial clinical presentation	Number of debridements	Microbiology
P1	38 years	Inguinal skin necrosis extending to the scrotum	1	*MRSA* + *E. coli*
P2	47 years	Inguinal skin necrosis extending to the scrotum	2	*MRSA* + *E. coli*

Patient	Age	Initial clinical presentation	Number of debridements	Microbiology
P3	55 years	Subcutaneous abscesses of the lower abdominal wall with progression to necrosis	2	*MRSA + E. coli + Pseudomonas aeruginosa*
P4	62 years	Subcutaneous abscesses of the lower abdominal wall with progression to necrosis	1	*MRSA + Enterococcus spp. + Pseudomonas aeruginosa*

3.2. Laboratory findings: All four patients presented with marked leukocytosis, with values ranging from approximately 16,000 to over 24,000 leukocytes/μL, accompanied by neutrophilia and a left shift of the leukocyte differential. C-reactive protein (CRP) values were greatly elevated, reaching 180–320 mg/L, which is consistent with the systemic inflammatory response expected in an infection of this severity. In addition, other laboratory disturbances reflecting the seriousness of the condition were observed:
hypoalbuminemia (values from 22 to 30 g/L), a sign of marked catabolism and capillary leakage;
hyperglycemia (values above 9–14 mmol/L), mainly in the two patients with previously diagnosed diabetes mellitus;
moderate elevation of urea and creatinine as a result of hypovolemia and the prerenal component of kidney injury;
electrolyte disturbances — hyponatremia (130–134 mmol/L) and hypokalemia in two cases;
moderate elevation of hepatic transaminases;
mild hyperbilirubinemia;
a tendency toward metabolic acidosis, with serum lactate elevation above 2.5 mmol/L in two patients;
mild coagulogram disturbances (prolonged INR, decreased platelets) which partly reflected the septic state.
After resolution of the infectious focus by debridement and after the initiation of targeted antibiotic therapy, the inflammatory parameters began to fall progressively. Leukocyte values normalized typically within 7–10 days, while CRP took 10–14 days to reach near-normal values. Serum albumin recovered more slowly and often required enteral and parenteral nutritional support.

3.3. Microbiological findings: Immediately after admission and during the first debridement, swabs were taken from the necrotic tissue and the wound discharge for aerobic culture and — when technically possible — anaerobic culture. All four cultures yielded polymicrobial growth, a fact that fully corresponds to the synergistic nature of Fournier's gangrene [6]. The pathogens isolated were: methicillin-resistant *Staphylococcus aureus* (MRSA), *Escherichia coli, Pseudomonas aeruginosa,* and *Enterococcus spp.* The combinations of pathogens for each patient were as follows:
Patient 1 and Patient 2: *MRSA + Escherichia coli;*
Patient 3: *MRSA + Escherichia coli + Pseudomonas aeruginosa;*
Patient 4: *MRSA + Enterococcus spp. + Pseudomonas aeruginosa.*

The notable observation in our microbiological context is the constant presence of MRSA in all four cases — a phenomenon that reflects a global epidemiological trend of increased MRSA colonization and infection in surgical patients [16,18]. The presence of *Pseudomonas* in two patients is consistent with the nature of the contaminating nosocomial environment and with an increased risk factor due to weakened immunity [6]. None of the cultures identified pure anaerobes, but this may reflect the technical difficulties of transporting and cultivating anaerobes, and does not exclude their participation, which is well known in the pathogenesis of FG [3,6].

3.4. Antibiotic susceptibility profile

The antibiogram was performed in accordance with the institutional standards and current guidelines (EUCAST/CLSI). The susceptibility profile of the pathogens isolated in our patients, combined with typical data from the current literature [15,16,20,21], is summarized in Table 2.

Table 2. Antibiotic susceptibility and resistance profile for the isolated pathogens (combination of our data and current literature)

Bacterium	Antibiotics with susceptibility (effective)	Antibiotics with resistance (ineffective)
MRSA	Vancomycin, teicoplanin, linezolid, daptomycin, clindamycin (when the strain is not iMLSB), tigecycline, trimethoprim-sulfamethoxazole, doxycycline, ceftaroline (5th-generation cephalosporin), rifampicin (only in combination).	All penicillins and antistaphylococcal penicillins (oxacillin, methicillin, cloxacillin), aminopenicillins with/without inhibitor (amoxicillin-clavulanate), most cephalosporins of generations I–IV (cefazolin, cefuroxime, cefotaxime, ceftriaxone, cefepime), carbapenems (imipenem, meropenem, ertapenem) — according to the classical laboratory definition. *Note: in our patients the antibiogram for MRSA showed susceptibility to meropenem and to ceftriaxone, a finding that is significant only in vitro and that must be interpreted with clinical caution.*
E. coli	Carbapenems (meropenem, imipenem, ertapenem), aminoglycosides (amikacin, gentamicin), ciprofloxacin, ceftriaxone, cefepime, piperacillin-tazobactam, fosfomycin, nitrofurantoin (only for urinary infections), tigecycline.	Ampicillin (frequent resistance), amoxicillin-clavulanate (variable resistance), cotrimoxazole (in ESBL-positive strains), generation I–II cephalosporins in many nosocomial strains. Strains producing extended-spectrum β-lactamases (ESBL) are resistant to all generation III–IV cephalosporins.
P. aeruginosa	Ceftazidime, cefepime, piperacillin-tazobactam, antipseudomonal carbapenems (meropenem, imipenem, doripenem — not ertapenem), aminoglycosides (amikacin,	All aminopenicillins, non-pseudomonal cephalosporins (cefazolin, cefuroxime, ceftriaxone, cefotaxime), ertapenem, cotrimoxazole,

Bacterium	Antibiotics with susceptibility (effective)	Antibiotics with resistance (ineffective)
	tobramycin, gentamicin), ciprofloxacin, levofloxacin, aztreonam, polymyxins (colistin, polymyxin B), ceftolozane-tazobactam.	tigecycline, macrolides, lincosamides.
Enterococcus spp.	Ampicillin (first choice for susceptible strains, especially *E. faecalis*), vancomycin, teicoplanin, linezolid, daptomycin, tigecycline, nitrofurantoin (for UTI), fosfomycin (for UTI). For endocarditis, combinations of ampicillin or vancomycin with an aminoglycoside are used.	All cephalosporins (intrinsic/innate resistance), oxacillin and antistaphylococcal penicillins, clindamycin (intrinsic resistance), aminoglycosides as monotherapy, cotrimoxazole (in vivo). VRE strains (*E. faecium*) are resistant even to vancomycin.

Note: The above table presents susceptibility according to the typical profiles of susceptible strains. The actual profile varies from case to case and should always be guided by the individual antibiogram of each patient. It must be particularly emphasized that Enterococcus spp. have intrinsic (innate) resistance to all cephalosporins; these should never be used as monotherapy for enterococci, regardless of any in vitro value.

3.5. Treatment and clinical course: In all four patients, treatment was initiated immediately according to the institutional algorithm. The empirical antibiotic therapy included a combination covering aerobes and anaerobes — usually meropenem or piperacillin-tazobactam, plus vancomycin (for MRSA coverage pending the antibiogram), plus clindamycin or metronidazole for additional anaerobic coverage [15,16]. After the antibiogram results were obtained, therapy was de-escalated and individually adapted: vancomycin was retained for MRSA in all cases, while for the gram-negative pathogens meropenem, ciprofloxacin, or ceftazidime (in the cases with *Pseudomonas*) were used.

Hemodynamic resuscitation was performed with crystalloid solutions and, as needed, colloids; electrolyte and acid-base correction was performed according to laboratory values; nutritional support was provided mainly via the enteral route with protein and vitamin supplements. Analgesia was administered according to the WHO scale, with a combination of paracetamol, non-steroidal anti-inflammatory drugs, and weak opioids when necessary. Thromboembolic prophylaxis with low-molecular-weight heparin was given after stabilization of hemostasis. We also maintained strict glycemic control with intravenous insulin in the acute phase.

Wounds were inspected and dressed twice daily during the initial phase. After the formation of a good granulation bed — usually achieved within 7–14 days — the phase of definitive closure was initiated.

Definitive treatment of the large defects of the abdominal wall and the inguino-scrotal area was performed with split-thickness skin autografting (according to the classical Thiersch–Ollier technique), harvested with a dermatome from the anterolateral region of the thigh [9,12]. The skin strips were processed using the meshing technique (1:1.5) in order to increase the coverage area and to allow drainage of any collections beneath the graft. In two patients, in addition to the skin autografting, the scrotal defects were resolved by advancement of the remaining scrotal skin and by bringing the testicles into a medial position. No patient required large local flaps or distant musculocutaneous flaps.

The length of hospital stay ranged from 10 days (in the youngest patient, with more limited spread) to three weeks (in the older patients with larger defects requiring longer wound bed preparation). All four patients were discharged in good general condition, with skin grafts adherent over more than 90% of the area at the first postoperative checks. No deaths, no major complications (resistant sepsis, multi-organ failure, massive hemorrhage), and no recurrence of the disease were recorded during the period of regular postoperative follow-up.

Discussion

Our experience with four patients confirms several fundamental principles of the management of Fournier's gangrene that have been established by the international literature of the last three decades, but also raises some questions that deserve more detailed discussion. The age of our patients (38–62 years) is slightly lower than the mean age reported in large European and American series (usually 50–60 years), but it is within the usual range [1,3,19]. All patients were male, which corresponds to the well-known male-to-female ratio of approximately 10:1 [1,3].

Part of the literature classifies FG according to the portal of entry: urogenital, anorectal, cutaneous (perineal or abdominal), and idiopathic [2–4]. In our patients, the two cases with an initial presentation of purulent collections in the lower abdominal wall suggest a cutaneous or subcutaneous portal of entry (perhaps a folliculitis, a subcutaneous abscess of the lower abdomen, or a local infection from the rupture of an infected atheroma) which then evolved very rapidly toward fascial and skin necrosis. This atypical presentation is a well-known diagnostic pitfall — the fact that the skin overlying the infection initially appears almost normal often leads to underestimation of the real severity and delays complete surgical intervention [4,5]. The practical lesson for the cautious surgeon: any open abscess of the lower abdominal wall in a patient with risk factors should be evaluated closely during the first 48–72 hours, with a low threshold for extended debridement if there are signs of fascial spread.

The microbiological profile in our patients confirms the polymicrobial nature of FG, which is reported in over 70–80% of cases in the literature [6]. The combination of gram-positive microbes (MRSA, *Enterococcus*) with gram-negative ones (*E. coli*, *Pseudomonas*) and — most likely — anaerobes that were not isolated for technical reasons, constitutes the classical synergy that sustains the necrotizing process [3,6]. The finding of MRSA in all four cases deserves attention. Some European series report a continuous increase in MRSA colonization in soft tissue infections, especially in units with high hospital traffic and extensive use of broad-spectrum antibiotics [16,18]. The practical importance is that in any case suspicious for FG, empirical antibiotic therapy must include an agent against MRSA (vancomycin, linezolid, or daptomycin) from the very first hour [15,16].

With regard to *Pseudomonas*, its presence brings its own considerations. This bacterium is classically resistant to most narrow-spectrum β-lactams and requires specific antipseudomonal antibiotics: ceftazidime, cefepime, piperacillin-tazobactam, antipseudomonal carbapenems (not ertapenem), aminoglycosides (amikacin, tobramycin), ciprofloxacin, aztreonam, and — for highly resistant strains — polymyxins (colistin), ceftolozane-tazobactam, or ceftazidime-avibactam [15]. In our patients, the antibiogram for *Pseudomonas* showed susceptibility to ceftazidime, ciprofloxacin, piperacillin-tazobactam, and amikacin, a fact that allowed a successful de-escalation.

A technical point that should be emphasized is the intrinsic resistance of *Enterococcus* to cephalosporins [20,21]. Despite any susceptibility that may be reported in vitro, cephalosporins do not reach clinically effective concentrations to kill enterococci, because this bacterial genus possesses penicillin-binding proteins (PBPs) with low affinity for β-lactams, as well as alternative pathways for peptidoglycan biosynthesis [21]. As a consequence, even if the

antibiogram suggests susceptibility to a cephalosporin, clinicians should disregard this finding and choose ampicillin (when the strain is susceptible), vancomycin, linezolid, or daptomycin [20,21]. In Patient 4, therapy was directed toward vancomycin (which covered both MRSA and *Enterococcus*) plus an antipseudomonal agent.

Surgical treatment remains the main pillar of healing. The literature is unanimous: the time between admission and the first debridement is an independent prognostic factor — delays beyond 24 hours double mortality in some series [3,11,19]. In our patients, debridement was performed within the first hours after the diagnosis was established. The size of the defect after debridement, although large, is not an indicator of poor prognosis if the antibiotic coverage is appropriate and if a strategy for secondary coverage is planned from the outset.

With regard to definitive reconstruction, split-thickness skin autografting remains the most common and most practical solution for large defects of the abdominal wall and the inguinal area [9,12]. Local flaps (medial thigh, inguinal flap, gracilis flap) and perforator flaps are valuable alternatives in centers with plastic surgery expertise, especially when defects are very large or when important organs are exposed (testicle, penis, sacrum) [9]. In our series, although extensive, the spread of the defect in the abdominal wall did not reach dimensions that would require complex flaps, and skin grafting provided satisfactory cosmetic and functional results.

Several adjuncts have been widely discussed in the literature. Hyperbaric oxygen, although not available in every center (including our institution), has shown potential benefits in anaerobic strains and in wounds with marked ischemia; however, current evidence is not sufficient to recommend it as a standard [11,12]. Fecal diversion with protective colostomy is reserved for cases with active perianal contamination or with damaged sphincters — conditions that were not observed in our patients [11,19].

The summary of findings and the limitations of this study should be honestly stated. The small number of cases (n=4) and the retrospective nature do not allow advanced statistical analyses, but they at least offer a clear picture of clinical practice in a regional center. The positive results (absence of mortality, reasonable length of hospital stay, effective wound closure) suggest that a unified algorithm — rapid diagnosis, radical debridement, antibiogram-guided antibiotic therapy,

Conclusion

Fournier's gangrene remains a surgical emergency that requires immediate and coordinated action. Our experience with four patients treated at the Surgery Clinic of the Clinical Hospital of Tetovo confirms that a structured algorithm — based on rapid clinical diagnosis, aggressive hemodynamic resuscitation, radical and early surgical debridement with repetition as needed, broad-spectrum empirical antibiotic therapy followed by antibiogram-guided de-escalation, and definitive reconstruction with split-thickness skin autografting — provides good clinical results even in a regional hospital [3,9,10,15].

The polymicrobial profile with predominance of MRSA in all four cases emphasizes the importance of empirical coverage against MRSA from the start of treatment, and raises the need for continuous epidemiological surveillance of antimicrobial resistance in our hospitals [16,18]. The atypical presentation of two patients with purulent collections in the abdominal wall, which evolved rapidly toward necrosis, reminds surgeons that any deep infection of the abdominal wall in a patient with risk factors should be evaluated with a high suspicion for an extended necrotizing fascial process [4,5].

Future studies with larger numbers of patients, the systematic inclusion of anaerobic pathogens, the systematic evaluation of prognostic indices (FGSI, UFGSI, LRINEC), and long-term functional follow-up of these patients would help to further refine local algorithms [7,8,13,14].

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