

Necrotizing soft tissue infections (NSTIs)

Literary review and description of a Fournier syndrome case



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Necrotizing soft tissue infections (NSTIs):Literary review and description of a Fournier syndrome case.

A 62 y.o. male with tight phimosis, swelling, redness, pain on palpating and necrosis of the penis and scrotum was admitted in our clinic, (FGSI = 6) with peripheral vasculopathy; and diabetes mellitus type II and he was in dialysis treatment before the hospitalization. The patient was HCV affected. In 24 hours he underwent radical surgical debridement with excision of all necrotic material from penis and scrotum up to the subdermal layer and tissue of doubtful viability for about 75% of the skin and circumcision. In third, fifth and seventh postoperative days he underwent to local infusion of autologous PLT growth factors. The patient was discharged in 9th postoperative day and FGSI was still 6; the skin and subdermal tissue was barely reskined, with low homogeneous granulation, edema was heavily reduced. In our case, deviation from homeostasis status at admission was the main worrying factor. We found that diabetes mellitus and renal dysfunction at admission was also important risk factor for FG. "E.Coli" was the most common organism isolated from patient wound cultures. The FGSI is an objective and easy to apply score method to quantify the metabolic status and can be used to evaluate therapeutic options and assess results.

KEY WORDS: Fascitis, Fournier, Gangrene, Infection, Necrotizing, Syndrome, Tissue.

Introduction

Fournier Gangrene was first described in 1883 by a French venereologist, Jean Alfred Fournier ¹, who identified the syndrome by three findings: abrupt onset in a healthy young man, rapid progression, and the absence of a specific causative agent. Fournier gangrene belongs to the group of local non-specific infection of soft tissues (NSTI) a group of uncommon, rapidly progressive, potentially fatal disorders ^{2,3}. It is characterized by extensive necrosis of superficial and deep fascial planes pro-

ducing a necrotizing fasciitis with loss of subcutaneous tissue and skin of the perineum and abdominal wall along with the scrotum and penis in males and the vulva in females ^{4,5}. Main feature is the predilection to patients with diabetes, immunosuppression, malignant diseases or other debilitating state ⁶. Mortality rate is still high at between 0% and 67% ^{2,4,5,7,15}. Keys to successful management of patients with necrotizing soft tissue infection are early recognition, complete surgical debridement and early initiation of appropriate broad-spectrum antibiotic therapy ⁸.

Materials and methods

A 62 years old male with tight phimosis, swelling, redness, pain on palpating and necrosis of the penis and scrotum was admitted in our clinic (Fig. 1). Laboratory results at the admission showed the following parameters used to evaluate FGSI: Temperature was 36,4° C;

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Fig. 1: Admission.



Fig. 2: Intraoperative.

Variables	High abnormal values					Low abnormal values			
	+4	+3	+2	+1	0	+1	+2	+3	+4
Temperature °C	>41	39-40.9	–	38.5-35.9	36-38.4	34-35.9	32-33.9	30-31.9	<29.9
Heart rate	>180	140-179	110-139	–	70-109	–	55-69	40-54	<39
Respiration rate	>50	35-49	–	25-34	12-24	10-11	6-9	–	<5
Serum sodium, mmol/l	>180	160-179	155-159	150-154	130-149	–	120-129	111-119	<110
Serum potassium, mmol/l	>7	6-6.9	–	5.5-5.9	3.5-5.4	3-3.4	2.5-2.9	–	<2.5
Serum creatinine, mg/100 ml, x2 for acute renal failure	>3.5	2-3.4	1.5-1.9	–	0.6-1.4	–	<0.6	–	–
Hematocrit, %	>60	–	50-59.9	46-49.9	30-45.9	–	20-29.9	–	<20
White blood cell count, x 10 ⁹ /l	>40	–	20-39.9	15-19.9	3-14.9	–	1-2.9	–	<1
Serum bicarbonate, venous, mmol/l	>52	41-51.9	–	32-40.9	22-31.9	–	18-21.9	15-17.9	<15

Fig. 3: (Fournier's gangrene severity index) 27.

heart rate 95 Bpm; respiration rate 18 bpm; serum Na 135 mEq/L; serum K 5.15 mEq/L; serum Creatinine 7.82 mg/dl; Hct 22.9 %; Wbc 14.8 10³/uL; serum bicarbonate 24.6 mmol/L. Admission FGSI was 6 (according to Fig. 3). Others parameters were: Hgb 7.5 g/dL; Plt 280 10³/uL; Gluc. 112 mg/dL; albumin 2.7 g/dL; ALT/GPT 43 u/L; AST/GOT 16 u/L. The patient underwent to Chest X ray and cardiological advice that showed circulatory compensation with periferic vasculopathy; he was affected by diabetes mellitus type II and he was in dialysis treatment before the hospitalization. The patient was HCV affected. He underwent to two blood transfusions of whole blood. In 24 hours he underwent radical surgical debridement (Fig. 2) with excision of all necrotic material from penis and scrotum up to the subdermal layer and tissue of doubtful viability for

about 75% of the skin and circumcision. In third, fifth and seventh postoperative days he underwent to local infusion of autologous PLT growth factors. The patient was discharged in 9th postoperative day and FGSI was still 6; Wbc were 8.0 10³/uL; Hgb 8.8 g/dL; serum K 4.48 mEq/L; the skin and subdermal tissue was barely reskined, with low homogeneous granulation, edema was heavily reduced. At 1 month follow up we found a good reepithelialization so that the patient did not need any other surgical treatment.

Results

The total reepithelialization of penis and scrotum occurred in six weeks.

Keys to successful management of patients with necrotizing soft tissue infection are early recognition and complete surgical debridement and early initiation of appropriate broad-spectrum antibiotic therapy. Soft tissue reconstruction should take into account both functional and cosmetic outcome¹⁶. Critical care management components such as the initial fluid resuscitation, end-organ support, pain management, nutrition support, and wound care are all important aspects of the therapeutic strategy. Additional innovative strategies for the treatment of Necrotizing soft-tissue infections (NSTIs), including intravenous immuno-globulin G (IVIG), hyperbaric oxygen, and vacuum-assisted closure, do not yet have definitive evidence of efficacy, but may be considered in patients at high risk of death.

Discussion

Necrotizing soft-tissue infections (NSTIs) are a group of uncommon, rapidly progressive, potentially fatal disorders that include necrotizing cellulitis, adipositis, fasciitis and myositis/myonecrosis. It is a class of aggressive severe soft tissue infection that cause rapid and widespread highly lethal infection and necrosis of the skin and soft tissues. NSTIs are associated with significant morbidity and mortality². Fournier's gangrene belongs to the group of local non-specific infection of soft tissues (NSTI)^{2,16}; it may extend to the abdomen, lower limbs and chest¹¹. The bacterial infection, in cases originating in the genital area, probably pass through Buck's fascia of the penis and spread along the Dartos' fascia of the scrotum and penis, Colles' fascia of the perineum, and Scarpa's fascia of the anterior abdominal wall. It is a rare process having an unknown origin and affects any age and gender; its incidence is relatively low, but the infection is extraordinary aggressive^{17,18}. It is often caused by a wide spectrum of pathogens and is most frequently polymicrobial by aerobic and anaerobic bacteria with a synergistic action¹⁹. The most commonly isolated microorganisms are *E. coli* and *Enterococcus faecalis*, but also *Pseudomonas aeruginosa*, *Acinetobacter*, *Peptostreptococcus* and streptococcal species, *Staphylococcus aureus*, *Providencia rustigianii*, and anaerobic flora^{7,14,18,20-22}. Fournier syndrome is characterized epidemiologically by

predilection to patients with diabetes, immunosuppression, or other debilitating state like superficial traumas, urological and colorectal diseases and operations⁶; alcoholism, low personal hygiene are perpetuating co-factors or inciting factor; a post varicella infection case is also reported in literature, but diabetes mellitus remains the most important risk factor in the outcome of disease, followed by cirrhosis of the liver, uremia and underlying malignancy^{4,23-25}. Mortality rate, from literature, remains high at between 0% and 67%^{2,4,5,7,8,9,12,14,15}, despite some Authors report a decrease from 25-50% in past years, to 10-16%, other reports from the last two years range it from 10 to 41.2% (average: 20.75%)^{2-6,9,11,14,18,21,26}. In literature the factors affecting survival were not finely assessed until the study of Laor et al. in 1995, in which measurable pathophysiologic data between survivors and non-survivors of FG were compared²⁷.

Despite FG seems to be an unpredictable disease process with wide variability in its presentation²⁶, the severity index for Fournier's gangrene, described by Laor in 1995 (Fig. 3), is useful for evaluating the prognosis of these patients. High Charlson Comorbidity Index (CCI) and Fournier's Gangrene Severity Index (FGSI) scores might be associated with a worse prognosis in patients with FG.²⁸ Different papers show a strong correlation between the FGSI and mortality²⁰. In the Laor case series the mean FGSI for survivors was 6.9 +/- 0.9 compared to 13.5±1.5 for non survivors.²⁷ Latest case series report similar results having admission scores from 3.0 to 6.7 for survivors and from 8.7 to 13.6 for non-survivors (20, 26, 27, 28, 29, 30) (Table I).

A FGSI threshold of 9 is considered a predictor of mortality during the initial assessment²⁸ (>9: 75% probability of death; <9 : 78% probability of survival)²⁷. We agree that the Fournier's gangrene severity index is an objective and simple method to quantify the extent of metabolic aberration that may be used to predict outcome²⁷. Patients' metabolic status and the extent of disease at admission are important factors in the prognosis of Fournier's gangrene²⁹, but deviation from homeostasis is the main predictive parameter of outcome more significant than the extent of disease or performance of surgical débridement. Laboratory parameters correlated with outcome included urea, creatinine, sodium, and

Table I

Authors / Year	Admission FGSI survivors	Admission FGSI non survivors	Regression analysis correlation coefficient	
Laor / 1995	6.9 ± 0.9	13.5 ± 1.5	= 0.934; p = 0.005	(27)
Yeniyol / 2004	3.0 ± 1.8	12 ± 2.4	P < or = 0.0001	(29)
Ulu / 2009	5.04 ± 2.49	13.6 ± 4.61	P < 0.0001	(20)
Cyzmet / 2009	5.04 ± 2.49 (0-9)	13.6 ± 4.61 (10-20)	P=0.001	(30)
Erol / 2010	5.00 ± 2.91	13.5 ± 2.62	P = 0.001	(28)
Luján / 2010	6.7 (0-14)	8.7 (6-13)	P = 0.12	(26)

potassium, hematocrit, albumin, bicarbonate, calcium, alkaline phosphatase and cholesterol levels. Low magnesium levels might be another parameter for a worse prognosis²⁸. White blood count, platelets, total protein, and lactic dehydrogenase were also associated with outcome^{20, 27}. The following factors even worsens the illness prognosis: delay in diagnostic, higher age, anorectal origin of infection, the amount of organ with dysfunction or failure³¹. The number of surgical debridements does not seem to influence outcome²⁷, but delayed debridement seems to be a significant factor affecting the survival rate, while early and aggressive debridement is a significant prognostic factor⁴. Good results, in recent years with aggressive surgical and medical management have been reached. Care for patients with necrotizing soft tissue infection requires a team approach with expertise from critical care, surgery, reconstructive surgery, and rehabilitation specialists. A comprehensive knowledge of the pathophysiology, diagnostic features, causative microbial pathogens, and treatment strategies (including surgical debridement and antimicrobial therapy) is required for successful management of NSTIs².

Conclusions

Fournier Gangrene is a progressive, fulminant infection. Its mortality rate can be cut down by early diagnosis, aggressive surgical intervention, and the use of broad-spectrum antibiotics. In our case, deviation from homeostasis status at admission was the main worrying factor. We found that diabetes mellitus and renal dysfunction at admission was also important risk factor for FG. "E.Coli" was the most common organism isolated from patient wound cultures. The FGSI is an objective and easy to apply score method to quantify the metabolic status and can be used to evaluate therapeutic options and assess results.

Riassunto

Paziente 62enne maschio con fimosi serrata edema e necrosi, del pene e dello scroto ricoverato nella nostra divisione di chirurgia, (FGSI all'ingresso = 6) con vasculopatia periferica, diabete mellito di tipo II, in trattamento dialitico e portatore cronico di HCV. In 24 ore si è sottoposto a debridement chirurgico radicale con asportazione di tutto il materiale necrotico dal pene e dallo scroto fino allo strato sottocutaneo per circa il 75% della cute e circoincisione. Nel post-operatorio (terza, quinta e settima giornata) è stato sottoposto a infusione locale di fattori di crescita piastrinici autologhi. Il paziente è stato dimesso in 9^a giornata con FGSI di 6. La cute e il tessuto sottocutaneo erano per la maggior parte in via di granulazione/riepitelizzazione ed anche l'edema si mostrava ridotto. In questo caso cli-

nico la principale problematica è stata l'omeostasi ionica, risolta con adeguata terapia infusiva. Abbiamo riscontrato che il diabete mellito e la disfunzione renale sono importanti fattori di rischio per la sindrome di Fournier. "E. Coli" è il più comune microrganismo isolato da colture di tessuto gangrenoso. Il FGSI è una scala obiettiva e facile da applicare per quantificare lo stato metabolico e può essere utilizzato per valutare le opzioni terapeutiche e i risultati.

References

1. Fournier AJ: *Gangrène foudroyante de la verge*, Sem Méd., 3, 345, 1883. Dis Colon Rectum, 1988; 31(12):984-88.
2. Sarkar B, Napolitano LM: *Necrotizing soft tissue infections*. Minerva Chir, 2010; 65(3):347-62.
3. Mills MK, Faraklas I, Davis C, Stoddard GJ, Saffle J: *Outcomes from treatment of necrotizing soft-tissue infections: Results from the National Surgical Quality Improvement Program database*. Am J Surg, 2010; 200(6):790-96; discussion 796-97.
4. Chen SY, Fu JP, Wang CH, Lee TP, Chen SG: *Fournier gangrene: a review of 41 patients and strategies for reconstruction*. Ann Plast Surg, 2010; 64(6):765-69.
5. Martínez-Rodríguez R, Ponce de León J, Caparrós J, Villavicencio H: *Fournier's gangrene: a monographic urology center experience with twenty patients*. Urol Int, 2009; 83(3):323-28. Epub 2009 Oct 13.
6. Katusin D, Crnica S: *Fournier's gangrene. Our experience with 17 patients*. Lijec Vjesn, 2010; 132(3-4):86-89.
7. Katsanos KH, Ignatiadou E, Sarandi M, Godevenos D, Asproudis I, Fatouros M, Tsianos EV: *Fournier's gangrene complicating ulcerative pancolitis*. J Crohns Colitis, 2010; 4(2):203-6. Epub 2009 Dec 21.
8. Yilmazlar T, Ozturk E, Ozguc H, Ercan I, Vuruskan H, Oktay B: *Fournier's gangrene: an analysis of 80 patients and a novel scoring system*. Tech Coloproctol, 2010; 14(3):217-23. Epub 2010 Jun 18.
9. Canbaz H, Calikulekci M, Altun U, Dirlik M, Turkmeno lu O, Ta delen B, Aydin S: *Fournier's gangrene: analysis of risk factors affecting the prognosis and cost of therapy in 18 cases*. Ulus Travma Acil Cerrahi Dergm 2010; 16(1):71-76.
10. Gamboa EO, Rehms EH, Haller N: *Fournier's gangrene as a possible side effect of bevacizumab therapy for resected colorectal cancer*. Clin Colorectal Cancer, 2010; 9(1):55-58.
11. Montoya Chinchilla R, Izquierdo Morejon E, Nicolae Pietricică B, Pellicer Franco E, Aguayo Albasini JL, Miñana López B: *Fournier's gangrene. Descriptive analysis of 20 cases and literature review*. Actas Urol Esp, 2009; 33(8):873-80.
12. Sorensen MD, Krieger JN, Rivara FP, Klein MB, Wessells H: *Fournier's gangrene: management and mortality predictors in a population based study*. J Urol, 2009; 182(6):2742-747. Epub 2009 Oct 17.
13. Czymek R, Hildebrand P, Kleemann M, Roblick U, Hoffmann M, Jungbluth T, Bürk C, Bruch HP, Kujath P: *New insights into the epidemiology and etiology of Fournier's gangrene: A review of 33 patients*. Infection, 2009; 37(4):306-12. Epub 2009 Jul 23.

14. Kara E, Müezzino lu T, Temeltas G, Dinçer L, Kaya Y, Sakarya A, Coskun T: *Evaluation of risk factors and severity of a life threatening surgical emergency: Fournier's gangrene (a report of 15 cases)*. Acta Chir Belg, 2009; 109(2):191-97.
15. Sorensen MD, Krieger JN, Rivara FP, Broghammer JA, Klein MB, Mack CD, Wessells H: *Fournier's Gangrene: population based epidemiology and outcomes*. J Urol, 2009; 181(5):2120-126. Epub 2009 Mar 14.
16. Phan HH, Cocanour CS: *Necrotizing soft tissue infections in the intensive care unit*. Crit Care Med, 2010; 38(9 Suppl):S460-68.
17. Rodríguez Cancio MC, Verdú Moresco A, Lorente Fernández G: *Fournier's gangrene, a battle won. Traditional cures versus a polyhexanide solution*. Rev Enferm. 2008; 31(11):39-43.
18. Morua AG, Lopez JA, Garcia JD, Montelongo RM, Guerra LS: *Fournier's gangrene: our experience in 5 years, bibliographic review and assessment of the Fournier's gangrene severity index*. Arch Esp Urol, 2009 ;62(7):532-40.
19. Sarkis P, Farran F, Khoury R, Kamel G, Nemr E, Biajini J, Merheje S: *Fournier's gangrene: A review of the recent literature*. Prog Urol, 2009; 19(2):75-84. Epub 2008 Nov 28.
20. Ulu M, Gedik E, Girgin S, Celen MK, Ayaz C: *The evaluation of microbiology and Fournier's gangrene severity index in 27 patients*. Int J Infect Dis, 2009; 13(6):e424-30. Epub 2009 May 2.
21. Jensen P, Zachariae C, Grønhoj Larsen F: *Necrotizing soft tissue infection of the glans penis due to atypical Candida species complicated with Fournier's gangrene*. Acta Derm Venereol, 2010; 90(4):431-32.
22. Torremadé Barreda J, Millán Scheiding M, Suárez Fernández C, Cuadrado Campaña JM, Rodríguez Aguilera J, Franco Miranda E, Biondo S: *Fournier gangrene. A retrospective study of 41 cases*. Cir Esp. 2010 Apr; 87(4):218-23. Epub 2010 Feb 11.
23. Dey S, Bhutia KL, Baruah AK, Kharga B, Mohanta PK, Singh VK: *Neonatal Fournier's gangrene*. Arch Iran Med, 2010; 13(4):360-62.
24. Jefferies M, Saw NK, Jones P: *Fournier's gangrene in a five year old boy. Beware of the child post varicella infection*. Ann R Coll Surg Engl, 2010; 92(5):W62-63.
25. Malik AM, Sheikh S, Pathan R, Khan A, Sheikh U: *The spectrum of presentation and management of Fournier's gangrene. An experience of 73 cases*. J Pak Med Assoc, 2010; 60(8):617-19.
26. Luján Marco S, Budia BM, Di Capua C, Broseta E, Jimenez Cruz F: *Evaluation of a severity score to predict the prognosis of Fournier's gangrene*. BJU Int, 2010; 106(3):373-6. Epub 2009 Nov 17.
27. Laor E, Palmer LS, Tolia BM, Reid RE, Winter HI: *Outcome prediction in patients with Fournier's gangrene*. J Urol, 1995; 154(1):89-92.
28. Erol B, Tuncel A, Hanci V, Tokgoz H, Yildiz A, Akduman B, Kargi E, Mungan A: *Fournier's gangrene: Overview of prognostic factors and definition of new prognostic parameter*. Urology, 2010; 75(5):1193-198.
29. Yeniyol CO, Suelozgen T, Arslan M, Ayder AR: *Fournier's gangrene: experience with 25 patients and use of Fournier's gangrene severity index score*. Urology, 2004; 64(2):218-22.
30. Czymek R, Hildebrand P, Kleemann M, Roblick U, Hoffmann M, Jungbluth T, Bürk C, Bruch HP, Kujath P: *New insights into the epidemiology and etiology of Fournier's gangrene: a review of 33 patients*. Infection, 2009 ;37(4):306-12. Epub 2009 Jul 23.
31. Zonca P, Mrázek T, Matusek A, Hajek M, Stiglerová S, Jurytko A, Kucera C, Nieslanik M: *Septic shock in a patient with the Fournier gangrene with fatal outcome* Rozhl Chir, 2009; 88(7):398-402.

