



SUCCESSFULLY MANAGED CASE OF Fournier's GANGRENE - A CASE REPORT.

General Surgery

**Prof. Dr.
S.R.Dhamotharan**

M.S.,F.I.A.G.E.S Professor And HOD, Department of General Surgery,velammal Medical College Hospital And Research Centre, Madurai, Tamilnadu.

**Anupritha
Arudhra
Hamshadhwanaa***

M.B.B.S INTERN,Velammal Medical College Hospital and Research Centre, Madurai,Tamilnadu. *Corresponding Author

ABSTRACT

Fournier's gangrene [FG] is an acute vascular gangrene of infective origin,causing fulminant inflammation of scrotal skin and subcutaneous tissues,progressing rapidly leading to infective necrotising fascitis of perineum and genitalia and even ascends to abdominal wall and pelvis..It is a rare condition with increased mortality rate.It affects colle's fascia,Dartos fascia,Buck's fascia and ascends to scarpa's fascia and fascia of camper.According to a study it affects 1 in 7500 people,majority of them being men.Here we report a case of fournier's gangrene in a 43 year old male managed successfully.

KEYWORDS

Fournier's Gangrene,Idiopathic scrotal gangrene.

INTRODUCTION

Fournier's Gangrene is a fulminant necrotising fascitis of the perineum, external genitalia,inguinal,anterior abdominal wall and perianal regions.

Reported incidence of Fournier's Gangrene was 1.6-3/1,00,000 with 10:1 male preponderance[7].It presents commonly in old aged men [6th-7th decade], diabetics,malnourished and immunosuppressed individuals.

Here we report a case of fournier's gangrene in a 43 year old male diabetic who presented with scrotal abscess initially which then progressed to gangrene.

CASE REPORT

A 43 year old male from Madurai came to the hospital with complaints of painful scrotal swelling for 10 days with fever for 3 days.The course was rapid and fulminant with skin gradually becoming blackish with a sore on the dorsum of scrotum.The patient is a known case of Type 2 DM for 5 years on medications prescribed elsewhere.

On general examination,patient was conscious,oriented,febrile Temperature-102 degree F,Heart rate-110/minute,Respiratory rate-18/minute,comfortable at rest with good hydration.His vitals were stable.On local examination,inspection of external genitalia,there was scrotal wall edema with areas of patchy gangrene and a pressure sore seen at the dorsum of scrotum with foul smelling discharge.On palpation, there was warmth and tenderness in whole scrotal skin with crepitus.Both testis and cord structures palpable normally.Hence it is diagnosed as fournier's gangrene evolving from scrotal abscess.

Lab investigation reports showed raised blood glucose-356mg/dL,urea-50mg/dL and serum creatinine 1.8mg/dL,raised WBC count-11.0 thou/mm³,platelet-425 thou/mm³,Neutrophils 81percent,ESR-60mm/hr,CRP-96mg/dL,PCV-40percent,Hemoglobin-12gms/dL,Sodium-130mEq/L,Potassium-5mEq/L,Bicarbonate-24mEq/L.Emergency pus drainage and scrotal exploration done under anaesthesia and around 200ml of pus drainage done.By an incision from median raphe extending into both inguinal regionsgangrenous scrotal skin was excised.Hydrogen peroxide with povidone iodine saline wash given and after perfect hemostasis,sterile dressing and scrotal support applied.

With daily slough excision and dressing,under antibiotic cover according to pus culture and sensitivity and with strict euglycemic control,after 15days wound granulated well except on the left side where patient developed peritesticular infection.Hence under spinal anaesthesia with aseptic precautions,edges of wound scraped and granulation tissue scooped out.Left side cord structures were dissected and ligated and left testis removed.Rest of scrotal skin undermined, scrotal skin flaps mobilised and wound closed with drain.Specimen sent for histopathology,reports ruled out testes involvement.

FIGURES



Fig. a.

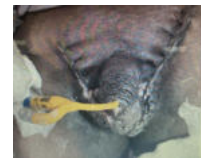


Fig. b.

a.Figure showing scrotum after gangrenous skin excision.
b.Figure showing reconstructed scrotum after left orchiectomy.

DISCUSSION

Fournier's Gangrene is a rare life threatening vascular gangrene of infective cause,infection commonly due to synergistic,polymicrobial origin.Its incidence peaks after 50 years of age.[1].The disease was first reported by Baurienne in 1764 [2].

In 1883,Jean-Alfred Fournier,a venereologist,reported five cases of previously healthy young male with progressive gangrene of the penis and scrotum in 1883[3].

The disease was named after him.Some predisposing factors for FG are diabetes mellitus,alcoholism,atherosclerosis,peripheral arterial disease,trauma,tissue injury,malnutrition,immunosuppression,HIV infection,liver disease and leukemia.[4].

Portals of entry of microorganisms in FG are through colorectal, cutaneous or urinary tract.Although overall mortality rate is nearly 20-40percent,it can be as high as 70-80 percent if the patient presents with sepsis.

The diagnosis of FG is usually made by physical examination.Clinical presentation is variable.Tenderness,erythema,swelling and pain are main symptoms.The patient can present with fever,malaise,local discomfort,purulent collection and sepsis[5].

Radiological examinations like USG,Computed Tomography (CT), MRI techniques may be helpful in the diagnosis where diagnosis is uncertain. [6].CT and MRI shows characteristic soft tissue thickening, inflammation, abscesses and subcutaneous gas.

We diagnosed FG in our patient based on characteristic clinical pattern and in our case predisposing factor was diabetes mellitus.

The Laboratory Risk Indicator for Necrotising Fascitis (LRINEC) scoring system includes age, gender, potassium, sodium, creatinine, platelet,C-Reactive Protein,leukocytes,hemoglobin and glucose.[6].

The Fournier's Gangrene Severity Index(FGSI) predict the prognosis

with some parameters, including body temperature, heart rate, respiratory rate, sodium, potassium, creatinine, bicarbonate, leukocytes and haematocrit values.

In our case blood glucose, urea and serum creatinine levels were raised, sodium was low, potassium was high and platelet was high.

FG is a polymicrobial infection of necrotising soft tissues.[6]. Aerobic organisms cause intravascular coagulation by inducing platelet aggregation and complement fixation while anaerobes produce heparinase. Hypoxic tissues lead to formation of oxygen free radicals leading to cell membrane disruption, decreased ATP and DNA damage which leads to decreased protein production. Anaerobic organisms secrete various enzymes toxins like lecithinase, collagenase and hyaluronidase which cause digestion of fascial planes and formation of gas in subcutaneous tissues clinically palpable as crepitus.

In our case the causative organism is *Streptococcus Agalactiae* which is a gram positive, facultative anaerobe.

The mainstays of treatment of FG include urgent radical surgical debridement of necrotic tissues, broad spectrum antibiotics and fluid resuscitation for haemodynamic support. FG is a surgical emergency since the rate of fascial necrosis has been noted to be up to 2-3 cms per hour.

In some cases, orchiectomy has been performed if there is severe peritesticular infection. Testis is not involved in pathological review. It is sometimes possible to place the testes in a temporary subcutaneous pouch until healing or reconstruction is complete. In our case left orchiectomy was done due to severe peritesticular infection on the left side.

After debridement, an open wound must be managed usually with sterile dressings or negative pressure wound therapy [NPWT], redebridement and if needed faecal or urinary diversion. The extensive debridement required in many cases of FG leads to skin and soft tissue defects that necessitates various reconstructive procedures for wound closure like transplantation of testes, free skin grafts, groin and myocutaneous flaps. Goals of reconstruction are acceptable functional and cosmetic results and preservation of penile function.

CONCLUSION

We report this case due to its rarity and to stress the importance of early diagnosis and appropriate management to reduce morbidity and mortality from this fulminant disease condition. It is a rare but life threatening emergency with a high mortality rate. In spite of improvement in treatment strategy, mortality in FG is still high.

COMPLIANCE WITH ETHICAL STANDARDS

Conflict of Interest: None declared.

Informed Consent: Obtained from the participant of the study.

Financial Disclosure: None declared.

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