Case Report

Fournier's gangrene developing secondary to infected hydrocele: A unique clinical scenario

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Abstract

We report the first case of Fournier's gangrene (FG) developing secondary to an infected hydrocele worldwide. We present a case report with a brief overview of the literature relating to FG and its aetiology, diagnosis and management. A 70 year-old male was referred by his General Practitioner with a 2 week history of worsening symptoms of scrotal discomfort and swelling. Following clinical examination, an initial diagnosis of an infected right-sided hydrocele was made and treatment, consisting of antibiotics, was initiated. Despite showing good clinical improvement, several days later, necrotic areas were observed over the right hemiscrotum with spreading cellulitis. A diagnosis of FG was made. The patient was started on triple–therapy antibiotics and taken to the operating room for urgent surgical debridement. Necrotic skin and subcutaneous tissue extending over the perineum and lower anterior abdomen was debrided down to healthy tissue. A further debridement took place 2 days later. The patient continued to improve and was eventually discharged under the care of Plastic Surgeons for reconstruction of the soft tissue defect. FG is a type of necrotising fasciitis predominantly affecting the male perineal, perianal, genital and anterior abdominal wall regions. It has a significant mortality rate, and the key to survival is early detection and treatment consisting of antibiotics and surgical debridement of the affected area. To the best of our knowledge, this is the first reported case of FG developing secondary to an existing hydrocele without any prior urological intervention. The case highlights the important clinical diagnostic and therapeutic interventions required to prevent complications associated with this, potentially fatal, condition.

Key Words: Fournier's gangrene, Fournier's gangrene severity index score, hydrocele, necrotising fasciitis

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INTRODUCTION

Fournier's gangrene (FG) is a necrotising fasciitis involving the perineal and genital regions. It is a life-threatening infection with a high mortality rate. Diagnosis and treatment must be implemented as soon as possible to avoid the potentially fatal consequences of this infection.

We describe a unique case of FG developing after the formation of an infected hydrocele in an adult male.

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CASE REPORT

A 70-year-old gentleman with a past medical history of chronic obstructive airways disease and cor-pulmonale was referred to the Surgical Admissions Unit by his General Practioner with a 2-week history of progressively worsening painful and swollen right scrotum with associated vomiting and fever for the last 1 day. The patient reported no preceding factors and no history of trauma/injury to his genitalia.

On admission, history and examination (consisting of a full systemic review and detailed urological examination) was performed. No lower urinary tract symptoms were reported other than long-standing nocturia (twice). Examination revealed a swollen right hemiscrotum and a visible and palpable hydrocele, which transilluminated. The hydrocele was pre-existing, with a symptom--free interval. No other structures were palpable. A per-rectum examination revealed a slightly enlarged prostate but no evidence of tenderness to suggest prostatitis. No evidence of epididymo-orchitis was suggested. No crepitus or areas of gangrene were noticed. A preliminary diagnosis of an infected hydrocele was made and intravenous antibiotics, including Gentamycin and Augmentin, were started. Blood samples taken on admission showed a white cell count (WCC) of 22.3 × 10^9/l, with marked neutrophil leucocytosis.

The following day, the patient reported much less pain and felt much better. Because of the grossly swollen hemiscrotum, the right testicle could not be palpated nor could a fluid level be elicited. An ultrasound scan confirmed the presence of a tense 7 cm hydrocele. In addition, echogenic material was identified, which was reported as possibly due to a haemorrhage or infection. No comment was made regarding the epidydymis or right testicle, thereby suggesting that this was not affected. The left testicle was reported as normal. Monitoring of the patient's fluid input and output suggested adequate amounts of urine voiding with no symptoms reported by the patient of hesitancy, dysuria, haematuria or frequency.

Three days post-admission, the patient developed a pyrexia of 38°C, with a C-reactive protein (CRP) level of 491.8 mg/L and a leucocytosis of 21.3 × 10^9/l. Four days post-admission, the pyrexia had settled and the antibiotics were changed to oral preparations of Augmentin with a view to the patient being discharged the same day. However, the patient reported difficulty with micturition and, therefore, an ultrasound scan of his bladder was arranged, which showed a large residual of 999 ml of urine. The patient was catheterised. A urine specimen obtained subsequently showed no significant growth. The patient continued to spike a temperature reading of >38°°C and became hypotensive (systolic 74 mmHg; diastolic 54 mmHg – usually >120 mmHg systolic) and further blood cultures were taken. A CRP of 472.5 mg/L was obtained however, the WCC improved to 15.9 × 10^9/l. It was decided to keep the patient in the hospital for further monitoring of his urinary retention.

Seven days post-admission, the patient reported a sizeable reduction in the scrotal swelling. However, on examination, two discrete black lesions were noticed on the scrotum, prompting the diagnosis of FG. A further review of the patient later in the day showed further areas of superficial necrosis as well as a spreading cellulitis (approximately 5 cm proximally toward his abdomen in 6 hours). Repeat blood tests showed a rise in the WCC to 27.4 × 10^9/l, which was predominantly a neutrophil leucocytosis of 25.7 × 10^9/L. IV antibiotics including Tazocin, Metronidazole and Clindamycin were started after discussion with the microbiologists. The patient was taken to the operating theatre for urgent debridement.

During the operation, the skin and subcutaneous tissue was radically debrided (extending throughout the perineum and lower anterior abdomen) to healthy tissue and a right orchidectomy performed due to the non-viable necrotic testicle. Pus and skin obtained was sent to the microbiology department for microscopy, culture and sensitivity. Culture later revealed a heavy growth of *E. coli* and anaerobes, all sensitive to the current antibiotic regimen. Dressings consisted of Jelonet, gauze padding and crepe bandage.

Following the initial debridement, the patient was transferred to the Post-Operative Surgical Unit, where he continued to improve. Two days post-operatively, the patient was taken back to theatre for a second look. Further debridement of additional necrotic material took place.

The patient continued to improve postoperatively and, 5 days post-surgery, his WCC was 12.7 × 10^9/l, with a CRP of 61.3 mg/l. Following microbiology advice, all antibiotics were stopped 7 days post-surgery as the WCC had normalised at 8.8 x 10^9, and his CRP came down to 38.6 mg/l. He eventually underwent skin grafting, under the care of the Plastic Surgeons, to his perineum, medial thigh and anterior abdomen.

No typical risk factors, typical for the development of Fourniers were identified on review of the patient's notes.

DISCUSSION

FG is a severe polymicrobial, infective necrotising fasciitis predominantly affecting the male perineal, perianal, genital and anterior abdominal wall regions.[1]

Given its eponymous name by Jean-Alfred Fournier in 1883 to describe a case of gangrene in the genitals and perineum of a previously healthy young male,[2] it is a devastating infection that can prove fatal if treatment is not initiated rapidly. Mortality rates range between 0 and 67%.[3]

Laor *et al.* developed a tool that enables the comparison of pathophysiological parameters likely to contribute to the disease process.[4] They suggest that those patients surviving the disease would often have a high hematocrit, serum calcium and albumin, and a lower blood urea nitrogen and lactate dehydrogenase levels. These parameters were suggested as

representing an underlying weakness in the host's ability to fight infection and, therefore, an inherent worsening of the effects of the infection.[4] Arising from this study was the Fournier's Gangrene Severity Index Score (FGSIS), which has been suggested as a useful tool in determining outcome following infection.[4-7] It is a numerical score based on a number of parameters, including vital signs (temperature, heart rate, respiratory rate) and clinical chemistry parameters (serum sodium, serum potassium, serum creatinine, hematocrit and WCCs).[4] However, Tuncel *et al.,* during their study of 20 patients, suggested that the FGSIS score does not predict the survival rate or the disease severity in those with Fourniers.[7]

FG, initially thought of as an idiopathic disease, has now been shown to have a higher incidence in those with other comorbidities. Diabetes, alcoholism, immunosuppression, liver disease and leukemia have all been shown to be risk factors for developing the disease.[8-12] Diabetes mellitus has been shown to exist in high proportions in those with FG,[9,11] while alcoholism has also been shown to be a poor predictor of survival.[12] Other predisposing factors include pelvic interventions (indwelling catheters, vasectomies, insertion of penile prosthesis, mucosal biopsy and hydrocele aspiration), anorectal (perianal abscess, appendicitis, hemorrhoidectomy, diverticulitis and circumcision), urogenital (urethral strictures, ureteric calculi, trauma) and perineal pathology.[13-15] Cem ozden *et al.* highlight other predisposing factors on survival rates, notably cardiac failure, hypertension and renal insufficiency. Interestingly, diabetes mellitus was not implicated.[5]

As already mentioned, FG is a polymicrobial infection. Both aerobic and anaerobic organisms have been implicated: *E. coli,* coliforms, kliebsiella, bacteroides, streptococci, enterococcus, pseudomonas, proteus sp. and clostridia,[3,4,8] with *E. coli* being the most prominent in the literature. Other organisms less commonly encountered include candida and *Lactobacillus gasseri*.[16-18] Yaghan *et al.* indicate that at least three different organisms are usually isolated in the culture of affected areas.[10]

The bacteria, normal commensals within the region, invade the tissue causing microthrombosis of the small subcutaneous vessels leading to ischaemia.[16] This leads to various cytotoxic agents (collagenases, streptokinases, etc.) being released, which cause progressive destruction of local tissue. An impaired host response, as mentioned above, contributes to the proliferation and subsequent gangrene of the overlying skin. Diabetes, with its microvascular and macrovascular complications, can be understood to cause a more severe form of infection and one that is heavily implicated in this disease.

FG is a disease process that, despite its original description, tends to affect the older age group,[3,4,19] and is more common in males than in females and young children.[4,5,13,20] Diagnosis is often made on clinical judgement. As with any necrotising skin infection, prompt recognition and treatment is essential. In our patient, the areas of necrosis spread rapidly (5 cm over 6 h). Other studies indicate a similar worrying spread of infection. Strikingly, our patient was quite comfortable, with the pain reducing at the point of diagnosis of the gangrenous areas. This is in contrast to other data in the literature, which indicate that FG is associated with significant pain and swelling. It is probable that the patient in our case was pain- free due to the antibiotics and analgesia received for almost 7 days. As with any infection, the myriad of symptoms include localised erythema and cellulits in conjunction with pain and swelling followed by a systemic upset, including fever, sweating and rigors. Crepitus, aided by gas-forming organisms, is often a feature and can, clinically, be elucidated quite easily.[4] Good knowledge of the local anatomy can point to the focus of infection, as the infection tends to spread along the line of the fascial planes.

The identification of FG takes place using sound clinical acumen and a high degree of suspicion. Nothing can replace or act sooner than a well-seasoned surgeon equipped to identify this serious infection. Radiography demonstrating surgical emphysema, ultrasonography (as used in our case early on) and computed tomography (CT) scanning modalities can be used.[21,22] CT scans are often the best modality, giving accurate assessment of disease extent as well as visualisation of the surrounding tissues where FG is likely to spread.[22,23]

The mainstay of treatment involves haemodynamic stabilisation, broad--spectrum antibiotics and surgical debridement. Hyperbaric oxygen has been postulated in the literature as adjuvant treatment, although this has not been proven in its efficacy.[13,19] Surgical debridement is paramount and, as in our patient, repeated surgical debridement may be necessary.[19,24] Our patient was started on "triple--therapy," which is described as the gold standard in the antimicrobial treatment in FG,[4,25] followed by urgent debridement of all necrotic areas. An orchidectomy was performed in our patient, indicating that FG had spread to the anterior abdominal wall.

Following the surgical debridement, the area can be left for regular surveillance prior to further covering. This will depend primarily on the deficit created following debridement and the depth of the tissue excised. It is important during the debridement process that thoughts about reconstruction do not cloud initial judgment. Correct surgical debridement saves lives. Our patient received meshed split--skin grafts (the donor site being the anterolateral aspect of the thigh), with a 100% subsequent take. Following his perineal reconstruction by the Plastic Surgeons, he made a complete recovery. This is

in keeping with studies indicating that although 50% of the individuals will have some residual pain post-recovery, most are satisfied with their eventual outcome.[25]

CONCLUSION

In conclusion, we have demonstrated a unique case of FG developing after the formation of an infected hydrocele. No aspiration was carried out. No convincing past medical history was identified as correlating to the typical aetiology associated with this devastating disease process. Diagnosis and treatment plans were instigated promptly, which resulted in halting of the rapidly spreading infection. Although no imaging modalities were employed in this case for diagnosis, a good surgical eye was paramount. Despite its rarity, all surgeons need to be aware of the clinical signs and to act promptly.

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