Case report

Localised necrosis of scrotum (Fournier's gangrene) in a spinal cord injury patient – a case report

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Abstract

Background: Men with spinal cord injury (SCI) appear to have a greater incidence of bacterial colonisation of genital skin as compared to neurologically normal controls. We report a male patient with paraplegia who developed rapidly progressive infection of scrotal skin, which resulted in localised necrosis of scrotum (Fournier's gangrene).

Case presentation: This male patient developed paraplegia at T-8 level 21 years ago at the age of fifteen years. He has been managing his bladder by wearing a penile sheath. He noticed redness and swelling on the right side of the scrotum, which rapidly progressed to become a black patch. A wound swab yielded growth of methicillin-resistant Staphylococcus aureus (MRSA). Necrotic tissue was excised. Culture of excised tissue grew MRSA. A follow-up wound swab yielded growth of MRSA and mixed anaerobes. The wound was treated with regular application of povidone-iodine spray. He made good progress, with the wound healing gradually.

Conclusion: It is likely that the presence of a condom catheter, increased skin moisture in the scrotum due to urine leakage, compromised personal hygiene, a neurogenic bowel and subtle dysfunction of the immune system contributed to colonisation, and then rapidly progressive infection in this patient. We believe that spinal cord injury patients and their carers should be made aware of possible increased susceptibility of SCI patients to opportunistic infections of the skin. Increased awareness will facilitate prompt recourse to medical advice, when early signs of infection are present.

Background

Men with spinal cord injury (SCI) appear to have a greater incidence of bacterial colonisation of genital skin as compared to neurologically normal controls. Differences in skin flora between the SCI patients and neurologically normal persons may be the result of variables such as an-
In addition to an increased incidence of bacterial colonisation of genital skin in SCI patients, SCI patients exhibit functional and morphological changes in the skin below the level of spinal cord lesion. These include clinical skin thickening, and histopathological findings of dermal fibrosis and perivascular inflammatory infiltrate. Denervation, loss of autonomic nervous system control, and other neuroendocrine dysfunction are suspected to be the causative factors in the pathogenesis of skin changes in SCI patients [2,3]. Spinal cord injury-related changes in the skin below the level of the lesion may contribute to the increased susceptibility of SCI patients to skin infection. Cellulitis is an important complication in the spinal cord injury patient [4]. Indeed, urinary and skin complications are the two main reasons for hospital readmission in people with chronic spinal cord injury [5].

Colonisation of perineum or urine with methicillin-resistant *Staphylococcus aureus* (MRSA) is common in spinal cord injury patients. Fortunately, infection of the genitourinary tract with MRSA is rare. When a SCI patient becomes infected with MRSA, the consequences can be serious. Another patient seen at this unit, a 61-year male sustained tetraplegia at C-5 level, when he fell down the stairs following a New Year’s eve party. He had an indwelling urethral catheter for drainage of neuropathic bladder. He developed severe degree of urethritis and marked swelling of penis. Pus from the penile urethra yielded a heavy growth of Methicillin-resistant *Staphylococcus aureus*.

**Case presentation**

This male patient developed paraplegia at T-8 level 21 years ago, at the age of fifteen years. He was lifting a pouffe above his head at a friend’s house, when he suddenly felt a pain in his back and developed slight weakness in the legs but the next morning, had a total paralysis with bladder and bowel dysfunction. He has been managing his bladder by wearing a penile sheath.

In February 2002, he developed infection under his left arm; a large swelling appeared which subsided with antibiotic treatment. Subsequently, he had infection of his gums followed by urinary infection. He noticed redness and swelling in right side of scrotum on 28 February 2002. The red area in the right side of scrotum became a localised black patch. This increased in size during the course of the next 48 hours. (Figure 2). He was feeling nauseated. The patient called his General Practitioner, who prescribed an antibiotic and referred him to the spinal unit. Examination revealed a black patch on the right side of scrotum. The surrounding area was red and oedematous.

Blood tests revealed elevated C-reactive protein (71.6 mg/l; reference range: 0.0 to 10.0). White cell count was $9.6 \times 10^9$/l. He was prescribed amoxicillin 500 mg every eight
hours for five days, and metronidazole 400 mg every eight hours for seven days. Ultrasound of scrotum, performed on 06 March 2002, demonstrated normal left testis and epididymis, with a normal skin appearance. (Figure 3). The right testis and epididymis were also normal, but the overlying skin was oedematous. (Figure 4). There was a small fluid collection in the subcutaneous tissue on the right side of scrotum. (Figure 5). The appearances were consistent with localised cellulitis. Wound swab culture on 07 March 2002 yielded growth of methicillin resistant

\textit{Staphylococcus aureus} resistant to flucloxacillin, penicillin and erythromycin. No other organism was grown. The necrotic tissue was excised on 12 March 2002. Culture of excised tissue yielded MRSA. No other organism was grown. Histology of excised tissue revealed full thickness coagulative necrosis with no viable skin remaining. (Figure 6). This histological finding is in contrast to the typical picture of Fournier's gangrene in which, necrotic dermis and subcutis are covered by intact viable epidermis. There were zones of polymorph infiltration; and occasional birefringent material was present, a few of which resembled hair shaft material. (Figure 7). Colonies of bacteria, both
cocci and bacilli, were present on the surface in places. (Figure 8). No vascular thrombosis was identified.

Wound swab, taken ten days later (22 March 2002), yielded growth of MRSA and mixed anaerobes. The patient was prescribed povidone-iodine spray for regular application over the wound. He was advised to use chlorhexidine ( Hibiscrub) cleansing solution instead of soap while showering. He was also advised to put his clothes and bed linen separately in the washing machine and not to mix his clothes with those of other family members. He made a good progress and the wound was healing gradually. (Figure 9).

Blood samples were taken for immunological tests. The samples were processed immediately. Measurements of serum immunoglobulins, haemolytic complement activity (CH-100) and neutrophil oxidative function were carried out in the immunology laboratory of Royal Liverpool University Hospital, Liverpool, England. The results of immunological tests were as follow:

- Immunoglobulin G: 10.10 g/l (6.00 – 16.00)
- Immunoglobulin A: 0.81 g/l (0.83 – 4.00)
- Immunoglobulin M: 0.86 g/l (0.45 – 2.10)
- Complement haemolytic screen: 1611 units (Reference range: 300 – 770)
- Neutrophil oxidative function by flow cytometry showed a bimodal population following stimulation of
neutrophils. The reason was unclear. However, the presence of at least some cells with an apparently normal respiratory burst effectively excluded chronic granulomatous disease (CGD) or neutrophil NADPH deficiency.

Discussion
Our patient developed skin infection of the scrotum, which progressed rapidly, resulting in necrosis of a significant area of scrotum. Understandably, this was a frightening experience to our patient. The region where he developed the black patch in the scrotum (Figure 2) was not an area commonly prone to pressure sores, in contrast to sacrum, ischial tuberosity, or greater trochanter, for example. Our patient did not have any skin lesion in the scrotum prior to the development of the necrotic patch. It is possible that decreased natural and adaptive immune responses, reported in the patients with spinal cord injury, may have played a role in the development of localised necrosis of scrotum in this patient. The altered immune responses specifically include the following:

- Sustained loss of NK cell function in the circulating lymphocyte population
- Transient reduction in in vitro T-lymphocyte transformation (mirroring a transient rise in urinary free cortisol)
- Decreased expression of adhesion molecules on lymphocytes and granulocytes
- A functional decrease in adhesion molecule-dependent binding of endothelial cells by patients’ lymphocytes [6].

Depressed immunity and impaired proliferation of haematopoietic progenitor cells in the patients with spinal cord injury may be explained by the fact that the innervation of the bone marrow below the injury lacked normal supraspinal activity, that is, a decentralised bone marrow [7]. Impaired status of the immunological system in spinal cord injury patients may contribute to the increased susceptibility of SCI patients to opportunistic infections of the urinary tract, lungs and skin, which are the major causes of morbidity in survivors of tetraplegia [8].

We do not know the reason for the occurrence of multiple infections – soft tissue infection in the left axilla, gingival infection, urinary infection, and then skin infection of scrotum – in our patient, within a short time. Only limited immunological tests were performed in this patient. The results of these tests ruled out major immunological disease such as chronic granulomatous disease (or neutrophil NADPH oxidase deficiency), but diagnosis of subtle immune dysfunction will require more sophisticated tests.

We used povidone-iodine spray to treat scrotal infection in our patient. In an in vitro study, Michael and Zach [9] demonstrated superior efficacy of a povidone-iodine preparation for local treatment of MRSA infection. In spinal cord injury patients with features of systemic sepsis due to MRSA, our antibiotic of choice is teicoplanin. The SCI patient whose clinical photograph is shown in Figure 1 received teicoplanin and Tazocin (Piperacillin and tazobactam) since pus culture yielded MRSA and urine culture grew Pseudomonas aeruginosa and MRSA. We also use teicoplanin as a prophylactic antibiotic in those SCI patients who have colonisation of urine with MRSA, before carrying out a urological endoscopic procedure such electrohydraulic lithotripsy of bladder stones and bladder biopsy.

Conclusion
We report a male patient with paraplegia, who developed rapidly progressive infection of scrotal skin with MRSA and anaerobes, which resulted in localised necrosis of the scrotum. It is likely that the presence of a condom catheter, increased skin moisture in the scrotum due to urine leakage, compromised personal hygiene, a neurogenic bowel, and possible subtle immune dysfunction, contributed to colonisation and then rapidly progressive infection with MRSA and anaerobes. We believe that spinal cord injury patients and their carers should be made aware of possible increased susceptibility of SCI patients to opportunistic infections of the skin, urinary tract and lungs. Increased awareness will facilitate prompt recourse to medical advice by patients and their carers, when early signs of infection are present.

Competing interests
None declared.

Authors’ Contributions
SV managed the patient in the clinic, arranged various investigations, performed excision of necrotic skin, and wrote the manuscript. PM interpreted histology slides of excised skin. JD supervised the immunological tests and provided expert advice. All authors contributed to the final version of the manuscript.

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