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Necrotizing Soft Tissue Infection: An Unusual and Devastating Complication of Pressure Sores

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Necrotizing soft tissue infections constitute a spectrum of devastating entities that despite rapid diagnosis and aggressive surgical treatment carry high mortality rates. Pressure sores are frequently encountered in clinical practice in bedridden patients. Although pressure sores could be deemed a proper environment for the development of NSTI, this association has been rarely reported in the English medical literature. To increase awareness of this association we present the case of a patient who developed NSTI as a complication of pressure sores. We describe the clinical course and the aggressive surgical treatment with its devastating consequences, and review the scarce available literature.

PATIENT DESCRIPTION

A 50 year old woman with a history of spinal cord injury, paraplegia and diabetes mellitus type 2 was admitted with fever. A few months before her admission she developed ischial pressure sores.

On physical examination she was febrile and hypotensive and three deep pressure sores (stages 3 and 4) were observed over the ischial areas and sacrum, one of which had a purulent discharge without surrounding erythema. Thorough evaluation by a plastic surgeon revealed chronic clean pressure sores that were less likely to be the source of the current infection.

She was admitted to the medical ward and treatment with ceftriaxone was initiated. Three days after admission, *Escherichia coli* was isolated on urine culture and blood cultures revealed growth of gram-negative rods and streptococci. Antibiotic treatment was changed to include intravenous gentamicin and ampicillin – against both types of bacteria, respectively, and chloramphenicol for anaerobic coverage. Defervescence occurred after 48 hours. On surgical consultation, the pressure sores were deemed clean, thus additional tests were ordered to reveal the source of infection. Abdominal ultrasound, transesophageal echocardiogram and bone scintigraphy scan were unrevealing. On the sixth day of hospitalization a new spike of fever was noticed and abdominopelvic computed tomography scan revealed edema and gas over the right ischial and gluteal areas and around the right hip joint, and signs suggesting osteomyelitis of the femoral head and ischium. A few hours later, erythema and crepitus appeared around the right ischial sore extending to the lateral aspect of her thigh.

Surgical exploration revealed an extensive necrotizing process involving the rectus femoralis, sartorius, vastus lateralis, gluteus medius, ileopsoas muscles and hip joint. An immediate disarticulation of the right hip was conducted leaving a large open wound with clean margins, which was protected from fecal spoilage by a diverting colostomy. Cultures were not taken during the operation. She was transferred to an intensive care unit and required mechanical ventilation support due to acute respiratory distress syndrome. The bacteria in her blood cultures were finally identified as *Streptococcus constellatus*, *Streptococcus gordonii* and *Bacteroides* species, susceptible to the antimicrobial agents used empirically. Pathological specimens revealed acute osteomyelitis of femoral head and neck and soft tissue necrosis with bacterial seeding.

The patient was discharged home 4 months later with a clean and partially closed surgical wound, tracheotomy and colostomy. Two additional hospitalizations culminated with her death 6 months later due to septic shock unrelated to the soft tissue infection.

COMMENT

This case of necrotizing soft tissue infection demonstrates the severe consequences of pressure sores and the difficulty in diagnosing and treating these infections. Necrotizing soft tissue infections constitute a spectrum of diseases characterized by rapidly progressive necrosis of soft tissues. In 1952 the term "necrotizing fasciitis" was popularized. Today NSTI is the preferred term since there is little utility in distinguishing its subcategories (e.g., Fournier gangrene, myonecrosis, necrotizing fasciitis), all of which have common features; namely extensive necrosis of subcutaneous and deeper tissues and need of urgent recognition, with a high average mortality rate

NSTI = necrotizing soft tissue infections

Table 1. Clinical features of patients with necrotizing soft tissue infection complicating pressure sores

Reference	Age/ Gender	Co-morbidities	Decubitus ulcers	Necrotizing tissue extension	Cultures results	Outcome
4	75F	Femoral neck fracture, diabetes mellitus type II, rheumatoid arthritis, IHD, CVA	Greater trochanter pressure ulcer stage IV and small sacral wounds	Fascial planes of the distal thigh	*Escherichia coli, Staphylococcus aureus, Streptococcus* group D	Alive, 21 operations, debilitated
4	59F	Diabetes mellitus type I, CVA	Sacral ulcer covered with eschar and draining pus	Rectum and along the thigh	*Streptococcus* group A	Alive, 6 operations, debilitated and non-verbal
5	62M	Spinal cord injury	Deep chronic sacral wound	Paraspinal muscles, spinal cord, retroperitoneal space and abdominal wall	*Clostrodium* sp., *Enterococcus* sp., *Prevotella loescheii*	Death
Present case	50F	Spinal cord injury, diabetes mellitus type II	Sacral and ischial pressure sores stages III-IV	Rectus, gluteus and iliopsoas muscles, and hip joint	*Strep. constelatus, Strep. gordonii, Bacteroides* sp.	Discharged after 4 mos Death after 6 mos

F = female, M = male, IHD = ischemic heart disease, s/p = status post, CVA = cerebrovascular accident

of 30% [1,2]. The case described here may be categorized as a synergistic mixed soft tissue infection, originating from pressure sores, as proven by the tomography and the surgical exploration. Rapid diagnosis and aggressive debridement are the mainstays of treatment.

In our case the presence of concomitant bacteriuria delayed the diagnosis of NSTI. Even expert opinion based on repeated physical examination failed to detect the underlying infectious process, highlighting the importance of early radiological evaluation. Thus a high index of suspicion is needed to diagnose NSTI, since there might be relative sparing of the overlying skin, as demonstrated in our case where the radiological findings preceded the clinical ones.

NSTI related to decubitus ulcers is very rare, and to date the English medical literature contains only two reports [3,4], describing three patients [Table]. Interestingly, three of the four patients (including the present case) were diabetic. We therefore speculate that diabetes played a central role in the development of NSTI, through multiple possible mechanisms, particularly immunological disturbances that involve polymorphonuclear leukocytes, diabetic neuropathy and ischemia. These factors may contribute to the rapid progression of the infection over hours or days.

The bacteria involved in these cases are predominantly aerobic gram-positive cocci, along with *Enterobacteriacea* and anaerobes, the same pathogens colonizing chronic pressure wounds. In three of the four patients, more than one pathogen was identified in cultures, highlighting the importance of broad antibiotic coverage in these infections, including antimicrobial agents covering anaerobic bacteria. No data are available on the appearance of the pressure sores in the cited three cases.

Although the mechanism of progression from chronic colonized wound to aggressive infection is unknown, it may be influenced by the efficacy of local wound care, bacterial inoculum and host defenses. A preliminary report suggests that patients with decubitus ulcers have reduced adhesion molecule expression and impaired cell-to-cell interaction [5] which makes them prone to invasive infections. Measures to prevent decubitus ulcers in chronic immobilized patients include early mobilization, pressure relief, enteral nutrition and careful examination for early diagnosis. When ulcer develops, early local therapy, early identification of infection and – when suspected – imaging for early diagnosis and treatment, is in order. These may prevent the devastating consequences of necrotizing invasive tissue infections.

The presence of multiple organisms in blood cultures could be a clue for underlying NSTI even before a final identification of each pathogen. We believe that NSTI related to pressure sores are under-diagnosed or under-reported and that additional reports are needed to improve the detection and management of these infections.

In conclusion, necrotizing soft tissue infection related to pressure ulcers is rarely reported, has a progressive and unpredictable course requiring aggressive surgical treatment, and carries devastating consequences such as severe disfiguring and death.

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