An Abnormal Presentation of a Delayed Necrotizing Soft Tissue Infection Secondary to Spinal Hardware Malfunction: A Surgical Case Report

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Abstract:

Necrotizing Soft Tissue Infections (NSTI) are characterized by “flesh-eating bacteria” that are rapidly progressive and requires immediate diagnosis and aggressive surgical debridement with broad-spectrum antibiotics. Common pathogens such as, Streptococcus Pyrogens, Staphylococcus Aureus, Enterococcus Coli, and Clostridium are the usual culprits of this disease. While NSTI have a relatively low incidence, they're associated with higher rates of morbidity and mortality. Due to the aggressive nature of the pathogen, NSTI can cause widespread necrosis of soft tissue and muscle leading to extensive surgical intervention and patient disfigurement. The patient population that is most susceptible to NSTI include the elderly, immunocompromised, and diabetics. In this case report, the patient is a 54-year-old female with past medical history of type 2 diabetes mellitus, Hepatitis C, an extensive spinal surgery with malfunctioning spinal hardware who developed an NSTI while in the Intensive Care Unit.

Introduction:

NSTI is characterized by infiltration of bacteria into the subcutaneous tissue, superficial fascia, and muscle causing widespread necrosis [1, 2]. NSTI serves as an umbrella term for wide range of pathogens that can cause necrosis of soft tissue and muscle. These types of infections can occur in different anatomical regions within the body which include but not limited to the orbital, cervical, upper and lower extremity, thoraco-abdominal, and perineal/genital region [1, 2]. Usually, the breakdown in the skin barrier can introduce these harmful bacteria into the body. In 10%-30% of NSTI cases, local trauma was indicated as the portal of entry [1, 2, 3]. Some examples include minor skin abrasions, post-operative surgical wounds, and insect bites where all noted as potential site of entry. Surgical site infections (SSI), of deep spinal instruments incidence rate account for 2.25% -20% due to inability of complete removal of hardware without causing spinal destabilization [4, 5].

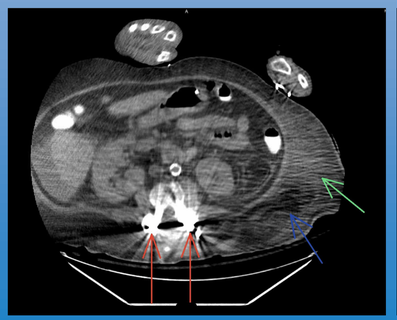


Figure #1: Axial view at level of spine CT scan of the abdomen/pelvis shows spinal hardware implants (red arrows), subcutaneous emphysema (blue arrow), and subcutaneous fat standing (green arrow)

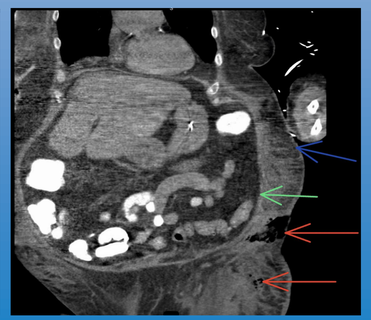


Figure #2: Coronal view shows subcutaneous emphysema in lower lateral abdomen and superior thigh (red arrow), subcutaneous fat stranding (blue arrow), peritoneal cavity (green arrow)

Case Report:

The patient is a 55-year-old female with past medical history of uncontrolled diabetes mellitus, urinary tract infections, spinal surgery with metal hardware implants brought by emergence medical services (EMS) to the emergence department (ED) due to elevated glucose levels, altered mental status, and tachycardia. Patient was then transferred to the intensive care unit (ICU) for further monitoring. In the ICU, the patient's altered mental status continued to deteriorate, requiring intubating and mechanical ventilation for airway protection as Glasgow coma scale (GCS) was 8. However, she constantly kept spiking fever with unidentified source of infection. CT scan revealed subcutaneous emphysema seen at the level of the left lower quadrant of the abdomen extending into the left upper thigh [Figure *1 and 2*]. Patients LRINEC was < 6 which indicates low suspicion for NSTI, however, does not rule out diagnosis of NSTI and may be required further imaging to confirm.

General surgery was consulted, and patient was transferred to the operating room for debridement. In the OR, upon incision necrotic soft tissue was visualized along with foul smelling dishwater fluid. Necrotic tissue extended down to external oblique, inferior into the lower portion of thigh, laterally then medially into the lumbar spine extending up the spinal column. Wound size was 30 x 30 x 8 cm. Further debridement was deferred due to the extensive involvement of the lumbar spine and patient deteriorating hemodynamic instability. Patient was unable to tolerate placement in prone position and it was decided to wait 24 hours until next debridement.

On thorough chart review, the radiology report of the patient's CT lumbar spine taken a month prior during hospitalization at another facility showed, extensive post-surgical changes with multiple posterior fusion rods and fixated screws. Unfortunately, the patients CT imaging was unable to be obtain from her previous hospital admissions, only the radiologist report was available. Per report, which depicted "fractures of rods of the right lumbar spine at the level of L1 and L4-L5, and on the left at the level of L2 and L5-S1. One of the left fixation rods extends posteriorly and laterally into the soft tissues with a surrounding fluid collection containing gas. There was lucency surrounding the base of the left iliac screw compatible with hardware loosening". Given the patient's presentation and her past surgical history, it was decided to transfer the patient to a tertiary facility for further management. Wound cultures taken during surgical procedure came back positive for non-hemolytic streptococcus and anaerobic negative rods.

Reference:

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Discussion:

In this cases report, the patient had a past medical history of spinal hardware implants. Two weeks prior to her admissions, an outpatient CT scan of the lumbar spine with contrast done at a neighboring hospital showed large fluid collection within the left lower back beginning at L2-L3 extending inferiorly into the upper buttocks, containing gas surrounding the displaced hardware rod. Given the patients clinical presentation, one can concur that the initial site of infection began with the dislodgement of the spinal hardware into the surrounding soft tissue spreading necrotizing bacteria. Another can propose that the management of this patient based on her initial CT scan would be to undergo immediate surgical debridement with broad-spectrum antibiotics and removal of spinal hardware, or a CT guided fluid aspiration with culture. It is also important to note the patients past medical history of uncontrollable type 2 diabetes mellitus could have contributed to her developing a NSTI secondary to spinal hardware malfunction.

During the patient ICU admissions for severe diabetic ketoacidosis (DKA), her LRINEC was < 6 suggesting relatively low probability of NSTI. However, a low LRINEC score does not rule out the chances of developing Necrotizing Fasciitis. Her repeated CT scan showed an extensive accumulation of subcutaneous gas extending from the left back, left flank, lateral portion of abdomen descending into the left thigh indicating Necrotizing Fasciitis. NF spreads along the thoracolumbar fascia which consist of two anatomical layers, that is the superficial layer (continuous with the aponeurosis fascia of the latissimus dorsi muscle) and the deep layer (continues towards the fibrotic bands of the lumbar spine) [9]. This intricate system allows for rapid colonization of fascia. Treatment of NF usually requires multiple rounds of surgical debridement and continuous antibiotics [9].

Conclusion:

The development of NSTI secondary to spinal hardware malfunction is a life-threatening infection. While NSTI are associated with the infiltration of necrotizing bacteria into the subcutaneous tissue and deep fascia, prosthetic devices implanted into the body carry an increases risk of developing SSI. Bacteria that can form biofilms proposes a difficult task to prevent infections. Early recognition of NSTI is imperative to provide effective surgical management to prevent excessive loss of vital tissue. Patients with concerning co-morbidities should be followed carefully to rule out the development of NSTI in a critical care setting. Ordering additional imaging on critically ill patients with known co-morbidities or spinal hardware malfunction could be the first step in diagnosing the formation of NSTI. Patient with NSTI should be aggressively treated with multiple rounds of surgical debridement and broad-spectrum antibiotics.