

Asian Journal of Case Reports in Surgery

Volume 7, Issue 1, Page 48-52, 2024; Article no.AJCRS.112275

Necrotising Soft Tissue Infection (NSTI), an Atypical Complication of Pressure Ulcer: A Case Report

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Authors' contributions

This work was carried out in collaboration among all authors. All authors read and approved the final manuscript.

Article Information

Open Peer Review History:

This journal follows the Advanced Open Peer Review policy. Identity of the Reviewers, Editor(s) and additional Reviewers, peer review comments, different versions of the manuscript, comments of the editors, etc are available here:

https://www.sdiarticle5.com/review-history/112275

Case Study

Received: 27/11/2023 Accepted: 03/02/2024 Published: 12/02/2024

ABSTRACT

Introduction: Infected pressure sore, also known as bedsore or pressure ulcer is a very common condition presented to hospitals especially in elderly and bedbound patients with reported incidence rate of 12%. Managing infected bedsore is challenging and involve multidisciplinary care. Unfortunately, despite best possible efforts, infected pressure sore may progress into necrotising soft tissue infection (NSTI).

Case Report: A 73-year-old lady presented to emergency department with infected pressure sore which turned out to be NSTI at gluteal region. She was treated aggressively with broad spectrum antibiotic and underwent multiple surgical debridement.

Conclusion: NSTI arising from pressure sores is rare but has high morbidity and mortality. Early diagnosis and treatment is crucial in handling such cases.

Keywords: Pressure; sore; ulcer; necrotising fasciitis.

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1. INTRODUCTION

Pressure sores are wounds over the skin and underlying tissues resulting from prolonged pressure over the affected area which usually affect those who are immobile for long periods or bedridden patients. This prolonged pressure which are higher than capillary pressure leads to localized soft tissue ischemic necrosis [1]. In time, if left untreated, the sores may grow large and lead to infections.

The reported incidence rate of pressure sores is 12% [2]. According to Malaysian Registry of Intensive Care Report 2017, the rate of pressure ulcer following the admission to Intensive Care Unit (ICU) was 4.9 per 1000 ICU days [3]. Pressure sore are classified into four grades based on National Pressure Ulcer Advisory Panel (NPUAP) system as per Table 1.

Necrotizing soft tissue infection (NSTI) previously known as necrotising fasciitis, is a type of life threatening, aggressive skin and soft tissue infection that causes necrosis of the muscle fascia and subcutaneous tissues. The rapid infection spreads initially along fascia plane which may then cause secondary infection of the overlying and underlying skin, soft tissue and muscle [4].

NSTI rarely developed from pressure sore. Whether it is really rare or under-reported is not known. Herein we present such case in which pressure bedsore from Immobilisation leads to infected bedsore and progress into NSTI.

2. CASE REPORT

A 73 year-old woman with underlying diabetes mellitus, hypertension and Parkinson's disease presented to emergency department with painful gluteal sore with foul smelling pus discharge for the past 1 month. She was treated for bilateral lower limbs cellulitis 2 month ago and was bedridden since then. Physical examination revealed gluteal sore measuring 10 x 15 cm with necrotic patch (Fig. 1A).

Blood investigations shows leucocytosis with white cell count (WCC) of 27 x10⁶ with high C-reactive protein (CRP) of 109.17. Broad spectrum antibiotics was initiated and early surgical wound debridement performed. Intraoperatively, necrotic tissue extend beyond the gluteal fascia with presence of fishy-odour smell. Left gluteal maximus muscle was non-viable and left gluteal medius appeared flaccid. Extensive wound debridement was done (Fig. 1B). Tissue culture growth *Escherichia coli (E. coli)* and *Proteus mirabilis*. Antibiotic then tailored according to sensitivity.

Second wound debridement was performed on Day 12 admission due to persistent slough with necrotic tissue (Fig. 1C and 1D). Third wound debridement was done on Day 22 due to foul smelling discharge with necrotic tissue over superior medial and superior lateral quadrant of the wound despite daily modern dressing (Fig. 1E, 1F and 1G). Post third wound debridement, wound appeared healthy with no pus discharge (Fig. 1H). She was discharged home with daily dressing at nearest clinic. Wound inspection at surgical outpatient clinic showed healthy granulation tissue with no slough or discharge (Fig. 1I).

Table 1. Gradation based on National Pressure Ulcer Advisory Panel (NPUAP) system [4]

Grade	Features
Grade I	Pressure sore with intact epidermis and non-blanchable redness over localised area, commonly over bony prominent
Grade II	Pressure sore involving partial thickness loss of dermis with red wound bed and without slough
Grade III	Pressure sore involving full thickness tissue loss. Bone, tendon or muscle are not exposed. Slough may present.
Grade IV	Pressure sore involving full thickness tissue loss with exposed bone, tendon or muscle. Exposed bone or muscle is visible and can be palpable. Slough may present.



Fig. 1. A case of gluteal sore progression to NSTI

(A) An infected gluteal sore with necrotic patch. (B) Finding intraoperatively showed necrosis tissue extending to fascia. (C) The patient's wound after a week of daily wound dressing. (D) Wound before second wound debridement shows slough with pocket. (E) The wound shown after second re-wound debridement, prior to third wound debridement. (F) Wound before third surgical debridement with necrotic and unhealthy tissue. (G) Wound prior third wound debridement. (H) Wound prior discharge shows clean, pink tissue. (I) Wound during follow-up

3. DISCUSSION

Population with conditions such as diabetes, malignancies, obesity, alcoholics and postsurgical infections are at higher risk of developing NSTI. Most common risk factor is diabetes mellitus which account for 40 to 60% [5]. Patient with diabetes are more susceptible to infections as higher glucose level gives an optimum medium for bacterial growth especially in low oxygen tension environment.

NSTI itself is a rare condition with an incidence rate ranges from 0.3-15 cases per 100,000 population [6]. A report has concluded that NSTI secondary to pressure sore is even uncommon.[7] Patient with grade III and IV pressure sore are at higher risk to develop NSTI.[8] However, the aetiology of pressure

sores evolve to necrotising fasciitis is yet, unclear.

Pressure ulcers only limits to pressured area which cause localized soft tissue ischemic necrosis meanwhile NSTI is differentiated by the extension of the infection. In pressure sores that develop into NSTI, the extension of infection is beyond the area of bony prominence and does not limited to the primary necrotic tissue caused by pressure induced ischemia.

Diagnostic criteria for infected pressure sore progressing to NSTI are not well established. The diagnosis can be determined based on presence of necrotic fascia and fat tissue. In addition, diagnosis can be made based on evidence found through direct observation during surgical debridement or by pathological findings or through evidence of presence of gas within

soft tissue adjacent to pressure ulcer in computed tomography [9]. Intraoperatively, fascia and muscle of a NSTI appear grey-blackish while infection pressure ulcers only display yellowish debris known as slough [7].

The Laboratory Risk Indicator for Necrotizing Fasciitis (LRINEC) scoring system was developed to differentiate NSTI from the other severe soft tissue infections. Yet, the sensitivity of this scoring system ranged from 43% to 80% in different studies which may limit it use in clinical setting.

The principle of management of NSTI and a high grade infected pressure sore are the same. Prompt, aggressive wound debridement with early initiation of broad spectrum antibiotic are crucial. Surgical debridement is required to remove dead tissue and control the infection. In most of the cases, multiple surgeries are needed to fully control the wide extension of infection and to remove all of the dead tissue [9].

In our study, diagnosis of NSTI was made through direct observation through surgical wound debridement as evidence by extension of necrotic tissue beyond the fascia. Our case was managed with a total of 3 extensive surgical wound debridement and 21 days course of intravenous antibiotics before being discharged home. At the same time, the patient was also by dietitian, physiotherapy, reviewed occupational therapist, general medicine specialist and other relevant team to optimise healing and prevent secondary infection.

Prevention of bedsore is of paramount importance. Few common bedsore prevention modalities that should be strictly applied include frequent repositioning, application of topical prophylaxis and use of special foam mattress and alternating pressure mattress. Frequent switching position helps relieve the constant pressure over susceptible areas. Applying topical prophylaxis such as cavilon spray or cream, zinc oxide and aqueous cream act as moisturiser to the skin which help to reduce development of pressure sores [10]. Ripple mattress, commonly used in bedridden patients in local setting helps to spread pressure over bigger surface area and minimize the pressure over bony prominence [11]. However, the most essential step in bedsore prevention is daily inspection over pressure sore areas as infected pressure sores often results from neglected wound. Pressure

sores grade one should be an alarming note for a more strict preventive measures [12].

4. CONCLUSION

In conclusion, NSTI develop from pressure sore is a rare condition with high morbidity and mortality. Therefore, early diagnosis of paramount importance. treatment is Aggressive surgical debridement, with early initiation of antibiotic are the gold standard in managing NSTI. As NSTI from pressure sores are more common to develop in a higher grade sores, care need to be taken seriously to prevent development of pressure sore in the first place. As the saying goes, prevention is always better than cure.

CONSENT

As per international standards or university standards, patient(s) written consent has been collected and preserved by the author(s).

ETHICAL APPROVAL

As per international standards or university standards written ethical approval has been collected and preserved by the author(s).

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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Peer-review history:
The peer review history for this paper can be accessed here:
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