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Case Report

Necrotizing soft tissue infection at an uncommon site following tooth extraction

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ABSTRACT

Necrotizing soft tissue infection (NSTI) is a disease characterized by rapidly progressive necrosis involving skin, subcutaneous tissue, fascia or muscle. It is commonly seen in extremities, perineum and lower abdomen. It is extremely uncommon for necrotizing fasciitis to occur along the chest wall, with only a few such cases being reported. We report one such rare case of NSTI in a 75 year old male who underwent left molar tooth extraction developed Ludwig's angina which progressed to present as a painful swelling over the left side of the neck and chest with a necrotic patch of skin over manubrium sternum managed successfully.

Keywords: Necrotizing soft tissue infection, Tooth extraction, Ludwig's angina

INTRODUCTION

Carrying an inherently high mortality rate between, 30% and 76%, when occurring in other regions of the body, NSTI of the chest wall carries with it a particularly poor prognosis.[1] Early diagnosis and treatment are the two main factors responsible for good prognosis and requires special management. Early commencement of broad spectrum antibiotics, debridement to prevent life threatening sepsis and delayed surgical reconstruction helps in rapid recovery.[2]

This is a report containing a description of a case of NSTI of chest wall.

CASE REPORT

A 75 year old male who underwent left molar tooth extraction developed Ludwig's angina which progressed to present as painful swelling over the left side of the neck and chest with a necrotic patch of skin over the manubrium sternum. On palpation, tenderness and crepitus were noted. Patient was febrile with tachycardia.

Chest radiography showed subcutaneous gas.

Figure 1: Representing necrotic patch in chest wall.

Figure 2: Representing pus drained from the chest wall.

Ultrasonography showed subcutaneous oedema of the involved site with emphysematous changes.

It was diagnosed as NSTI. Patient underwent multiple debridements.

Pus culture showed poly-microbial growth repeatedly.

Figure 3: Representing extensive debridement.

Once he was clinically stable he underwent meshed split skin grafting. Post op period uneventful.

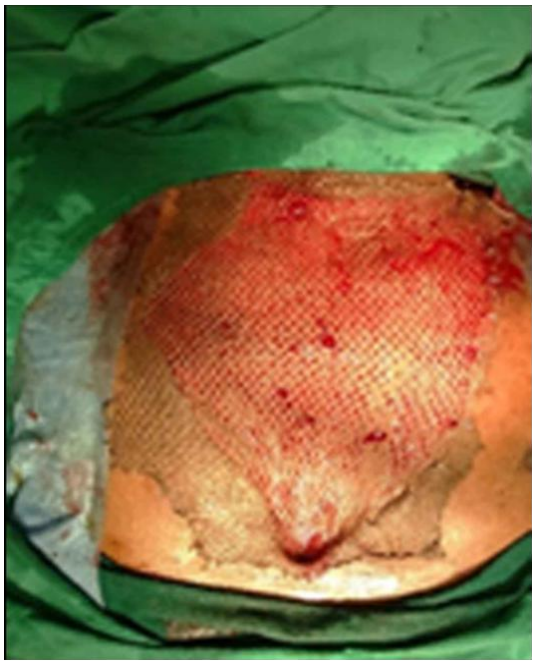

Figure 4: Meshed skin graft placed over the debrided area.

DISCUSSION

NSTI is a derma‑hypodermitis affecting the soft tissue and muscular fascia. An uncommon infection caused by microorganisms called 'flesh eating bacteria, mainly represented by group A beta hemolytic streptococcus. NSTI remains a life threatening condition occurring most commonly in immuno compromised patient (diabetics, alcoholics, immunosuppressed patients), in drug users, and in patients with peripheral vascular disease, although it can also occur in young, previously healthy individuals.[3]

Its location on the chest wall is extremely rare.[1,2] In a large series of 166 patients with, NSTI, the extremities were the most common site of infection (57.8%), followed by abdomen (12.1%), perineum (12.1%), buttocks (10.2%), head and neck (8%), and chest (5%).[3]

These infections are often multi microbial, although a single organism may be responsible in as many as 77% of infected patients.[1] When a single organism is responsible, group A streptococci and clostridium perfringens have been among the most frequently isolated pathogens.[4] Mono microbial infections often present in a fulminate fashion, with systemic toxicity highlighted by fever, leukocytosis and occasionally shock. In contrast, poly microbial infections may present in a more insidious manner as in our case.

These infections are caused by synergy among anaerobes and facultative aerobes, involving both gram negative and gram positive organisms, and can affect any and all layers of soft tissue, including skin, fat, fascia, and muscle.[4]

Severe necrotizing soft tissue infections involving the upper body and chest wall, however, are distinctly uncommon, thoracic location is most frequently reported after chest drainage or after thoracic surgery.[5] However, contagions from empyema have been reported.[1,2,6]

The characteristics of NSTI in the chest wall adjacent to the axilla are quite different from other sites; axillary NSTI may be recorded as chest wall NSTI.[7] Clinical features of chest wall necrotizing soft tissue infections are similar to those seen at other sites: Wound pain, skin blistering, crepitus, foul smelling watery discharge and dramatic deterioration in the patient's condition.[1]

As there are only few early symptoms (cutaneous manifestations appear late), diagnosis is difficult and may require blood culture, wound culture, and radiographic imaging.[1]

Because of the delay in diagnosis and inadequate debridement, prognosis of chest wall NSTI has been highly lethal.[7] It appears that factors other than bacteriology can account for the extremely high fatality rate in chest wall NSTI. Most of the patients reported to have died with the condition had chronic predisposing conditions, and more than 80% experienced significant diagnostic delays before treatment was initiated.[1]

Antibiotic treatment must be started immediately, even before results of the microbiological analyses.[5] Antibiotics are usually adjuvant to the surgical treatment because the local vascular thrombosis results in poor antibiotic tissue diffusion.[5] The objective of the antibiotic treatment is to limit the progression of the infection.[5] The recommended antibiotic treatment consists in the association of beta-lactam, imidazole, and ± aminoglycosides.[5]

Treatment of NSTI entails a radical excisional debridement.[7] The extent of debridement has been a topic of debate.[7] Sarani et al recommended that the excision boundaries in NSTI should be at least as wide as the rim of cellulitis.[8]

Wong et al proposed a different approach to debridement in NSTI, involving the classification of the affected area into the three surgical zones: (i) Necrotic tissue, which is completely excised; (ii) infected but potentially salvageable soft tissue, which is carefully assessed and progressively cut back; and (iii) non infected skin, which is left alone.[9]

Wound closure has been difficult in patients who survived the initial septic phase.[2] Literatures tell us, for the reconstruction of defect after NSTI follow the step ladder pattern of repair.

In our case, we chose extensive debridement and split skin grafting to close the debrided wound.

CONCLUSION

The NSTI progresses to a general septic appearance and spreads more through muscle and subcutaneous cellular tissue. It may lead to fatal outcome not only because of its severity, but also delay in diagnosing it during the early stages due to the paucity of skin findings early in the disease. Early start of broad spectrum antibiotics with aggressive debridement can save the patient.

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