rare case of four limbs necrotizing fasciitis.

Albisher Nesreen

Abstract— Necrotizing soft tissue infections is one of the most difficult and sever disease processes that is encountered by physicians and surgeons. This disease require an immediate and aggressive surgical management. According to literature review there have been very few reports of necrotizing fasciitis involving all the four limbs. Here, we report a rare case of four limbs necrotizing fasciitis.

Index Terms— Necrotizing soft tissue infections, Necrotizing fasciitis , Four limbs

1 Introduction

Necrotizing soft tissue infections (NSTIs) is one of the most difficult and sever disease processes that is encountered by physicians and surgeons. NSTIs can arise primarily in the dermis and epidermis, but they more commonly affect the deeper layers of adipose tissue, fascia, or muscle. NSTIs have a very rapid progression course with significant local tissue destruction. Early diagnosis and treatment are crucial for survival. The Diagnosis maybe delayed as the disease progresses below the surface. Physicians must maintain a high index of suspicion to rapidly diagnose NSTIs. The standard treatment consists of broad-spectrum antibiotics, wide surgical debridement, and supportive care. Most patients require multiple surgical debridement, and survivors often have large and complex wounds requiring soft tissue coverage and prolonged hospitalizations. Even with optimal treatment, NSTIs have a very high risk of morbidity and have mortality rates of approximately 25%-35% .

Her we present a case of four limb necrotizing fasciitis in otherwise known healthy man

2 Case report

37 YO Sudanese male medically free work as a Sheperd , occasional smoker with no known comorbidities. Came to Saudi Arabia 2 month back. He was referred to the hospital as a case of aplastic anemia where he was transfused there 1 unit of platelet which was +ve cocci.

10 month ago he started complaining of headache, fatigue, SOB and palpation on exertion, on and off hematuria, epistaxis, cough, night sweet, diffuse mild abdominal pain without chest pain. In the last 2 months he started having Left upper limb, Left lower limb painful swelling with subjective fever with blister in Left lower limb after 8 days. Without any history of previous trauma.

On examination, Middle aged man, well built, not in pain or respiratory distress. V/S: temp 37.7 C, P 119, BP 120/55 mmHg, RR 20 breath/minute. Left upper limb: swelling in the arm and forearm, distal pulse is intact, no tenderness with restriction in the ROM in the finger. Left lower limb: swelling reaching above the knee with tenderness to touch. Blister and bullae in the dorsum of the foot and Punctured bullae in the anterolateral foot, anterolateral surface of the tibia showed necrotic area about 20*10cm and the .the distal pulses was sluggish posterior tibial artery and un-palpable dorsalis pedis. Stretch test of the toe was negative, cap. Refill normal, intact sensation. Left knee: was swollen, NO ROM can be elicited and weight bearing was impossible. Right leg was mildly tender and tense, ROM are intact in the ankle and the knee.

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Chest examination showed equal air entry without any added sound. Abdomen showed four signs of cautery at the level of umbilicus that that patient done to himself 1 month after the starting of his complain without any sign of infection. The abdomen was soft and lax without organomegaly. CVS examination normal S1+S2 without audible murmur.

Initial Labs workout WBC 0.5 , Hgb 5.6, PLT 20 , CRP 339, RFT normal. Total bilirubin 33, Direct bilirubin 24, Albumin 16, INR 1, random sugar 8. Gaint platelet is present, direct comb test was negative, transferrin 63.5, Rct 0.67%, Rct count 0.012, sickling test was negative.

US left lower limb vein : no evidence of DVT, or collection. US of the left knee : joint effusion in the suprapatellar pouch measure about 4*3*2, volume 12ml suggesting septic arthritis for further evaluation. Paracentesis of the left knee showed: WBC 101 neutrophil <29% , normal chemistry, gram stain was negative.

Patient was taken to the operation room and Surgical debridement was done to the left Lower limb up to the level of the thigh (figure 1,2). Debridement was done to the left upper limb up to the level of the forearm . Dressing was done with adaptive and crepe bandage. Patient received 10 unit of platelet transfusion and 3 PRBC during the operation. And was started in antibiotics, IV imipenem 100mg IV Q8H , vancomycin 1000mg IV Q12H, filgrastim 300mcg SQ, , fluconazolm, acyclovir

Patient was screened for infectious disease as possible causes next day. HBS abs 13.66, HBS AG is none reactive, HIV screening AB and AG negative. CMV igG reactive, CMV igM non-reactive, ferritin 2725.8, folate 14.1. Synovial fluid aspiration of the left knee showed yellow fluid slightly hazy, WBC 101, RBC 181, Neutrophil 29, macrophage 22, high viscosity, No crystal. Bone marrow aspiration confirmed the finding of sever aplastic anemia.

Further imaging was ordered to make a full assessment. Left Femur CR : diffuse soft tissue edematous changes. Left Ankle CR: no definite lesion are detected , no bony or articular abnormality are noted, the ankle mortise is grossly configuring multiple opaque mass are seen (figure 3). Left Knee CR: diffuse soft tissue edematous changes with joint effusion. Left Leg CR : no definite lesion is detected, diffuse soft tissue edematous changes with multiple radio opaque masses at the lower leg, air loci are seen in the soft tissue raising the possibility of infection (figure 4). Right Hand CR: no abnormality. Right Humors CR: normal. Right Forearm CR: diffuse soft tissue edematous changes (figure5).

After 2 days the patient chest examination showed diminished air entry bilateral and CR chest ordered and showed Mildly enlarged cardiac size. Bilateral hilar lung opacities and interstitial lung smooth thickening. Left CP angle is mildly blunted, the right CP angle is normal. Osseous structures are grossly intact (figure 6). US Right Lower Extremity Veins: There is no evidence of deep venous thrombosis but there is Subcutaneous edema of the leg. MRI of the right thigh showed Multi compartmental intramuscular and deep intermuscular fascia edematous changes in the right thigh and leg along with intramuscular hemorrhage of the soleus muscle; however, no abnormal soft tissue signal void to suggest soft tissue gas, Findings could represent necrotizing fasciitis for clinical correlation. Labs workout showed: WBC 0.47 HGB 6.8 PLT 14 PT 12.7 PTT 21.7, Urea 8.5 creatinine 75 Na 139 K 2.8 Mg 1.01   Ca 1.75   Cl106, total bilirubin 21.22   direct bilirubin 15.63 protein total 44 albumin 12 ALT 23 AST 27. ANCA negative, anti-cardilipin IgA negative, anti-cardilipin IgG negative, anti-cardilipin IgM negative, anti DNA ABS negative, Brucella igG negative, Brucella igM negative, malaria screening negative.The Patient was scheduled to OR for further debridement of the left leg and possible skin graft from the right leg. In the OR, the left leg showed further extension of necrotic tissue reaching above the middle of thigh and new blister formation over the thigh and skin

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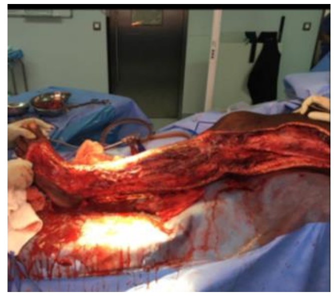
grafting was canceled, the right leg showed black discoloration from the foot till above the ankle, with crepitation and blisters suggesting necrotizing fasciitis. The left leg was unsalvagble, and above knee amputation was advised but consent needed to be signed first and debridement was done instead. Right arm showed black discoloration with crepitation suggesting early signs of necrotizing fasciitis. The patient received 2 unit of platelet prior to starting the operation. His lab today showed WBC 0.7, hgb 7.3 PLT 7 PT 15 PTT 26 Urea 7.3 creatinine 60 Na 140 K 2.7 MG 0.79 PO4 0.89 CA 1.72 CL 107 total bilirubin 28.51 direct bilirubin 17.06 protein total 51 albumin 16 ALT 15 .

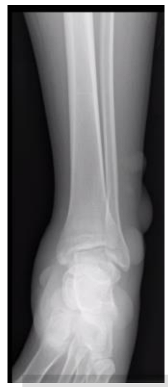
The patient was febrile after the operation and urgent US abdomen showed hepatomegaly, without any focal lesion, with free fluid in the pelvic. CT chest: bilateral pleural effusion with bilateral nodular pulmonary opacities, multiple wedge shaped bilateral renal cortical hypodense suggesting renal infarct vs hydronephrosis, hepatomegaly with small to moderate ascites.

Final assessment of the patient confirmed presence of four limbs necrotizing fasciitis with sever aplastic anaemia and sepsis which suggested no favourable outcome and the patient was signed for DNR and transferred to hematology and palliative care.

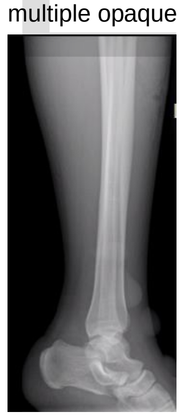
debridement the left Lower limb up to the level of the thigh(Figure-1)



debridement the left Lower limb up to the level of the thigh (Figure-2)



Left Ankle CR: no definite lesion are detected , no bony or articular abnormality are noted, the ankle mortise is grossly configuring multiple opaque mass are seen(Figure-3)



Left Leg CR : no definite lesion is detected, diffuse soft tissue edematous changes with multiple radio opaque masses at the lower leg, air loci are seen in the soft tissue raising the possibility of infection (Figure-4)

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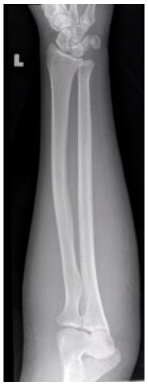
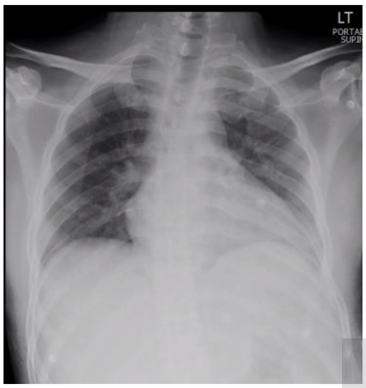


right forearm x-ray showing soft tissue edema (Figure-5)



CXR: Mildly enlarged cardiac size. Bilateral hilar lung opacities and interstitial lung smooth thickening. Left CP angle is mildly blunted, the right CP angle is normal. Osseous structures are grossly intact (Figure-6)

3 Discusion

For papers accepted for publication, it is essential that the Necrotizing soft-tissue infections (NSTIs) are very rapid progression and lethal infections. They can be defined as infections involving any layers of the soft tissue compartment (dermis, subcutaneous tissue, superficial fascia, deep fascia, or muscle) with necrotizing changes (1).

Early recognition and management of NSTI is very crucial and challenging because of the very rapid disease course and high mortality rate. Patients without surgical interventions report a mortality rate of 80% (2)

The diagnosis usually start by identifying the high risk group which include patient with known DM, advance age (3). Also patient who carry a high index of suspicion. For example, postoperative wound, history of trauma, known cutaneous disease (4).

Clinical characteristic of NSTI can mimic cellulitis or abscesses, which make the correct diagnosis difficult. The clinical presentation can vary depending on the microbiologic pathogen responsible, as well as the anatomical region and depth of infection. In general, erythema, pain beyond the margins of obvious infection, swelling, and fever are the most common clinical finding. There are some clinical sign that suggest NSTI who are known as "hard but they tend to occur later on. These include (1) the presence of bullae, (2) skin ecchymosis that precedes skin necrosis, (3) presence of gas in the tissues by examination or radiographic evaluation, and (4) cutaneous anesthesia.(5)

Lab workup may help in identifying early signs of infection. WBC greater than 15.4 × 109/L and serum Na less than 135 mmol/L suggest presence of necrotizing fasciitis (6). Recently, Wong et al. created a scoring system (laboratory risk indicator for necrotizing fasciitis score). There are a total of 6 independent variables associated with NSTI which are (total white cell count, hemoglobin, sodium, glucose, serum creatinine, and C-reactive protein) (7) .The total score had a range of 0–13,score indicate a intermediate and high-risk patients which have PPV of 92% and a NPV of 96% (8).

Imaging study maybe used to establish the presences of NSTI, for example. Plain radiography may show subcutaneous gas. This is a very specific finding, but it is not very sensitive in patients with NSTI. CT can identify the presence of other causes of infection like deep abscesses. MRI have a higher sensitivity rate than specificity in the diagnosis of NF (9, 10) an it may show increase in the thickness of the fascial layer with or without enhancement. The primary limitation of these studies is that they tend to compare the involved site (usually a limb) with the contralateral or

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uninvolved limb, rather than comparing it with a non necrotizing soft-tissue infection (1). bedside incisional biopsy is one of the method that can help in the early recognition and treatment, especially when there is no enough evidence in the physical examination in early disease process. incisional biopsy is supported by the work of Loh et al (10) but ,negative results can result from insufficient sampling. This condition may involve any part of body but most commonly it involves extremities, abdomen or perineum.(11)

The ministry of treatment of NSTI is debridement with the use of appropriate broad-spectrum antibiotic coverage, combined with adequate organ support and close monitoring. There are many acceptable regimens include monotherapy agents, such as imipenem, meropenem, ertapenem, piperacillin/tazobactam, and tigecycline. Multidrug regimens have also been described, including triple-drug therapy regimens, such as high-dose penicillin, high-dose clindamycin, and a fluoroquinolone or an aminoglycoside for coverage of gram-negative organisms. Vancomycin, daptomycin, or linezolid should be included in the regimen until methicillin-resistant staphylococcal infection has been excluded. The use of clindamycin, may help by inhibiting toxin production, which can be crucial for controlling the inflammatory response in patients with NSTI, particularly in those with clostridial and streptococcal infections (12).

4 conclusion

NSTI is an infrequent but life threatening condition. Establishing the diagnosis is very challenging in the early stages but can improve the patient mortality and morbidity .Aggressive management, include surgical debridement with broad-spectrum antibiotic and organ support is the ministry of treatment.

5 Refrances

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