



## NECROTISING FASCITIS OF CHEST WALL AND UPPER LIMB IN YOUNG ADULT- RARE CASE REPORT

### General Surgery

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| <b>Dr Paulia Devi</b>    | Assistant Professor, Institute of General Surgery, Madras Medical College, Chennai, Tamil Nadu, India |
| <b>Dr Manivannan</b>     | Associate Professor, Institute of General Surgery, Madras Medical College, Chennai, Tamil Nadu, India |
| <b>Dr Kishaan Prabhu</b> | Resident, Institute of General Surgery, Madras Medical College, Chennai, Tamil Nadu, India            |

### ABSTRACT

Necrotising fasciitis is a subset of aggressive skin and soft tissue infection that cause necrosis of muscle, fascia and subcutaneous tissue. Delay in treatment can result in significant morbidity and mortality. Although necrotising fasciitis can affect all body part, lower limb followed by abdomen and perineum is more common. Necrotising fasciitis of upper limb and chest wall are both rare conditions which requires aggressive and prompt treatment. Here we present a case of 24 year old male who came with chief complaint of progressive redness and swelling around left nipple. Two month back following an unknown bite developed necrotising fasciitis of left forearm. Patient was subjected to two debridement procedure and secondary suturing of the wound was done. Now without any inciting factor, patient developed necrotising fasciitis of left anterior chest wall. Patient was taken for two debridement procedure. After adequate granulation of chest wall, split thickness skin graft was done. Patient was discharged uneventfully. Necrotising fasciitis is a surgical emergency. Due to non-specific symptoms in early stage they are usually misdiagnosed. High suspicion even at atypical site is required. Key factors in successful treatment of necrotising fasciitis is early diagnosis and serial and adequate debridement procedures.

### KEYWORDS

### INTRODUCTION

Necrotising fasciitis is a subset of aggressive skin and soft tissue infection that cause necrosis of muscle, fascia and subcutaneous tissue. Delay in treatment can result in significant morbidity and mortality. Although necrotising fasciitis can affect all body part, lower limb followed by abdomen and perineum is more common. Necrotising fasciitis of upper limb and chest wall are both rare conditions which requires aggressive and prompt treatment.

### Case Report

A 24 year old male patient presented to emergency department with 4 days history of pain associated with progressive swelling with redness around the left nipple. Patient had a history of low grade fever for the past 4 days.

Patient had a past history of unknown bite in his left cubital fossa 2 months back following which he developed redness in the bite region and fever and tiredness for 3 days. Patient was treated with oral paracetamol, antibiotics and antiallergic in local hospital. After 10 days of insect bite, patient had persistent fever and developed swelling of the left forearm. Patient was treated with glycerine and Mgso4 application, compressive dressing and limb elevation. As there was no improvement in swelling and symptoms and developed necrotic skin patch, patient was diagnosed as necrotising fasciitis of Left forearm and emergency wound debridement was done. After 1 week patient was taken up for second wound debridement and secondary suturing of the wound was done. Patient was discharged after 1 month of hospital stay.

In the present admission, patient febrile with fever of 100.2 F and tachycardia 105 beats/min. On examination, patient had diffuse redness and swelling over the periareolar region of size 6x 6 cm. He had a pustule of size 3mm\*3mm at 8'o clock position near the areola with seropurulent discharge. Immediately, routine investigations were sent and he was subjected to bedside POCUS. It revealed e/o diffuse subcutaneous edema over the left anterior chest wall with left pectoralis major edema and ill defined collection of size 1.7\*1.8 cm beneath left nipple areolar region. Impression was given as p/o acute myofasciitis involving the left anterior chest wall.

Under IV sedation, patient was taken up for emergency OT and incision was made in the periareolar region connecting the pustule. All loculi were broken down and seropurulent discharge was drained and send for pus c/s. Through wash was given.

Post- operatively, patient was started on injection cefotaxime and injection metronidazole. On Pod 1 and 2, patient had progressive

redness of the left anterior chest wall region and persistent intermittent fever and diffuse pain. As the patient had progression of disease, we decided to do second debridement. On Pod 3, patient was taken to OT and under general anesthesia, large Y-shaped incision was made in the anterior chest wall connecting the previous incision. All the sloughed out subcutaneous tissue and small portion of sloughed out pectoralis fascia was removed. Underlying pectoralis muscle was healthy and was left as such. Through wash was given and sterile compressive dressing applied.

Post- operatively, antibiotics was escalated to injection vancomycin and injection metronidazole. Patient clinical improved and there was no fever episode after the second debridement. Daily saline wash with minimal removal of Slough was done. After healthy granulation of the chest wound, plastic surgery opinion was obtained and planned for split thickness skin graft. On Pod - 22 after second debridement, STSG was done and patient was discharged Pod-7 with 100 percent uptake of graft

### Culture Sensitivity:

In the previous admission for left forearm necrotising fasciitis, culture reports were negative( No growth). In the admission for chest wall necrotising fasciitis, two consecutive culture sensitivity reports were negative. Anaerobic culture was also sent, which also turned out to be negative.



**Figure 1 - Healed Wound In Left Forearm**



**Figure 2 - Post Incision And Drainage Wound With Slough On Pod-1**



**Figure 3 - Chest Wound After 2nd Debridement**



**Figure 4 - Healthy Granulation On Pod - 20**



**Figure 5 -After Split Thickness Skin Graft**

### DISCUSSION

Necrotizing fasciitis affects about 0.4 in every 100,000 people per year in the United States. In some areas of the world, it is as common as one in every 100,000 people.[1] Necrotizing fasciitis of the chest wall is a rare pathology, but is a life-threatening condition with a high mortality rate. In the 2000's, a total of 4 cases per million were reported in the USA and 10 cases per million were reported in Western Europe, while the incidence on the North American continent was approximately 1,000 cases per year.[2]

All body parts can be affected by NF, but the extremities remain the most frequent site of infection, making up 45% - 74% of all cases, according to different studies. About two-thirds of these cases concern the lower extremities, while NF of the upper limb is less common, being involved in approximately 6% - 27% of the patients and severe necrotizing soft tissue infections involving the upper body and chest wall are distinctly uncommon.[3] The first mention case was made by Hippocrates in the 5th century BC, as a complication of an erysipelas. In the 18th century, it was described by English doctors as 'gangrenous ulcer', 'rotten ulcer' or 'hospital gangrene'. There is no predisposition in regards to sex and it has been observed more often in elderly patients and rarely in children.[2]

It can affect any and all layers of soft tissue, including skin, fat, fascia, and muscle causing widespread necrosis and microvascular thrombosis of blood vessel. The infection usually begins at the level of hypodermis and in the superficial fascia, as the outermost layers (dermis and epidermis) are not affected in the early stages. The offending organism(s) release toxins and initiate a cascade of endogenous cytokine reaction that contributes to widespread tissue destruction. The presence of a hypoxic environment favors the growth of anaerobic bacteria. Invasive pathogen(s) cause thrombosis to the nutrient vessels that are in the epidermis, leading to extensive tissue ischemia, aggravated by the presence of edema. Subsequently, tissue ischemia promotes infectious dissemination that eventually leads to skin necrosis at a later stage. The gas formed by anaerobic bacteria may lead to crepitus.[3]

NF of the upper extremity is usually attributed to the inoculation of the pathogen into the subcutaneous tissue, which can occur via any break in an epithelial or mucosal surface. Skin traumas, lacerations, burns, bites (from animals, insects, or even humans), iatrogenic invasive causes (venipuncture, percutaneous catheter injections, intra-articular injections, insulin injections, postoperative complications), chronic skin infections, fistulas, neglected wounds, ulcers, and abscesses have been incriminated as causative factors. Nevertheless, many times there is an absence of an obvious external portal of entry for pathogens. Idiopathic necrotizing fasciitis accounts for 11%-20% of all cases in various studies. Spontaneous cases without a portal of entry reportedly comprise nearly half of all NSTI cases caused by GAS. Insect bites can

cause various types of infections such as impetigo, folliculitis, cellulitis, erythema, papule or papulo-vesicle hemorrhagic bulla and lymphangitis. Although rare, insect bites can cause benign subcutaneous emphysema and necrotizing fasciitis.[3,5,6]

These infections are often polymicrobial, although a single organism may be responsible in as many as 77% of infected patients. Monomicrobial infections often present in a fulminate fashion, with systemic toxicity highlighted by fever, leukocytosis and, occasionally, shock. In contrast, polymicrobial infections may present in a more insidious manner. These infections are caused by synergy among anaerobes and facultative aerobes, involving both gram-negative and gram-positive organisms. The first class is a polymicrobial one, with combinations of gram-positive cocci, gram-negative bacilli and anaerobes of the Clostridium type. They are located especially in the chest and perineum. It especially affects the elderly and the immunocompromised. Type II involves fasciitis with group A beta hemolytic Streptococcus alone or in combination with Staphylococcal species. In addition, it shows a rapid evolution with high toxicity, observing an impairment of younger and healthier patients than in type I. Type III is specifically described on the West coast of the USA with gram-negative marine Vibrio vulnificus type. Type 4 includes fungal infections, commonly from Candida spp. and Zygomycetes. These infections usually occur after trauma, mainly affect immunocompromised patients, and demonstrate an aggressive and rapidly extensive clinical course.[1,4]

Necrotising fasciitis has an unpredictable clinical course. Clinical manifestations are nonspecific at the time of admission. It gives a false impression of less severe condition like cellulitis therefore leading to misdiagnosis. It has a rapidly progressive clinical course with pain disproportionate to the skin color changes. Necrotizing infections are more commonly present with excruciating pain out of proportion to presenting symptoms and systemic septic signs than non-necrotizing infections. After several days the overlying skin, which appears unaffected initially, will transition to an erythematous, reddish-purple to bluish-gray hue. The texture of the skin will become indurated, swollen, shiny, and feel warm in temperature. The processes of fasciitis with induration, collection, erythema, pain, crackles, bullae and cutaneous gangrene have been obviously identified, usually at 3-4 days. A characteristic frequently present in this pathology is represented by the discrepancy between the apparently minimal visible skin damage and the noisy systemic symptoms. For any suspicion of necrotizing fasciitis, the finger test is recommended. Thus, after local anesthesia, an incision 2-3 cm wide enough to insert the index finger to the deep fascia is made. The lack of bleeding or the appearance of a gray necrosis fluid are very suggestive for necrotizing fasciitis. The easy digital dissection of the planes with the minimum tissue resistance is a golden standard for diagnosis. Plain radiographs have low specificity and sensitivity in recognizing NF and are unremarkable until the infection is advanced, but they can show the gas formation and soft tissue swelling. Imaging confirmation is required to assess the extent. The presence of air bubbles is easily made based on the standard radiological examination and, more recently, POCUS (point of care ultrasound). The routine paraclinical imaging examination for diagnosis and for the evaluation of extension is the CT examination. Thus, a CT examination can show specific signs of necrotizing fasciitis: signs of collections, edema in the subcutaneous and fatty tissue, fascial edema, gas collections locally or with locoregional extension. POCUS examination is becoming more widespread due to its ease of use even when we have minimal suspicion. (1)MR imaging can also help determine the extent of necrosis and differentiate necrotizing from non-necrotizing fasciitis. Intravenous administration of gadolinium-based contrast material can distinguish whether a muscle is viable or necrotic and delineate abscesses.[3]

Some laboratory-based scoring systems have been proposed for establishing the early diagnosis of necrotizing fasciitis and determining the prognosis. Laboratory Risk Indicator for Necrotizing Fasciitis score (the LRINEC score), proposed by Wong et al., is one of them. The Laboratory Risk Indicator for Necrotizing Infection (LRINEC) Score was developed in a 2004 report to distinguish NSTIs from other severe soft tissue infections. The scoring system is hinged on abnormalities in six independent variables:

- C-Reactive protein (mg/L). :≤150 -0  
: >150 -4
- Total white blood cell count(1000 cells/μL): <15 -0  
: 15 to 25 -1  
: >25 -2
- Hemoglobin (g/dL). : >13.5 -0

- Sodium (mmol/L). :  $\geq 135-0$   
:  $< 135-2$
- Creatinine (mg/dL). :  $\leq 1.6-0$   
:  $> 1.6-2$
- Glucose (mg/dL). :  $\leq 180-0$   
:  $> 180-1$

A score of six has a positive predictive value of 92% and a negative predictive value of 96%. A score of eight or greater represents a 75% risk of necrotizing infection.[7]

when imaging findings are nonspecific, and clinicopathologic correlations must be sought. Therefore, necrotizing fasciitis can be diagnosed from the wound biopsy and the diagnosis should be supported with clinical and laboratory tests. Tissue obtained from the operating room after debridement will usually show extensive superficial fascial necrosis. The majority of small and medium-sized blood vessels will be thrombosed. Aggregates of neutrophils will be observed in the fascia and subcutaneous tissues. Small vessel vasculitis and extensive fat necrosis will also be evident. All the glands in the dermis and subcutaneous tissues will be necrotic as well. Gram stain will show clusters of various types of microorganisms.[8]

The progression of the disease is usually fulminant, and NF quickly deteriorates to a life-threatening condition, potentially leading to septic shock, SIRS (Systemic Inflammatory Response Syndrome), concomitant multisystem organ failure, and eventually death. Thus, aggressive multimodality medical and surgical treatment is needed.

These patients are extremely ill and should be transferred immediately to the intensive care unit. The sepsis causes refractory hypotension and diffuse capillary leak. Thus the patient will need aggressive resuscitation with fluids and the use of inotropes to maintain blood pressure. The patient must be kept NPO (nothing by mouth) until seen by the surgeon. (Sp). Broad-spectrum intravenous antibiotics should be done immediately, covering gram-positive cocci, gram negatives rods, anaerobes.[9]

The amount of initial debridement in NSTI patients is controversial. Although radical resection has been recommended in terms of infection control, a large skin and soft tissue deficit would cause loss of anatomical function and make later reconstruction difficult. Initial Surgical treatment should be based on debridement through wide drainage incisions. Serial surgical debridement coupled with peroxidase, hyperbaric oxygen therapy, negative-pressure wound therapy (NPWT) has been reported as safe, and it saves skin and soft tissue if needed for flaps in cases of Fournier's gangrene. [2,5]

## CONCLUSION

Necrotising fasciitis is a surgical emergency. Due to non-specific symptoms in early stage they are usually misdiagnosed. High suspicion even at atypical site is required. Key factors in successful treatment of necrotising fasciitis is early diagnosis and serial and adequate debridement procedures.

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