Case Report

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Polymicrobial Necrotizing Fasciitis – A Fatal Outcome following Motor Vehicle Accident

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ABSTRACT

Necrotizing fasciitis is an uncommon skin infection characterized by rapidly progressing, extensive skin necrosis involving the soft tissues and superficial fascia. It begins with non-specific local cutaneous signs, posing a diagnostic difficulty for the clinicians, evolving into life-threatening systemic toxicity. Trauma is more often known to be a causal infective etiology. The presence of underlying comorbidities has been observed in cases of necrotizing fasciitis. Various microorganisms, including a mix of anaerobes and aerobes, marine creatures like vibrio, or even fungi, can cause necrotizing fasciitis. Thus, necrotizing fasciitis may be polymicrobial or monomicrobial. We report a case of an 80-year-old pillion rider of a motorbike involved in a road traffic accident. The injury sustained resulted in an uncommon complication of polymicrobial necrotizing fasciitis, causing the individual's death. The microbial invasion of the skin is considered to release toxins, damaging tissues and causing necrosis. Coagulase-negative staphylococci is a common human skin aerobic, gram-positive coccus. It has shown the propensity to cause bacteremia. Candida, a known invasive organism, can play a potential role in fasciitis. This inter-microbial synergism may stimulate an aggressive systemic inflammation leading to septic shock.

Keywords: Necrotizing Fasciitis, Polymicrobial, Motor Vehicle Accident, Coagulase-Negative Staphylococci, Candida Albicans.

INTRODUCTION

Necrotizing fasciitis (NF) is an uncommon skin infection characterized as a rapidly progressing extensive necrosis of the skin involving the soft tissues and superficial fascia. It usually begins with non-specific local clinical signs and often leads to life-threatening systemic toxicity.[1,2] Wilson first used the term NF in 1952 to describe the necrosis involving the subcutaneous tissues and layers of superficial fascia with sparing of deep fascia and muscle.[3] Trauma is the most identified initiating infective etiology, including a chronic abscess, peripheral vascular disease, osteomyelitis, injury by fish fins, etc. Mostly, the trauma can be minor or significant, such as abrasion, bruise, laceration, penetrating injuries, or surgical procedure.[1-3] Diabetes mellitus is the most predominant underlying co-morbid disease condition. Other underlying conditions include peripheral vascular disease,

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smoking, alcoholism, intravenous drug use, immunosuppression, obesity, old age, etc. Lower limbs were more frequently involved than the upper limbs.[4] NF is more typical in individuals of more than 50 years.[5]

Erythema is the most frequent presenting sign, followed by a hallmark out-of-proportion pain beyond the areas of apparent involvement along with fever.[2] In later stages of NF, cutaneous manifestations such as bullae or blister formation with infected pus discharge, which finally extends into a total thickness skin necrosis leading to skin anesthesia.[5,6] At least one underlying comorbidity has been frequently observed in cases of NF.[6] The microbial etiology of NF can be monobacterial, polymicrobial or fungal.[7,8] The mortality rates reaching from 25% to 73%.[9] The present case discusses how injury sustained during a road traffic accident, developed into a fatal polymicrobial NF.

CASE REPORT

Alleged history of a motor vehicle accident where another 2-wheeler hit a 2-wheeler, and an 80 years old male pillion rider injured his right leg from the wheel spoke. He was treated at a local hospital and discharged the next day. The patient had known diabetes mellitus and hypertension comorbidities, but he was not on regular medications. After two months, the patient presented to our hospital with purplish-black discoloration of the right lower limb, pain, and multiple blisters with putrid discharge from the same injury site with a non-healing penetrating injury. The patient was febrile, and tenderness of the right leg was present. Yellowish-green pus discharge was noted from an ulcerated lesion on the right leg. Hematological screening showed hemoglobin (Hb) of 7.8 g/dl, a white blood cell (WBC) count of 25,420/µL, and a platelet count of 85,000/µL. The urea and creatinine levels were elevated to 183 mg/dl and 5.38 mg/dl, respectively, and the physician did a urine culture.. The random blood glucose was 209 mg/dl. The liver enzymes were all raised.

On day two of hospital re-admission, fasciotomy and extensive debridement of the right leg were performed. A tissue section from the debrided region was sent for culture, and a repeat urine culture was sent. Procalcitonin levels of 75.59 ng/ml. Blood for culture was collected. The patient's condition further deteriorated; he developed shortness of breath with a drop in saturation and was intubated. On day five, blood for fungal culture was sent. On day 8, Hb was 6.6 g/dl, and WBC was 18,280/µL. The peripheral smear examination demonstrated neutrophilic leucocytosis with toxic changes in WBCs, and the Red blood cells (RBCs) were microcytic hypochromic. Both urine cultures isolated budding yeast cells. The debrided tissue culture isolated Coagulase-Negative Staphylococci (CoNS) and Candida albicans, while the blood culture isolated only Candida albicans. On day nine, the patient died, and the body was referred for a medicolegal autopsy.

During the autopsy, extensive debridement with areas of blackish discoloration was present over the right leg, with skin sloughing over the right knee and thigh. Unhealthy granulation tissues were present covering the debrided areas of the right leg (Figure-1). Multiple tiny blebs were noted over the dorsum of the right foot. Blackening and purplish discoloration of the toe and sole were present (Figure-2). On internal examination, the lung cut surfaces were markedly congested and edematous. Fluid-filled cysts were present over both kidneys' surfaces, and on the right kidney's cut section, a cortical cyst was present. The other internal organs were grossly unremarkable. Histopathology (H & E staining, 200X) of skin from the right leg showed epidermal disruption and necrosis beneath the basement membrane involving the upper dermis (Figure-3). Lung histology showed broncho-pneumonic changes with intra-alveolar edema. Kidneys had focal lymphoplasmacytic infiltration. The cyst wall on histology demonstrated a simple cortical cyst. The other internal organs were histologically within normal limits.

Fig. 1: (A) Extensive debridement with areas of blackish discoloration (black arrow) over the right leg, with sloughing of skin over the right knee and thigh. (B) Unhealthy granulation tissues covering the debrided areas of the right leg.

Fig. 2: (A) Multiple tiny blebs (black arrow) over the dorsum of the right foot. (B) Blackening and purplish discoloration of the toe and sole (black arrow).

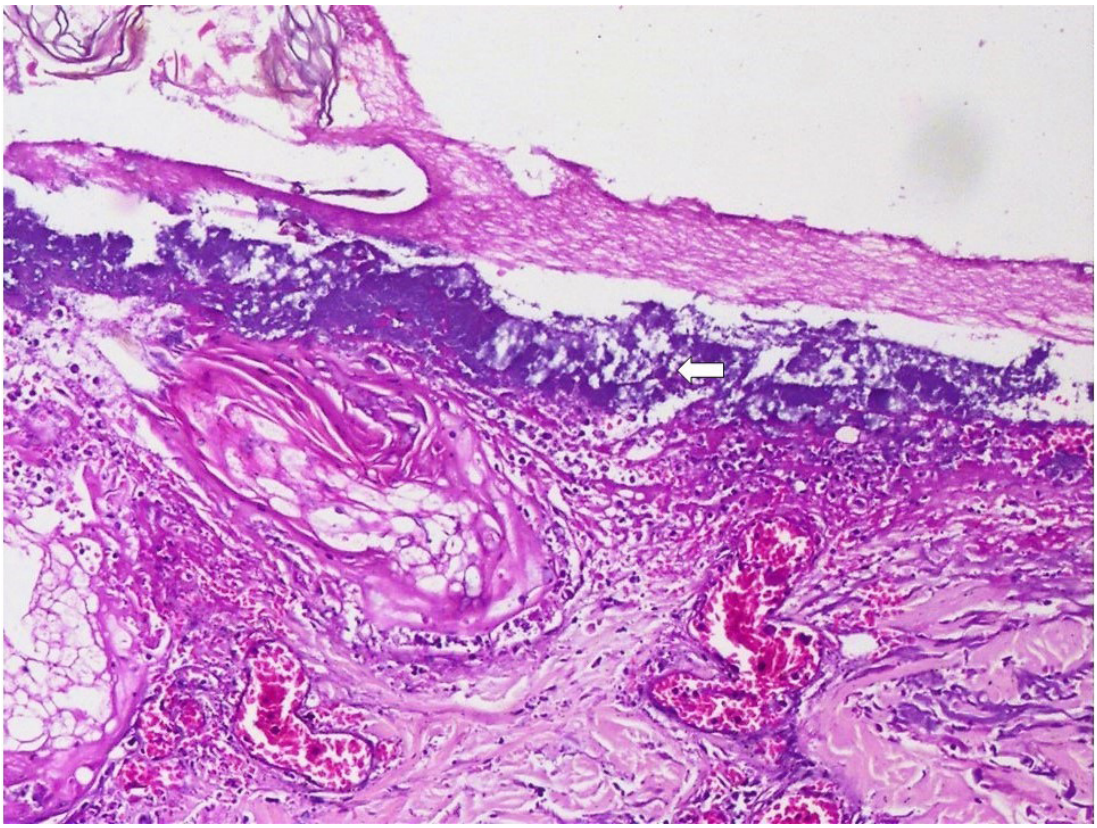

Fig. 3: Histopathology (H & E staining, 200X) of skin from the right leg showed epidermal disruption (white arrow) and necrosis beneath the basement membrane involving the upper dermis.

DISCUSSION

Classifying Necrotizing Fasciitis

The NF is categorized into four types I to IV. Type I NF, is usually polymicrobial and etiology of mixed bacterial anaerobes or aerobes. Type I NF have greater causative tendency in patients with diabetes mellitus, old age, and immunocompromised status.[5] Study conducted in Scotland, found polymicrobial NF of majority type (58%) with Streptococcus pyogens as the most frequently associated organism.[10] Type II involves a single bacterial species, usually group A Streptococci or Staphylococcus Aureus as reported by other authors.[5,11,12] Type III NF is associated with gram-negative and marine organisms. Type IV NF is related to traumatic etiology and has fungal involvement. Among fungi, candida species had shown high mortality.[5] A prospective study from India has identified more of monomicrobial (55.6%) involvement in NF than polymicrobial (44.4%) NF.[13] To the best of our literature search, the co-infection of CoNS and Candida albicans in causing NF has not been conversed.

Pathogenesis of necrotizing fasciitis and coagulase-negative staphylococci

The exact pathogenesis of NF is yet to be understood, mostly considered a microbial invasion of dermal layers releasing toxins, tissue damage, and advancing to skin necrosis. Bacterial toxins stimulate cytokine production, severe systemic inflammatory reaction, progressing into septic shock, causing multiorgan dysfunction.[14] Clinically significant in immunocompromised individuals.[15] Shen et al. described NF with CoNS, an aerobic, gram-positive coccus commonly found on human skin, increasingly recognized as a role in bacteremia. They isolated CoNS in 11 patients. Similar to the present case, lower limb skin lesions and underlying diabetes mellitus were common associations in eight and nine patients, respectively.[15] Sato et al. also isolated CoNS

in a case of NF from right lower extremity, similar to our case.[16]

Candida as a pathogen in NF

Candida is a well-recognized human pathogen known to cause a variety of diseases.[17] Candida albicans is the most frequently isolated organism in human fungemia. Candida albicans are known invasive organisms that play a potential role in causing fasciitis. Eisen et al. reported candida spp. as the solely isolated organism from tissue culture in a case of NF following a motor vehicle accident. One of the few pieces of literature available on Candida albicans NF.[18] Buchanan et al., has reported isolation of Candida albicans from sputum, blood and central catheter site cultures in a patient who was admitted with gunshot injuries and progressed into complications of NF and septecemia.[19] In our case, tissue culture isolated CoNS along with yeast cells. On microscopy, fungal culture revealed Candida albicans as an organism grown. The management of NF requires awareness of the causative spectrum of microbes to initiate targeted antimicrobial therapy.[20]

Microbial involvement in the case

Our case demonstrated putrid discharge from an ulcerated wound and blisters over the leg, suggesting the NF has already progressed to the intermediate to late stage of cutaneous manifestation.[2,6] In the present case, massive leucocytosis and hypergylcemia was present. Hung et al. noted similar observation in there case of NF caused by Staphylococcus lugdunensis, a CoNS.[21] In the present case, the patient had severely raised procalcitonin (75 ng/ml, normal - <0.05 ng/ml), a clinical marker of septic shock. These findings suggested systemic infection and evolvement into septicemia. The histopathology examination in reported cases of NF had observed dermo-epidermal necrosis.[1-3,7] Similar histopathological findings were noted in our case, and additional bronchopneumonia was noted. Our case demonstrated the cause of polymicrobial NF and septicemia as mixed infection due to CoNS and Candida albicans. In contrast to our case, literature reports polymicrobial NF involment mainly in the trunks and perineum while monomicrobial NF in the limbs.[11] The cause of death in our case was opined as polymicrobial NF and septicemia as complications of injuries sustained due to the road traffic accident. Inter-microbial synergism is still not studied well and may result in highly aggressive infection, as considered in Type I NF involving multiple species.[20]

CONCLUSION

The lack of definitive pathognomonic signs makes NF an early diagnostic challenge for clinicians and may threaten medical negligence claims against the treating physicians. The physicians must be vigilant while managing wounds in motor vehicle accidents, and the consequence of NF must be considered. Predisposing factors may decide the kind of microbial involvement.In patients with a history of diabetes and trauma following injuries, CoNS and Candida species, though rare, with only a handful of literature, should be considered as a differential in fatal NF. Therefore, histopathology examination, wound tissue, and blood culture should complement gross findings and clinical history. If microbial culture reports are available, clinicians and forensic pathologists can determine the progression toward systemic complications due to the spectrum of microbes and opine such death due to NF.

Declarations

Conflict of Interest: There is no conflict of interest.

Source of Funding: Nil.

Ethical Clearance: Not required.

NF – Necrotizing Fasciitis

CoNS - Coagulase-Negative Staphylococci

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