Bullous Erysipeloid: An Atypical Manifestation in an Immunocompromised Patient – A Case Report

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ABSTRACT:

Erysipeloid is a rare infection caused by *Erysipelothrix rhusiopathiae.* We report the case of a 58-year-old woman with HIV and spondylarthritis on immunosuppressants, who had a history of direct contact fish and shrimp, who presented with a progressive, painful plaque with central bullae on her left index finger. At her initial visit, she was diagnosed with bullous cellulitis and was treated with oral clindamycin (300 mg three times daily) and intravenous levofloxacin 750 mg once daily. Despite treatment, her condition worsened, necessitating hospitalization and surgical debridement. Microbiological culture from skin lesion confirmed the presence of *Erysipelothrix rhusiopathiae*, leading to the diagnosis of erysipeloid. Following a 14-day course of penicillin V, the patient showed significant clinical improvement. Atypical presentations of erysipeloid, as bullous lesions, can occur, particularly in immunocompromised host.

Keywords: Erysipeloid, *Erysipelothrix rhusiopathiae,* Immunocompromised host, localized infection, Zoonotic pathogen

Introduction

Erysipelothrix rhusiopathiae is a zoonotic pathogen that typically causes erysipeloid, a localized cutaneous infection resulting from contact with animal products such as fish, meat, or shellfish[1,2]. While erysipeloid is generally mild and self-limiting, it can present more severe manifestations, such as rapidly enlarging lesions that progress to bullae and necrosis, mimicking conditions like necrotizing fasciitis in immunocompromised patients[1-5].

We report a rare case of a progressive painful erythematous-violaceous plaque with a tense bulla in an immunocompromised host. This case highlights the importance of recognizing exposure history, utilizing microbiological diagnostics, and ensuring appropriate management.

Case Presentation

A 58-year-old woman with well-controlled HIV infection, diabetes mellitus, and peripheral spondylarthritis presented with a solitary, well-defined violaceous plaque with a central bulla on the base of her left index finger, which had developed over three days (Figure 1A). Her most recent laboratory results showed an HbA1C of 7.1%, a CD4+ T cell count of 385 cells/μL (23%), and an undetectable viral load (<40 copies/mL). She was adherent to antiretroviral therapy (dolutegravir, lamivudine, and tenofovir disoproxil fumarate) and was on immunosuppressive medications including methotrexate (7.5 mg weekly), leflunomide (60 mg weekly), prednisolone (5 mg daily), and sulfasalazine (2000 mg daily). She worked as an accountant.

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The patients denied a history of trauma, animal bites, chemical injuries or new medications. However, she reported handling fish and shrimp without protective gloves one week prior to the onset of the lesion. At her initial outpatient visit, she was diagnosed with bullous cellulitis and a skin biopsy was performed to investigate the etiology. According to the patient's immunocompromised status, which increased susceptibility to be infected by a wide range of pathogens, she was treated oral clindamycin (300 mg three times daily) and intravenous levofloxacin to provide broad-spectrum coverage against gram-positive, gram-negative, and anaerobic bacteria. Five days later, she returned the clinic due to disease progression. The lesion had rapidly extended from base of left index finger to proximal phalanx, accompanied by significant swelling and severe pain (Figure 1B). She was subsequently admitted to the hospital.

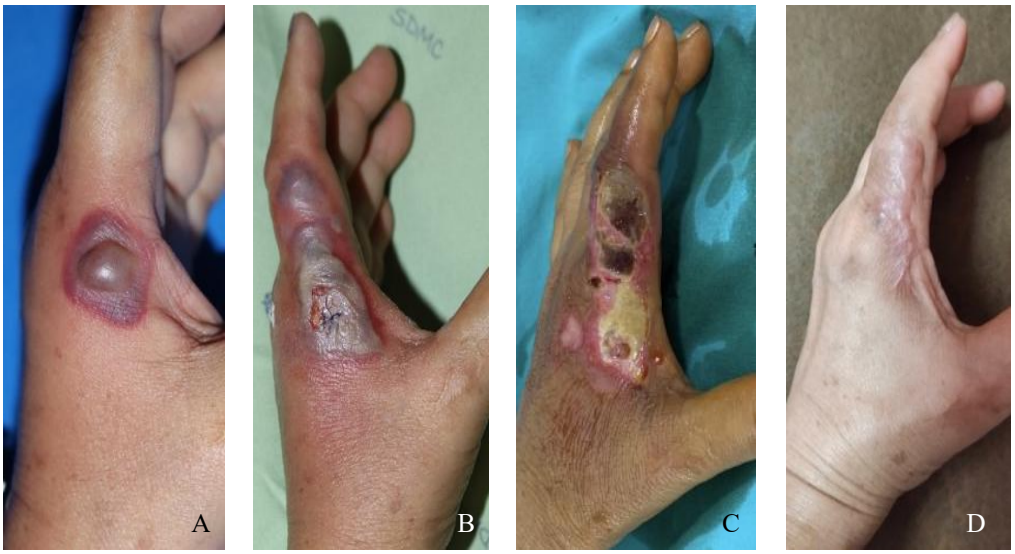

Figure 1 (A) A solitary, well-defined violaceous plaque with a central bulla on the base of left index finger at initial presentation. (B) Rapid extension of the lesion to proximal phalanx five days after treatment with oral clindamycin and intravenous levofloxacin. (C) Progression to ulceration with central necrosis, requiring debridement before switching to penicillin. (D) Complete healing after a 14-day course of penicillin, leaving a scar by the 1-month follow-up

On physical examination, her vital signs were normal, and there was no axillary lymphadenopathy. However, the lesion on her left index finger demonstrated pronounced ulceration with central necrotic changes (Figure 1C). A plastic surgery consultation was obtained for surgical debridement of necrotic tissue.

The complete blood count and blood chemistry were unremarkable. A pus swab for Gram staining revealed no organisms. Histopathological examination demonstrated subepidermal separation, marked papillary dermal edema (Figure 2A), and dense diffuse cellular infiltration with numerous neutrophils, without evidence of vasculitis (Figure 2B). Special stains, including Brown and Brenn, GMS, PAS, Fite, and AFB, showed no organisms. These findings were non-specific and may be consistent with neutrophilic

dermatoses, such as bullous Sweet syndrome, or infectious causes like bullous cellulitis, and should be correlated with clinical findings. Additional investigations, including tissue cultures for mycobacteria and fungi, were negative. PCR tests for mycobacteria, 16s rRNA and 18s rRNA also yielded negative results, and aerobic blood cultures showed no growth.

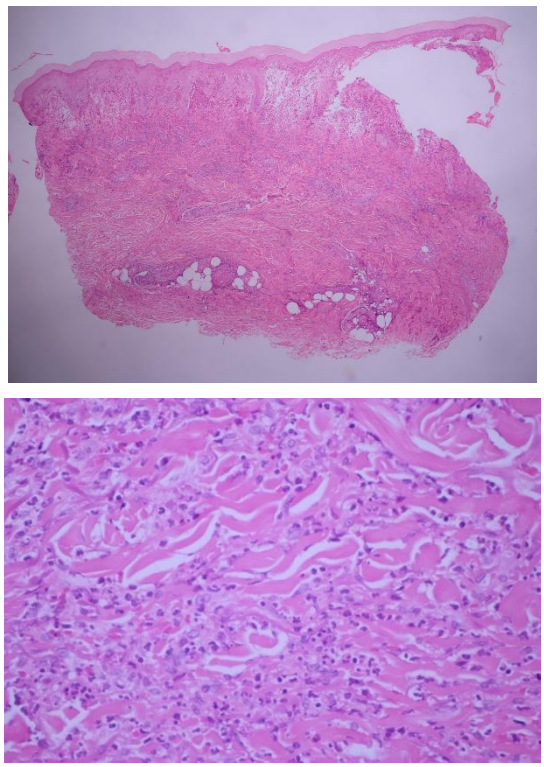

Figure 2 Histopathology with Hematoxylin-eosin stain showing subepidermal separation, marked papillary dermal edema and dense diffuse infiltration of inflammatory cells (A, X40). Numerous neutrophils infiltration in the superficial and deep dermis without evidence of vasculitis (B, X600)

However, microbiological culture from the specimen identified *Erysipelothrix rhusiopathiae*. The isolate demonstrated susceptibility to penicillin [minimum inhibitory concentrations (MIC) < 0.06 µg/mL], ampicillin (MIC < 0.12 µg/mL), ciprofloxacin (MIC 0.5 µg/mL), and levofloxacin (MIC 0.25 µg/mL). It showed intermediate susceptibility to erythromycin (MIC 0.5 µg/mL). Susceptibility to clindamycin was not tested.

Upon confirmation of *Erysipelothrix rhusiopathiae* as the causative organism and a final diagnosis of the localized cutaneous form of erysipeloid, the antibiotic regimen was switched to oral penicillin V (500 mg 4 times daily) for 14 days. Due to the absence of organ-specific symptoms such as dyspnea or fever and a negative aerobic blood culture, further evaluation for systemic involvement, including chest radiography and echocardiography, was not performed. The patient demonstrated significant clinical improvement, with gradually resolution of pain and ulceration. By the end of the treatment, the lesion had healed completely, leaving a scar (Figure 1D).

Discussion

Erysipeloid is an uncommon infection caused by *Erysipelothrix rhusiopathiae*, a gram-positive, facultative anaerobic bacillus. The infection is associated with direct contact with animals, animal-derived products, or contaminated environments. Common reservoirs include aquatic animals such as fish and shrimp, as well as poultry and pigs. Consequently, erysipeloid is typically linked to occupational exposure, affecting individuals such as housewives, fishermen, farmers, and veterinarians who frequently handle these animals or their products. The incubation period typically ranges from 2 to 7 days[1].

The pathogenicity and virulence of *Erysipelothrix rhusiopathiae* are mediated by several factors. Neuraminidase disrupts immune defenses, hyaluronidase facilitates bacterial spread through tissues, the bacterial capsule is resistance to phagocytosis, and surface proteins aid in biofilm formation, enhancing resistance to immune responses and antibiotics treatments[2]. Most of infections are

more common in males (75%) , and predominantly affect adults aged 19 to 59 years. Moreover, underlying conditions such as diabetes mellitus, chronic alcohol consumption, and immunosuppression significantly increased susceptibility to this pathogen[3].

The clinical manifestation of *Erysipelothrix rhusiopathiae* infection can be categorized into three forms[1,2]. The first and most common form is the localized cutaneous form, also referred to as erysipeloid. A 2022 systematic review reported that this form was observed in 29% of 62 patients[3]. It typical manifests as an erythematous to violaceous plaque. Hemorrhagic vesicles, bullae, or swelling may be present. The lesion usually affects the fingers or the back of the hands. Symptoms such as pruritus, pain, fever, lymphadenopathy can be present, but they are typically mild and self-limiting in healthy individuals[1-4]. However, this presentation is rarely documented in immunocompromised hosts[5]. Bullous and ulcerated cutaneous lesions are rare, with few reported cases in both immunocompetent and immunocompromised individuals, typically resolving without complications[1]. Hemorrhagic bullae may occur in immunocompromised patients, suggesting localized disease progression. However, no recent reports link these features to poor prognosis[5-8]. In our case, the lesion demonstrated extensive ulceration and necrosis with severe pain, indicating a more aggressive clinical course likely influenced by the patient's immunocompromised state.

The second form, diffuse cutaneous erysipeloid, involves widespread cutaneous lesions with negative blood cultures and accounting for 4.8% of reported cases[1,3,9]. The third and most severe form is septicemia, which occurs in approximately 36% of patients with prior or concurrent skin lesions. This severe form is more common in immunocompromised patients (17%) and individuals with chronic alcoholism (33%). Septicemia is often associated with endocarditis, contributing to a high mortality rate of 38%[10].

The diagnosis of erysipeloid relies on clinical presentation, exposure history, and microbiological confirmation. A detailed history of exposure to animal products or contaminated environments is essential for suspicion. Microbiological culture remains the gold standard for confirming *Erysipelothrix rhusiopathiae*. Specimens from skin lesions or blood (in systemic cases) can be cultured, revealing characteristic small, alpha-hemolytic colonies on blood agar. Advanced techniques such as 16S rRNA PCR and mass spectrometry (e.g., MALDI-TOF) are valuable for identification, particularly in culture-negative cases. Gram staining is generally unhelpful due to the low visibility of the pathogen[4].

Histopathological findings, while nonspecific, typically reveal marked superficial dermal edema, vascular dilatation, and neutrophilic perivascular infiltration. In vesiculobullous lesion, epidermal separation may be seen. Special stains (e.g., PAS, GMS) are used to exclude other infections[1].

Blood cultures are crucial for evaluating septicemia, especially in immunocompromised individuals at increased risk of developing severe or systemic infection[4,10]. Additional investigations should be guided by organ-specific symptoms. For example, echocardiography is indicated when infective endocarditis is suspected, chest radiography or computed tomography should be considered to assess pulmonary involvement[10,11].

Laboratory findings may show nonspecific inflammatory markers such as leukocytosis, elevated ESR, and CRP[1]. Accurate diagnosis requires integrating clinical, microbiological, and histopathological findings. Early diagnosis and treatment are crucial to prevent complications such as septicemia and endocarditis.

In terms of treatment options, penicillin remains the first-line treatment for

Erysipelothrix rhusiopathiae infections[1,2]. Alternative options that may be effective are tetracyclines, quinolones, and imipenem[1]. For localized cutaneous infections, oral penicillin V (500 mg every 6 hours) is recommended, while intravenous penicillin G (2–4 million units every 4 hours) is preferred for diffuse cutaneous or systemic disease. The duration of treatment is primarily guided by clinical response. Localized cutaneous infections typically require approximately one week. Diffuse cutaneous infections require a minimum of one week of treatment, with a possible transition to oral therapy after clinical improvement. However, the optimal duration of therapy has not been established in previous studies. Systemic infections generally require 2–4 weeks of treatment[4].

Previous studies have evaluated the MICs of various antibiotics against *Erysipelothrix rhusiopathiae*, with penicillin and ceftriaxone showing excellent activity with low MIC values[12]. For the patients with penicillin allergies, cephalosporins such as ceftriaxone (2 g once daily), are considered the most appropriate alternatives[2]. Although some reports have documented successful treatment with clindamycin and fluoroquinolones, their overall efficacy remains limited. Moreover, *Erysipelothrix rhusiopathiae* exhibits resistance to vancomycin, reduced susceptibility to the bacteriostatic agents clindamycin and erythromycin, and emerging resistance to tetracyclines and quinolones in recent animal studies, underscore the importance of appropriate antibiotic selection[2,13,14].

In our case, despite the absence of overt trauma, the patient had a clear history of exposure to aquatic animals and presented with atypical clinical manifestations of erysipeloid within the first few days. Her immunocompromised status and empirical treatment with levofloxacin and clindamycin likely contributed to the rapid progression of skin lesions. Although the skin biopsy was nonspecific, tissue culture confirmed *Erysipelothrix rhusiopathiae* as the causative organism. Antimicrobial susceptibility testing showed the lowest MIC for penicillin, followed by ampicillin, ciprofloxacin, and levofloxacin. This may explain suboptimal clinical response to initial empirical therapy.

Following a switch to oral penicillin and surgical debridement of necrotic tissue, the patient responded well. These findings further support the use of penicillin as first-line treatments, while underscoring the limitations of certain antibiotics in clinical practice. Most importantly, antimicrobial susceptibility testing should guide antibiotic selection. To reduce the risk of infection, the use of protective gloves is strongly recommended when handling fish, animal products, or potentially contaminated materials.

Conclusion

Occupational and environmental exposures, even without evident trauma, should raise suspicion for zoonotic pathogens in cutaneous infections. Immunocompromised patients may present with an extensive localized cutaneous form of erysipeloid. Crucially, diagnostic confirmation through microbiological culture is essential to identify the pathogen and appropriate antibiotic administration is crucial for effective management.

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