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Case Report

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# Bullous Erysipelas: A Case Highlighting Diagnostic and Therapeutic Management

Thien **Nguyen**[1*], Phuoc Nghia **Tran**[1], Anh Phuong **Dang**[2], Tuan Anh **Vu**[3]

## Abstract

A 73-year-old male with a history of stroke and residual right-sided hemiparesis developed bullous erysipelas on his immobilised right lower limb. The reduced mobility and compromised lymphatic drainage associated with hemiparesis are key predisposing factors for such infections. The presentation featured painful, asymmetric lesions with haemorrhagic bullae, leukocytosis, and neutrophilia. Histopathology confirmed the diagnosis, showing a mixed inflammatory infiltrate and marked dermal oedema. The bullous variant of erysipelas is associated with significant morbidity. Management with intravenous cloxacillin, a beta-lactam antibiotic, adjunctive systemic corticosteroids, and meticulous non-adhesive wound care led to significant clinical improvement within seven days, permitting hospital discharge for ongoing outpatient management. This case underscores the impact of timely, combined therapy, particularly in patients with neurological deficits, in reducing acute morbidity and hospitalisation duration.

**Keywords**: Erysipelas; Disease management; Infection; beta Lactam Antibiotics

**Author Details**:
1 Emergency Resuscitation Department, Quy Hoa National Dermatology Hospital, Gia Lai, Vietnam.
2 Pathology and Cytology Center, Bach Mai Hospital, Ha Noi, Vietnam.
3 Hospital Director, Quy Hoa National Dermatology Hospital, Gia Lai, Vietnam.

***Correspondence:**
Thien Nguyen
thien.r.py@gmail.com

## INTRODUCTION

Erysipelas is a common bacterial skin and soft tissue infection predominantly affecting children and the elderly.[1] The condition is primarily caused by *Streptococcus pyogenes* (group A Streptococcus), with a smaller proportion of cases attributed to methicillin-resistant *Staphylococcus aureus* (MRSA).[1] The infection involves the superficial dermis and superficial lymphatic vessels, manifesting clinically as well-demarcated erythema, warmth, swelling, and tenderness, occasionally accompanied by formations of bullae.[2] Diagnosis is chiefly clinical, though histopathological examination, bacterial culture, and imaging studies

may provide adjunctive support.[3] As it is a bacterial infection, the first-line therapy consists of beta-lactam antibiotics, which is preferred due to their efficacy against causative pathogens.[4] Adjunctive measures such as wound care and close clinical monitoring are critical to mitigating complications associated with inadequate management.

This report details a case of bullous erysipelas in a patient with post-stroke limb immobility. Reduced mobility and sensory perception in such patients increase susceptibility to minor trauma and infection, potentially complicating diagnosis due to atypical presentation or delayed recognition, and challenging treatment due to impaired healing and oedema management. We illustrate the diagnostic approach and highlight a therapeutic regimen that facilitated a rapid recovery.

## CASE REPORT

A 73-year-old male presented with sudden-onset erythematous patches accompanied by swelling and pain on the posterior aspect of the right lower leg. The lesions subsequently expanded, forming tense bullae over an erythematous base. The condition remained localised to the right leg, with no secondary lesions elsewhere. The patient had a history of stroke resulting in residual right-sided hemiparesis, including reduced mobility of the right leg. Associated symptoms included mild fever and malaise.

Upon admission, clinical examination revealed a well-demarcated, erythematous, warm, and tender plaque with hemorrhagic bullae (**Figure 1**). A clinical diagnosis of erysipelas was established.

Laboratory tests demonstrated leukocytosis (15.42 × $10^9$/L) with neutrophilia (85.60%). A Tzanck smear was negative for acantholytic cells. Histopathological examination confirmed marked subepidermal oedema, vascular dilatation with infiltrates of neutrophils and mononuclear cells, and scant eosinophils, consistent with bullous erysipelas (**Figure 2**). Bacterial culture from the bullous fluid or blood was not performed.

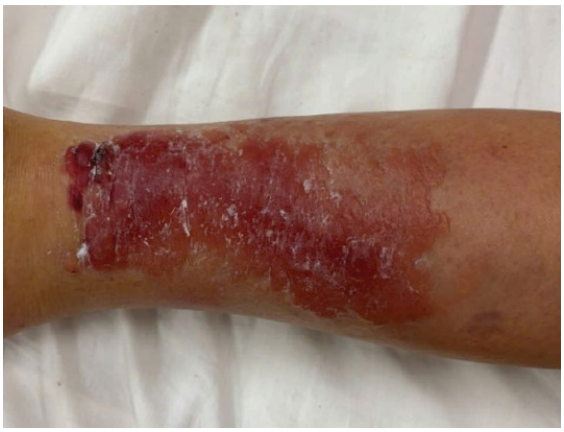

**Figure 1: The right lower leg: a well-demarcated erythematous plaque with tense haemorrhagic bullae, localised oedema, and warmth.**

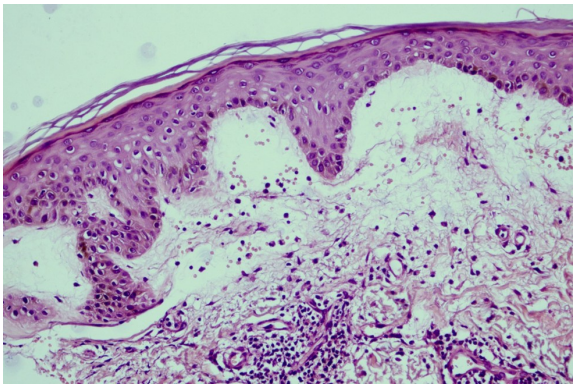

**Figure 2: Histopathological slide: Marked subepidermal oedema, vascular dilatation with neutrophil and mononuclear cell infiltrates, and scant eosinophils (Hematoxylin and eosin, 40x).**

The patient was treated with intravenous cloxacillin (4 g/day) and methylprednisolone (40 mg/day). Wound care included aspiration of bullous fluid, antisepsis with 10% povidone-iodine, and dressing with Urgo non-adhesive gauze (UrgoTul Absorb, Urgo Medical, Chenôve, France; 15 cm x 20 cm) followed by compressive bandaging (**Figure 3a, 3b**). The right leg was elevated during rest. After 7 days of treatment, no new bullae developed, and the existing lesions showed reduced swelling, erythema, and pain. The patient met discharge criteria and was transitioned to outpatient management (**Figure 3c**).

## DISCUSSION

Bullous erysipelas can be considered a severe variant of erysipelas, marked by the progression of haemorrhagic bullae and tissue necrosis.[5] The condition may advance to cellulitis, cutaneous abscesses, necrotising fasciitis or even sepsis.[6] However a meticulous diagnostic approach is essential to avoid confusion with other bullous disorders. Notably, subepidermal blistering diseases such as bullous pemphigoid share clinical similarities, as both predominantly affect older adults, manifest as tense bullae on an erythematous base, and may yield negative Tzanck smear results.

Histopathological examination with hematoxylin-eosin staining distinguishes these entities: bullous pemphigoid exhibits subepidermal blistering with eosinophil predominance, whereas bullous erysipelas demon-


**Figures 2: a and b) An Urgo non-adhesive gauze dressing is applied directly over the lesion, followed by a compressive bandage to reduce oedema and stabilise the affected area. c) The lesions after 7 days of treatment: The blister roof has dried, and peeled off, leaving intact skin underneath.**

strates marked dermal oedema and a mixed inflammatory infiltrate.[7] Clinically, bullous pemphigoid often presents with symmetric, widespread lesions, while bullous erysipelas typically involves asymmetric lower limb distribution.[2]

Though useful, we did not perform bacterial cultures on the bullous fluid or blood samples, and this decision was based on two factors. First, the patient had already received empirical antibiotics prior to admission, which significantly reduces the yield of positive cultures. Second, in typical erysipelas, bacterial cultures are positive in only a minority of cases (approximately 5%) and they are generally not required for confirmation when the clinical and histopathological presentation is classic.[8]

Our patient exhibited classic features of bullous erysipelas, including age, lesion characteristics with localised to the right lower leg, and systemic symptoms, enabling a prompt clinical diagnosis. The lesions developed on the limb affected by post-stroke hemiparesis, where reduced sensory perception and susceptibility to external trauma likely predisposed the patient to infection.[9] Following diagnosis, the patient received intravenous beta-lactam antibiotic targeting Group A *Streptococcus* and *Staphylococcus aureus*, the primary pathogens implicated in erysipelas. Intravenous methylprednisolone was used as an adjunctive therapy at a moderate dose (40 mg/day) for its potent anti-inflammatory and anti-oedematous effects, aiming to rapidly reduce tissue swelling and inflammation, potentially mitigating local damage and pain. Its use in erysipelas is supported by some evidence showing reduced recovery time without increasing complications when combined with appropriate antibiotics. Although many clinicians may reasonably be concerned about the potential for corticosteroids to mask infection progression or exacerbate underlying comorbidities, their adjunctive use in our case was justified. Administered at a moderate, short-term dose under close inpatient monitoring, this approach was not associated with significant adverse effects.[10,11] Oedema due to fluid accumulation in the superficial dermis was managed with compressive bandaging and bullae aspiration to reduce swelling and prevent deeper tissue invasion. Non-adhesive Urgo gauze was used for dressing, a technique refined through years of managing blistering disorders to avoid disrupting the epidermal roof. Tense bullae were aseptically aspirated using a sterile syringe to relieve pain and reduce pressure, preserving the overlying epidermal roof. The denuded areas were cleansed with 10% povidone-iodine antiseptic solution. The primary dressing served a dual purpose: its non-adherent nature prevented disruption of the fragile blister roof during changes, and its absorbent layer managed minimal exudate. It was covered with an absorbent pad and a compressive bandage to manage oedema. Dressings were changed daily to monitor progression and for hygiene.

Clinical improvement, including resolution of bullae and reduced erythema, was observed within 7 days. This contrasts with the average hospitalisation period of 20.6 days reported by Guberman *et al*., potentially attributable to our adjunct use of systemic corticosteroids to suppress inflammation.[12] However, further studies are needed to evaluate the efficacy and safety of corticosteroids in such infections.

## CONCLUSION

This case highlights the importance of a structured diagnostic and therapeutic approach in bullous erysipelas, particularly in patients with predisposing factors like post-stroke immobility. Careful clinical and histopathological examination is essential for accurate diagnosis.

Prompt antibiotic therapy, adjunctive corticosteroids to control severe inflammation, and tailored non-adhesive wound care with compression can facilitate a rapid recovery and shorten hospital stay. It must be emphasised that corticosteroid use is adjunctive and not routine; its benefits and risks should be weighed on a case-by-case basis, and more robust studies are needed to standardise its role in managing bullous erysipelas.

## Take Home Message

- Suspect bullous erysipelas in patients with limb immobility presenting with asymmetric, haemorrhagic bullae on an erythematous base..
- Confirm with histopathology to distinguish it from other blistering diseases like bullous pemphigoid.
- Combine antibiotics with adjunctive corticosteroids to .rapidly reduce severe inflammation and shorten hospital stay..
- Use meticulous wound care including non-adhesive dressings and compression bandaging to protect the blister roof and manage oedema.

### Declarations

All the authors declared no competing interests.

### Patient Consent

Written consent was obtained from all patients for publications of the clinical details and accompanying images.

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