



International Journal of Innovative Technologies in Social Science

e-ISSN: 2544-9435

Operating Publisher
SciFormat Publishing Inc.
ISNI: 0000 0005 1449 8214

2734 17 Avenue SW,
Calgary, Alberta, T3E0A7,
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ARTICLE TITLE	ERYSIPELAS AS AN ACUTE STREPTOCOCCAL SKIN INFECTION: A LITERATURE REVIEW
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DOI	https://doi.org/10.31435/ijitss.3(51).2026.6504
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RECEIVED	17 June 2026
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ACCEPTED	14 September 2026
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PUBLISHED	29 September 2026
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ERYSIPELAS AS AN ACUTE STREPTOCOCCAL SKIN INFECTION: A LITERATURE REVIEW

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ABSTRACT

Erysipelas is an acute bacterial infection of the upper dermis and superficial lymphatic vessels. Beta hemolytic streptococci cause most typical cases. The disease often starts suddenly with painful erythema, warmth, swelling, fever, chills, and a raised or sharply demarcated skin border. Most patients respond to antibiotic therapy, but recurrent episodes can damage lymphatic drainage and create chronic edema. This narrative review summarizes current evidence on etiology, risk factors, clinical variants, diagnosis, treatment, complications, prevention, and care organization. It gives special attention to recurrent lower limb disease, because recurrence links infection with mobility loss, work absence, repeated antibiotic exposure, compression therapy, community nursing, and social support. The review also distinguishes infectious erysipelas from carcinoma erysipeloides, a malignant mimic that can delay cancer diagnosis when clinicians treat persistent erythema as infection.

KEYWORDS

Erysipelas, Cellulitis, Streptococcus pyogenes, Lymphedema, Antibiotic Prophylaxis, Social Services

CITATION

Kasperek, G., Kasperek, K. M., Obersztyn, J., Bajer, J. M., Kulawinek, H., Szczecińska, A., Glanert, W., Mliczek, A., Mrozek, P., & Czech, M. (2026). Erysipelas as an acute streptococcal skin infection: A literature review. *International Journal of Innovative Technologies in Social Science*, 3(51). [https://doi.org/10.31435/ijitss.3\(51\).2026.6504](https://doi.org/10.31435/ijitss.3(51).2026.6504)

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Introduction

Erysipelas is an acute bacterial infection of the upper dermis and superficial lymphatic vessels. It may extend into nearby subcutaneous tissue, but it usually remains more superficial than classic cellulitis. The term comes from the Greek words erythros, meaning red, and pella, meaning skin. This origin describes the visible red skin change that often leads patients to seek care. The disease usually begins suddenly. A person may feel well and then develop fever, chills, malaise, pain, and a sharply red area of skin within hours. The affected area becomes warm, swollen, tender, and tense. The border often appears raised and clearly separated from normal skin. These findings help clinicians distinguish erysipelas from other causes of painful red skin, although erysipelas and cellulitis overlap in routine practice (Raff & Kroshinsky, 2016; Stevens et al., 2014; Sullivan & de Barra, 2018).

Erysipelas remains important because antibiotic availability has not removed its clinical and social burden. Prompt therapy usually leads to recovery, but delayed assessment may lead to hospital admission, intravenous therapy, imaging, and surgical review when the diagnosis is uncertain. Recurrent disease creates a separate long term problem. Each episode may injure lymphatic vessels, worsen edema, and increase the chance of another episode. This cycle connects infection with chronic swelling, reduced mobility, work absence, repeated consultations, and long term skin care needs (Cox, 2006; Inghammar et al., 2014; Webb et al., 2020).

This review presents erysipelas as a clinical infection and as a condition that can burden patients, caregivers, primary care, hospitals, and social services. It discusses pathophysiology, risk factors, clinical presentation, diagnosis, treatment, complications, prevention, and care organization. It also explains why integrated care, community nursing, compression access, and timely follow up matter for patients with recurrent disease.

Etiology and Pathophysiology

Streptococcus pyogenes, also called group A beta hemolytic streptococcus, causes many classic cases of erysipelas. Other beta hemolytic streptococci, including groups B, C, and G, may also contribute, especially in older adults and people with chronic disease. *Staphylococcus aureus* can cause some skin and soft tissue infections, but classic non purulent erysipelas has a strong streptococcal pattern (Bisno & Stevens, 1996; Ki & Rotstein, 2008; Lazzarini et al., 2005).

Bacteria usually enter through a defect in the skin barrier. The entry point may be small and difficult to find. Common portals include interdigital fissures, tinea pedis, eczema, ulcers, surgical wounds, pressure injury, insect bites, minor trauma, and macerated skin. In the lower limb, chronic dermatomycosis of the foot increases risk because it breaks the epidermal barrier and allows streptococci to reach the dermis (Dupuy et al., 1999; Roujeau et al., 2004). Impaired venous or lymphatic drainage increases susceptibility. Venous insufficiency raises tissue pressure and promotes edema. Lymphatic damage reduces clearance of fluid, proteins, immune cells, and microbes. These changes create tissue conditions that support bacterial spread and reduce local immune control. They also explain why erysipelas often recurs in the same limb and why risk may rise after mastectomy, lymph node dissection, pelvic surgery, radiotherapy, vascular surgery, or trauma (Cox, 2006; Vignes et al., 2022; Warren et al., 2007). The inflammatory response explains the visible features. Streptococcal antigens and tissue injury activate innate immune pathways. Local vessels dilate and become more permeable. Fluid moves into the dermis. Neutrophils migrate to the infected area. Cytokines and chemokines amplify inflammation. Superficial lymphatic vessels become inflamed and congested. These processes cause redness, swelling, warmth, pain, and a raised border (Raff & Kroshinsky, 2016; Stevens et al., 2014).

Systemic symptoms can appear early because the host response is not only local. Cytokines affect temperature regulation and cause fever. Patients may develop chills, malaise, headache, nausea, or weakness. High fever is more likely when the infection spreads quickly or when comorbid disease limits host reserve (Raff & Kroshinsky, 2016; Sullivan & de Barra, 2018).

The clear border of erysipelas reflects superficial dermal and lymphatic involvement. Inflammation can spread along lymphatic channels and produce a raised edge. Deeper cellulitis often has less distinct borders. This distinction helps bedside diagnosis, but many patients show mixed features. For this reason, many clinical studies analyze erysipelas and cellulitis together (Lazzarini et al., 2005; Raff & Kroshinsky, 2016).

Epidemiology and Risk Factors

Erysipelas can affect any age group, but risk rises in older adults, neonates, young children, and immunocompromised patients. Older adults often have several overlapping risk factors, including venous disease, chronic edema, diabetes, obesity, reduced mobility, and fragile skin. Neonates and young children can become ill quickly because their immune responses and fluid reserves differ from those of adults. Immunocompromised patients may have atypical signs, severe infection, or slow recovery (Cannon et al., 2018; Mistry, 2013; Stevens et al., 2014).

Diabetes mellitus increases risk through impaired neutrophil function, vascular disease, neuropathy, and delayed wound healing. Peripheral neuropathy may reduce awareness of minor trauma. Peripheral arterial disease and foot deformity can increase skin breakdown. These mechanisms make foot care, early treatment of small lesions, and glycemic control important parts of prevention (Ki & Rotstein, 2008; Mistry, 2013).

Obesity is a consistent risk factor for recurrence. It increases lower limb edema, venous pressure, skin folds, sweating, friction, and difficulty with hygiene. It can also make compression therapy harder to fit and use. Studies of recurrent erysipelas and cellulitis link obesity with repeated episodes, especially when chronic edema is present (Brishkoska-Boshkovski et al., 2019; Inghammar et al., 2014; Sapała et al., 2020).

Chronic venous insufficiency and chronic edema have major clinical importance. Edema stretches the skin, weakens the barrier, slows oxygen diffusion, and impairs immune cell movement. Chronic edema may result from venous disease, lymphedema, heart failure, kidney disease, immobility, obesity, or surgery. A randomized trial showed that compression therapy reduced recurrence in patients with chronic leg edema and recurrent cellulitis, which supports edema control as a prevention strategy (Webb et al., 2020).

Impaired lymphatic drainage is one of the strongest recurrence risks. Secondary lymphedema may follow breast cancer surgery, axillary lymph node dissection, pelvic surgery, lymphadenectomy, radiotherapy, vascular bypass, trauma, or cancer treatment. Primary lymphedema may also predispose to infection. Once erysipelas occurs, inflammation can further damage lymphatic vessels, so risk and outcome reinforce each other (Burian et al., 2024; Vignes et al., 2022; Warren et al., 2007).

Tinea pedis and other fungal infections deserve attention because they are common, treatable, and often missed. Interdigital fissures allow bacteria to enter the skin. Treatment of fungal infection and maintenance of dry interdigital skin may reduce recurrence risk. Skin ulcers and chronic wounds create similar entry points and require routine wound care (Dupuy et al., 1999; Roujeau et al., 2004).

Alcohol use disorder can increase risk through poor nutrition, reduced immune function, liver disease, trauma, poor hygiene, and delayed care seeking. It may also reduce adherence to antibiotics, compression, and follow up visits. In practice, alcohol related risk may overlap with poverty, unstable housing, and limited access to primary care. These factors can increase the clinical burden even when the biological infection is treatable (Cannon et al., 2018; Quirke et al., 2017).

Case control and cohort studies support a consistent risk pattern. Local trauma, toe web lesions, venous insufficiency, edema, obesity, and previous episodes increase risk, although the measured strength of each factor differs between populations (Bjornsdottir et al., 2005; Dupuy et al., 1999; Halpern et al., 2008; Karppelin et al., 2010; Quirke et al., 2017).

Clinical Presentation

The clinical picture often develops quickly. Patients report acute pain, tenderness, warmth, swelling, and redness in a defined area of skin. The lesion may look irregular, but the border is usually sharp. The surface may appear shiny and tense. The edge may be raised. Regional lymph nodes may become tender, and red streaks may indicate lymphangitis (Raff & Kroshinsky, 2016; Stevens et al., 2014).

The lower limbs and face are common sites. Lower limb erysipelas often occurs in patients with edema, venous insufficiency, diabetes, obesity, tinea pedis, or previous episodes. Facial erysipelas may involve the cheeks, nose, and periorbital region. Facial disease requires careful assessment when swelling approaches the orbit or when the patient has severe systemic symptoms (Lazzarini et al., 2005; Raff & Kroshinsky, 2016).

Systemic symptoms are common. Patients may have chills, fever, malaise, headache, nausea, or weakness. In severe cases, fever may reach 40 to 41 degrees Celsius. Older adults may present with confusion, falls, poor intake, or functional decline rather than a detailed symptom history. Immunosuppressed patients may have less fever despite serious disease (Cannon et al., 2018; Mistry, 2013).

Several clinical variants need recognition. Bullous erysipelas includes blister formation and may reflect intense local inflammation. Hemorrhagic erysipelas includes purpura or bleeding into blisters. Necrotic erysipelas includes tissue death and needs urgent reassessment because necrosis may indicate severe local compromise or a different diagnosis, such as necrotizing infection. Bullous forms may involve additional pathogens or more complicated clinical courses, so clinicians should assess severity, purulence, and risk factors carefully (Krasagakis et al., 2006; Stevens et al., 2014).

Erysipelas migrans describes spreading or migrating erythematous inflammation. The active border can move across adjacent skin as lymphatic inflammation extends. This pattern can confuse diagnosis if the first lesion begins to improve while a new edge appears nearby.

Recurrent erysipelas often affects the same anatomical region. Recurrent inflammation can thicken the skin, worsen edema, and damage lymphatic vessels. Patients may report that the limb never returns fully to normal between episodes. This history changes management because the clinician must treat the infection and also address edema, skin care, fungal infection, footwear, compression, and access to follow up (Inghammar et al., 2014; Ong et al., 2022; Vignes et al., 2022).

Clinical series show that erysipelas and cellulitis often overlap in routine care. Patients may have classic sharply demarcated erysipelas, deeper cellulitis, or mixed features. This explains why many treatment studies use the broader term skin and soft tissue infection or combine erysipelas with cellulitis (Ki & Rotstein, 2008; Lazzarini et al., 2005; Morris, 2008).

Diagnosis and Differential Diagnosis

Clinicians usually diagnose erysipelas at the bedside. The key findings are acute onset, painful erythema, warmth, swelling, clear borders, and systemic symptoms. Laboratory tests may support the diagnosis, but they do not confirm it. White blood cell count and C reactive protein may rise, but these markers are not specific (Raff & Kroshinsky, 2016; Sullivan & de Barra, 2018).

Blood cultures have limited yield in uncomplicated erysipelas. They are more useful when the patient has severe systemic illness, sepsis, immunosuppression, unusual exposure, recurrent unexplained episodes, or suspected bacteremia. Swabs from intact skin are usually not helpful. Culture may help when an open wound, ulcer, abscess, or purulent drainage is present (Stevens et al., 2014; Sullivan & de Barra, 2018).

Imaging is not routine, but it has value in selected cases. Ultrasound can assess suspected abscess or deep vein thrombosis. CT or MRI may be needed when pain is severe, the patient is toxic, infection spreads fast, gas is suspected in tissue, or necrotizing infection is possible. Imaging should not delay surgery when necrotizing infection is clinically likely (Stevens et al., 2014).

Erysipelas usually affects the upper dermis and superficial lymphatics. It often has raised and clear borders. Cellulitis often involves the deeper dermis and subcutaneous tissue and may have less distinct borders. Many studies combine the two because clinical overlap is common and microbiological confirmation is often unavailable (Lazzarini et al., 2005; Raff & Kroshinsky, 2016).

Important differential diagnoses include deep vein thrombosis, venous stasis dermatitis, contact dermatitis, allergic reaction, gout, septic arthritis, herpes zoster, necrotizing fasciitis, abscess, and inflammatory breast cancer. Deep vein thrombosis may cause swelling, pain, and warmth, but it usually lacks a raised red border and high fever. Venous stasis dermatitis is often chronic or bilateral and may include scaling, pigmentation, and itch. Contact dermatitis often has itch and an exposure history. Gout and septic arthritis focus on a joint and require joint assessment (Phoenix et al., 2012; Raff & Kroshinsky, 2016).

Necrotizing fasciitis is the most urgent differential diagnosis. Pain out of proportion, rapid progression, skin anesthesia, bullae, crepitus, shock, or severe systemic toxicity should prompt emergency surgical evaluation. A missed diagnosis can be fatal (Stevens et al., 2014).

Carcinoma erysipeloides, also called erysipelas carcinomatosum in some texts, is not infectious erysipelas. It is an inflammatory skin manifestation of cancer spread through dermal lymphatics or metastatic infiltration. It may mimic erysipelas because the skin becomes red and swollen. Fever and chills are often absent. Antibiotics usually do not produce the expected response. Clinicians should reconsider the diagnosis when erythema persists, recurs in an unusual pattern, appears near a known malignancy, or lacks systemic infectious signs (Al Ameer et al., 2015; Carlesimo et al., 2009; Nava et al., 2009; Shien et al., 2014; Sundheimer & Henghold, 2019).

Diagnostic error has direct cost. Dermatology consultation can change management when cellulitis is overdiagnosed, and misdiagnosis may expose patients to unnecessary antibiotics, avoidable admission, and missed treatment of mimics such as venous dermatitis (Li et al., 2018; Weng et al., 2017). Emergency clinicians also need to identify patients at risk of oral treatment failure, because early review can prevent delayed escalation when outpatient therapy is not enough (Phoenix et al., 2012; Yadav et al., 2015).

Treatment and Pharmacotherapy

Treatment has two aims. The first aim is to control the acute bacterial infection. The second aim is to reduce future episodes by treating modifiable risk factors. Antibiotic choice should cover streptococci in typical non purulent erysipelas. The route depends on severity, comorbidity, oral tolerance, and signs of systemic illness (Stevens et al., 2014; Sullivan & de Barra, 2018).

Mild to moderate cases can often be treated with oral therapy. Penicillin remains a traditional first line option when streptococcal erysipelas is likely. Amoxicillin is another common oral option because it has activity against streptococci and is simple to use. Some clinicians use amoxicillin with clavulanic acid when broader coverage is justified, such as after bites, selected wound related infections, polymicrobial risk, or concern about mixed flora. Broad coverage should not replace careful diagnosis and risk assessment (Raff & Kroshinsky, 2016; Stevens et al., 2014).

Severe erysipelas may require intravenous therapy and hospital care. Indications include sepsis, hemodynamic instability, rapidly spreading infection, severe pain, facial infection with orbital concern, inability to take oral medication, immunosuppression, serious comorbidity, or failure of outpatient therapy. Intravenous penicillin or cefazolin may be suitable in many streptococcal cases. Broader therapy may be required if necrotizing infection, polymicrobial infection, or resistant pathogens are suspected (Stevens et al., 2014).

Dosing frequency depends on the drug, formulation, kidney function, patient age, and clinical setting. Some regimens use dosing every 12 hours, while others use dosing every 8 hours or more often. Amoxicillin

with clavulanic acid can be prescribed every 8 or 12 hours depending on formulation and local policy. Exact dosing should follow local guidelines, renal function, disease severity, allergy history, and antimicrobial stewardship policy (Stevens et al., 2014; Walsh et al., 2016).

Penicillin allergy requires careful classification. Type I allergy includes anaphylaxis, angioedema, bronchospasm, or immediate urticaria. These patients should avoid penicillins unless specialist evaluation supports safe use. Alternatives may include macrolides or clindamycin, but local resistance patterns matter. Type IV delayed skin reactions differ from immediate allergy. Some patients with mild delayed rash may tolerate selected cephalosporins, but clinicians should assess the reaction history and follow local guidance (Stevens et al., 2014).

Local care supports systemic therapy. Limb elevation may reduce swelling and pain. Analgesia improves mobility and sleep. Hydration and fever control support recovery. Tinea pedis, ulcers, eczema, and fissures require treatment because they may be entry points. Compression therapy should usually wait until acute pain and intense inflammation improve, but long term edema control is important. Ichthammol dressings or compresses may be used as supportive local care in some settings, but they do not replace systemic antibiotics (Raff & Kroshinsky, 2016; Webb et al., 2020).

Recurrent erysipelas, also called erysipelas recidivans, needs prevention. Prophylactic penicillin can reduce recurrence during prophylaxis in selected patients with repeated episodes, although benefit may decrease after prophylaxis stops. Prevention works best when antibiotics are combined with edema management, fungal treatment, skin care, weight management, and venous disease care (Dalal et al., 2017; Oh et al., 2014; Thomas et al., 2012; Thomas et al., 2013). Table 1 summarizes treatment options. The table does not replace local guidelines.

Evidence for prophylaxis comes from randomized trials and systematic reviews. The PATCH studies reported benefit from low dose penicillin during prophylaxis, and later reviews found that antibiotic prophylaxis reduces recurrence while treatment continues. These data support prophylaxis for selected patients, but they also show why clinicians must treat edema and skin entry points instead of relying only on repeated antibiotic courses (Dalal et al., 2017; Oh et al., 2014; Thomas et al., 2012; Thomas et al., 2013).

Table 1. Pharmacological treatment options for erysipelas and recurrent erysipelas

Clinical scenario	Drug choice	Dosage and frequency	Rationale	Important cautions
First line therapy for mild to moderate non purulent erysipelas	Oral penicillin V or oral amoxicillin	Use local adult or pediatric dosing. Many oral regimens use two or three daily doses depending on the drug and formulation.	Covers beta hemolytic streptococci, which cause most classic non purulent erysipelas.	Check allergy history, kidney function, adherence, and response after 48 to 72 hours.
Severe erysipelas requiring hospital care	Intravenous penicillin G or cefazolin. Broaden therapy if sepsis, necrosis, bite injury, or mixed infection is suspected.	Use hospital protocols. Intravenous beta lactams often require dosing every 6 to 8 hours, with renal adjustment when needed.	Provides reliable blood and tissue levels in severe disease and allows close monitoring.	Assess for necrotizing infection, bacteremia, orbital involvement, and need for surgical review.
Second line therapy if there is no clinical response	Reassess diagnosis first. Consider amoxicillin with clavulanic acid, cefazolin, ceftriaxone, or broader therapy based on findings.	Frequency may be every 8 or 12 hours for amoxicillin with clavulanic acid depending on formulation and local policy.	Poor response may reflect wrong diagnosis, abscess, resistant pathogen, inadequate dose, non adherence, or deep infection.	Do not broaden antibiotics automatically without reassessing abscess, thrombosis, venous dermatitis, necrosis, and adherence.
Alternative for Type I penicillin allergy, including anaphylaxis	Clindamycin, clarithromycin, azithromycin, or another locally accepted non beta lactam option.	Use guideline based dosing. Frequency varies by drug, for example once daily for azithromycin or multiple daily doses for clindamycin.	Avoids penicillins in immediate allergy while maintaining streptococcal coverage.	Macrolide and clindamycin resistance vary by region. Clindamycin increases the risk of Clostridioides difficile infection.

Clinical scenario	Drug choice	Dosage and frequency	Rationale	Important cautions
Alternative for Type IV penicillin allergy, including delayed skin reaction	Selected cephalosporins may be considered after risk assessment. Macrolide or clindamycin may be used when beta lactams should be avoided.	Use local dosing guidance and adjust for kidney function where needed.	Many delayed mild rashes do not carry the same risk as anaphylaxis, but the allergy history must be clear.	Avoid cephalosporins in severe delayed reactions such as Stevens Johnson syndrome, toxic epidermal necrolysis, or DRESS.
Treatment when mixed flora is suspected	Amoxicillin with clavulanic acid, ampicillin with sulbactam, or another regimen guided by exposure and severity.	Often every 8 or 12 hours for oral amoxicillin with clavulanic acid depending on formulation.	Covers streptococci plus selected gram negative and anaerobic organisms when wounds, bites, or polymicrobial infection are plausible.	Use broad spectrum therapy only when the clinical setting justifies it. Narrow therapy when possible.
Treatment when MRSA risk is clinically relevant	Add or choose MRSA active therapy according to local policy, such as doxycycline, trimethoprim sulfamethoxazole, clindamycin, vancomycin, or linezolid in selected cases.	Depends on drug, severity, route, kidney function, and local resistance.	MRSA is not the usual cause of classic non purulent erysipelas, but risk rises with purulence, previous MRSA, injection drug use, or severe infection.	Some MRSA active drugs have weak streptococcal activity. Ensure streptococcal coverage when needed.
Prophylaxis for recurrent erysipelas, also called erysipelas recidivans	Oral penicillin V or intramuscular benzathine penicillin in selected patients. Alternatives depend on allergy and resistance.	Trial regimens vary. Oral penicillin V has been used twice daily. Intramuscular benzathine penicillin is often given every 2 to 4 weeks.	Reduces recurrence during prophylaxis in selected patients, especially when non purulent streptococcal disease is likely.	Use with risk factor control. Benefit may decrease after stopping prophylaxis. Monitor adherence, adverse effects, and recurrence.
Supportive local treatment and skin care	Analgesia, limb elevation, hydration, skin emollients, treatment of tinea pedis, wound care, and selected local dressings such as ichthammol where used.	Apply according to local practice and patient tolerance.	Reduces pain, supports skin barrier repair, and treats entry points.	Local therapy does not replace systemic antibiotics in true erysipelas. Avoid irritant products on inflamed skin.
Management of edema and lymphedema after the acute phase	Compression garments, edema assessment, physiotherapy when indicated, skin care, and referral to lymphedema services.	Use daily compression after acute pain and inflammation allow safe fitting and wear.	Reduces chronic edema, which is a major driver of recurrence.	Avoid poorly fitted compression. Assess arterial supply when clinically indicated.

Drug selection and dosing must follow local protocols, renal function, severity, age, allergy history, pregnancy status, and antimicrobial stewardship policy. The table summarizes general options from peer reviewed clinical guidance and trials (Dalal et al., 2017; Stevens et al., 2014; Thomas et al., 2013; Webb et al., 2020).

Socio Economic Impact and Healthcare Management

Erysipelas often appears as a short acute infection, but its social effects can be large. Pain, fever, and lower limb swelling can stop a patient from walking, standing, driving, or working. People with manual jobs, caregiving roles, or work that requires prolonged standing may lose income during each episode. Even a short episode may create financial stress if the patient lacks paid sick leave or flexible work conditions. These effects are most visible in recurrent lower limb disease, where infection interacts with chronic edema and reduced mobility (Cannon et al., 2018; Webb et al., 2023; Weng et al., 2017).

Families may carry a practical burden. A patient with painful leg erysipelas may need help with meals, hygiene, transport, medication collection, dressing changes, and child care. Older adults may need temporary home care. Patients who live alone may delay seeking care because they cannot arrange transport. Delay can increase the chance of emergency assessment or admission (Phoenix et al., 2012; Weng et al., 2017).

Primary care carries much of the management burden. General practitioners assess red legs, distinguish infection from venous dermatitis or thrombosis, choose antibiotics, monitor response, and arrange follow up. Emergency departments see patients with fever, rapid spread, diagnostic uncertainty, or treatment failure. Hospitals admit patients who need intravenous therapy, monitoring, imaging, or surgical review. Wound care clinics and community nurses manage ulcers, dressings, compression, and skin protection (Li et al., 2018; Zervos et al., 2012).

Recurrent disease increases healthcare use. Repeated episodes lead to repeated consultations, laboratory tests, prescriptions, medical certificates, emergency visits, and admissions. The patient may receive many antibiotic courses over time. Some patients need prophylaxis for weeks or months, and some require specialist supervision. This raises antimicrobial stewardship concerns. Clinicians must treat true bacterial infection effectively, but they should avoid unnecessary broad spectrum therapy and repeated antibiotics without prevention of risk factors (Ong et al., 2022; Stevens et al., 2014; Walsh et al., 2016).

Chronic lymphedema creates ongoing costs. Patients may need compression garments, fitting visits, physiotherapy, skin care products, wound care supplies, and regular review. Compression garments may require replacement and may not be fully reimbursed in all systems. Poor access to compression can increase recurrence and hospital use. Economic analyses suggest that compression therapy can reduce recurrent cellulitis events and may save costs in patients with chronic edema and previous cellulitis (Webb et al., 2020; Webb et al., 2023).

Quality of life may decline after repeated episodes. Chronic swelling affects body image, clothing, footwear, mobility, sleep, and physical activity. Fear of another infection may reduce travel and social participation. Patients may avoid walking because of pain or swelling, but reduced activity may worsen edema and weight. This creates a cycle that medical care alone may not break (Vignes et al., 2022; Warren et al., 2007).

Healthcare management should be coordinated. Dermatology can support diagnosis and skin barrier care. Infectious disease specialists can advise on recurrent episodes and prophylaxis. Vascular medicine can assess venous disease. Diabetology can improve foot risk and glycemic control. Rehabilitation and physiotherapy can support mobility and edema care. Community nurses can monitor skin and help with dressings and compression. Social workers can address transport, home support, income loss, and access to supplies (Li et al., 2018; Webb et al., 2020).

Prevention may reduce costs more effectively than repeated acute care. Treating tinea pedis, improving skin care, controlling edema, funding compression, and educating patients may reduce recurrence. Digital reminders can support antibiotic adherence, compression use, and follow up visits. Telemedicine may help monitor wound healing or early recurrence in stable patients with transport barriers. These tools should support clinical judgment and should not replace direct examination when severe infection is possible (Ong et al., 2022; Webb et al., 2023).

The IJITSS context is relevant because erysipelas connects infection with inequality and service design. A patient with transport, stable housing, paid leave, and compression access can prevent recurrence more easily than a patient without these resources. Health policy can support prevention by funding compression garments, community nursing, foot care for diabetic patients, wound services, and rapid primary care access. These measures may reduce avoidable system costs and patient dependence (Webb et al., 2023; Weng et al., 2017).

Complications

Most patients recover with timely antibiotic therapy. Complications occur when infection is severe, treatment is delayed, host defenses are impaired, or risk factors persist. Recurrent erysipelas is the most common long term problem. It can create a cycle of inflammation, lymphatic injury, chronic edema, and new infection (Cox, 2006; Inghammar et al., 2014; Vignes et al., 2022). Chronic lymphedema may follow repeated episodes. The limb becomes swollen, heavy, and prone to skin thickening. In severe long standing cases, elephantiasis can develop. Elephantiasis describes marked enlargement and skin change caused by lymphatic failure. It can limit walking, footwear use, hygiene, and work ability (Warren et al., 2007). Lymphangitis and lymphadenitis may occur during acute infection. The patient may have red streaks along lymphatic channels and tender regional lymph nodes. These signs indicate lymphatic involvement and may contribute to future lymphatic dysfunction (Raff & Kroshinsky, 2016; Stevens et al., 2014).

Phlegmon describes diffuse soft tissue inflammation that may extend beyond superficial erysipelas. Abscess is less typical in classic non purulent erysipelas, but it must be considered when there is fluctuance, purulence, focal severe tenderness, or poor response to antibiotics. Ultrasound can help identify a drainable collection (Stevens et al., 2014; Sullivan & de Barra, 2018).

Deep vein thrombosis is both a differential diagnosis and a concern in immobile patients with swollen painful legs. Clinicians should assess thrombotic risk and use ultrasound when the clinical picture is uncertain. Superficial thrombophlebitis can also coexist with skin infection (Raff & Kroshinsky, 2016). Sepsis and bacteremia are severe systemic outcomes. They are more likely in frail patients, immunocompromised patients, and those with delayed care. Hypotension, confusion, tachypnea, oliguria, or rapid decline require urgent assessment. Necrotizing soft tissue infection is rare but must remain in the differential diagnosis because it requires emergency surgery (Stevens et al., 2014; Zervos et al., 2012). Facial erysipelas can have specific risks. Infection near the eye can threaten orbital structures. Severe infection in the central face can rarely spread through venous channels and contribute to cavernous sinus thrombosis. This outcome is uncommon, but it is serious and requires urgent multidisciplinary care (Stevens et al., 2014).

Carcinoma erysipeloides deserves separate mention because it is not a complication of infection. It is a cancer related mimic. Patients may show erythema and swelling without fever or chills. Lack of antibiotic response should prompt reconsideration of the diagnosis, especially when the patient has a known malignancy or persistent skin changes near the breast, chest wall, or surgical site (Al Ameer et al., 2015; Carlesimo et al., 2009; Nava et al., 2009; Sundheimer & Henghold, 2019).

Prevention and Long Term Management

Prevention starts with the skin barrier. Patients should keep skin clean, dry interdigital spaces carefully, moisturize dry skin, and treat fissures early. Tinea pedis should be treated with topical or systemic antifungal therapy when indicated. Eczema and dermatitis should be controlled because scratching and cracks create bacterial entry points (Dupuy et al., 1999; Roujeau et al., 2004).

Edema control is central. Patients with chronic venous disease or lymphedema need assessment for compression therapy after the acute episode improves. Compression should be fitted correctly and used consistently. Education matters because some patients stop compression when it feels uncomfortable or difficult to apply. Devices that help apply stockings may improve adherence in older adults or patients with limited hand strength (Webb et al., 2020; Webb et al., 2023).

Diabetes care reduces skin infection risk by improving wound healing and reducing foot complications. Patients need foot inspection, nail care, appropriate footwear, and rapid attention to blisters or ulcers. Weight management may reduce edema and skin friction, but it requires long term support and should not be framed as a simple instruction (Ki & Rotstein, 2008; Quirke et al., 2017).

Venous disease management may include compression, leg elevation, exercise, ulcer treatment, and referral to vascular services when needed. Mobility matters because calf muscle activity supports venous return. Rehabilitation can help patients who walk less after repeated infections (Cox, 2006; Webb et al., 2020).

Patient education should be practical. Patients should know early warning signs, including new redness, warmth, swelling, pain, chills, fever, rapid spread, blisters, confusion, or worsening general condition. They should know when to contact primary care and when to seek emergency help. Patients with recurrent disease may benefit from written action plans and rapid review routes (Ong et al., 2022; Phoenix et al., 2012).

Prophylactic antibiotics are appropriate for selected patients with repeated non purulent episodes after modifiable risk factors have been addressed. Oral penicillin V and intramuscular benzathine penicillin are

common approaches in studies and practice. The decision should consider recurrence frequency, severity, allergy history, adherence, local resistance, and patient preference (Dalal et al., 2017; Oh et al., 2014; Thomas et al., 2013).

Long term lymphedema care can improve function when it combines compression, education, exercise, skin care, and specialist input. Modern cohorts show that cellulitis remains common in primary and secondary lymphedema, which supports regular skin review and rapid access to treatment (Burian et al., 2024; Ko et al., 1998; Vignes et al., 2022).

Follow up after hospitalization should not focus only on whether redness improved. It should identify the entry point, assess edema, review footwear, screen for tinea pedis, check diabetes control, review vascular risk, and plan compression or wound care. This approach reduces the chance that the next episode will be treated as a new isolated event (Ong et al., 2022; Webb et al., 2020).

Relevance to Social Services and Health Policy

Erysipelas fits the scope of social services and health policy because it shows how an acute infection can reveal gaps in care. The patient may need antibiotics, but recovery also depends on transport, home support, nursing care, access to dressings, compression garments, and ability to attend follow up. These needs fall between medical treatment and social support (Li et al., 2018; Webb et al., 2023; Weng et al., 2017).

Integrated care pathways can improve management. A clear pathway can guide primary care clinicians on diagnosis, red flags, antibiotic selection, referral, and follow up. It can also link patients with chronic edema to compression services and community nursing. Patients with diabetes can be connected with foot care. Patients with recurrent admissions can receive case review (Ong et al., 2022; Webb et al., 2020).

Community nurses have a practical role. They can observe skin changes, support dressing changes, check adherence, teach skin care, and identify social barriers. For older adults, nurses may also notice falls, poor nutrition, poor living conditions, or inability to use compression. These observations can trigger social service involvement.

Digital follow up may help selected patients. Secure photo monitoring can document whether erythema is spreading or improving. Electronic reminders can support antibiotic adherence and compression use. Teleconsultation may reduce travel for stable patients. These tools are less suitable when the patient has severe pain, systemic symptoms, facial involvement, or suspected necrotizing infection. In such cases, direct assessment is needed (Phoenix et al., 2012; Stevens et al., 2014).

Public health education can target groups with higher risk, including patients with diabetes, chronic edema, obesity, venous insufficiency, and prior erysipelas. Education should be simple and practical. It should explain foot care, fungal infection treatment, skin breaks, edema, and early medical contact (Dupuy et al., 1999; Quirke et al., 2017).

Social policy can reduce recurrence by funding preventive resources. Compression garments, wound care products, podiatry, transport support, and home nursing may cost less than repeated admissions. Policy should also recognize that patients with low income may not be able to purchase skin care products or compression garments. Prevention must be accessible to be effective (Webb et al., 2023; Weng et al., 2017; Zervos et al., 2012).

Conclusions

Erysipelas is an acute streptococcal infection of the skin and superficial lymphatic vessels. It usually presents with rapid onset of painful redness, warmth, swelling, clear borders, and systemic symptoms. *Streptococcus pyogenes* remains the main pathogen in classic disease, and small breaks in the skin barrier often provide the entry point.

The disease is usually treatable with appropriate antibiotics, but recurrence changes its meaning. Recurrent erysipelas can worsen lymphatic damage, deepen chronic edema, reduce mobility, and increase patient dependence on healthcare and social support. Risk factors such as lymphedema, chronic edema, venous disease, obesity, diabetes, fungal infection, wounds, and impaired access to care need direct attention.

Good management combines early diagnosis, appropriate antibiotic therapy, skin care, edema control, treatment of entry points, patient education, and follow up. Selected patients may need antibiotic prophylaxis. Many patients also need compression therapy, nursing support, rehabilitation, or social assistance. Erysipelas therefore belongs not only to infectious disease and dermatology, but also to public health, healthcare management, and social policy.

Ethics approval: Not applicable. This review does not involve human participants, animal subjects, or patient level data.

Conflict of interest: No conflicts of interest to declare.

Funding: No external funding was received.

Author contribution: The authors conceptualized, wrote, and revised the manuscript.

Data availability: No new datasets were generated or analyzed.

Use of artificial intelligence: Language editing and structural assistance were supported by an AI tool. The authors reviewed, verified, and approved the final content of the manuscript.

REFERENCES

1. Al Ameer, A., Al Nemer, A., Alharbi, A., Sait, K., Alsaif, F., & Alkaff, T. (2015). Carcinoma erysipeloïdes as a presenting feature of breast carcinoma: A case report and brief review of literature. *Case Reports in Oncology*, 8(3), 396–401. <https://doi.org/10.1159/000441338>
2. Bisno, A. L., & Stevens, D. L. (1996). Streptococcal infections of skin and soft tissues. *The New England Journal of Medicine*, 334(4), 240–245. <https://doi.org/10.1056/NEJM199601253340407>
3. Bjornsdottir, S., Gottfredsson, M., Thorisdottir, A. S., Gunnarsson, G. B., Rikardsdottir, H., Kristjansson, M., & Hilmarsson, I. (2005). Risk factors for acute cellulitis of the lower limb: A prospective case control study. *Clinical Infectious Diseases*, 41(10), 1416–1422. <https://doi.org/10.1086/497127>
4. Brishkoska-Boshkovski, V., Kondova-Topuzovska, I., Damevska, K., Petrov, A., & Dimovski, A. (2019). Comorbidities as risk factors for acute and recurrent erysipelas. *Open Access Macedonian Journal of Medical Sciences*, 7(6), 937–942. <https://doi.org/10.3889/oamjms.2019.214>
5. Burian, E. A., Karlsmark, T., Franks, P. J., Keeley, V., Moffatt, C. J., & Quéré, I. (2024). Factors associated with cellulitis in lymphoedema of the arm: An international cross sectional study. *BMC Infectious Diseases*, 24, 103. <https://doi.org/10.1186/s12879-023-08839-z>
6. Cannon, J., Dyer, J., Carapetis, J., & Manning, L. (2018). Epidemiology and risk factors for recurrent severe lower limb cellulitis: A longitudinal cohort study. *Clinical Microbiology and Infection*, 24(10), 1084–1088. <https://doi.org/10.1016/j.cmi.2018.01.018>
7. Carlesimo, M., Rossi, A., La Pietra, M., Cacchi, C., Feliziani, G., Mari, E., & Narcisi, A. (2009). Erysipelas like carcinoma of the breast: A case report and review of the literature. *Dermatology Online Journal*, 15(3), 12.
8. Cox, N. H. (2006). Oedema as a risk factor for multiple episodes of cellulitis or erysipelas of the lower leg: A series with community follow up. *British Journal of Dermatology*, 155(5), 947–950. <https://doi.org/10.1111/j.1365-2133.2006.07419.x>
9. Dalal, A., Eskin-Schwartz, M., Mimouni, D., Ray, S., Days, W., Hodak, E., Leibovici, L., & Paul, M. (2017). Interventions for the prevention of recurrent erysipelas and cellulitis. *Cochrane Database of Systematic Reviews*, 2017(6), CD009758. <https://doi.org/10.1002/14651858.CD009758.pub2>
10. Dupuy, A., Benchikhi, H., Roujeau, J. C., Bernard, P., Vaillant, L., Chosidow, O., Sassolas, B., Guillaume, J. C., Grob, J. J., & Bastuji-Garin, S. (1999). Risk factors for erysipelas of the leg (cellulitis): Case-control study. *BMJ*, 318(7198), 1591–1594. <https://doi.org/10.1136/bmj.318.7198.1591>
11. Halpern, J., Holder, R., & Langford, N. J. (2008). Ethnicity and other risk factors for acute lower limb cellulitis: A UK based prospective case control study. *British Journal of Dermatology*, 158(6), 1288–1292. <https://doi.org/10.1111/j.1365-2133.2008.08489.x>
12. Inghammar, M., Rasmussen, M., & Linder, A. (2014). Recurrent erysipelas: Risk factors and clinical presentation. *BMC Infectious Diseases*, 14, 270. <https://doi.org/10.1186/1471-2334-14-270>
13. Karpelin, M., Siljander, T., Vuopio-Varkila, J., Kere, J., Huhtala, H., Vuento, R., & Syrjänen, J. (2010). Factors predisposing to acute and recurrent bacterial non necrotizing cellulitis in hospitalized patients: A prospective case control study. *Clinical Microbiology and Infection*, 16(6), 729–734. <https://doi.org/10.1111/j.1469-0691.2009.02906.x>
14. Ki, V., & Rotstein, C. (2008). Bacterial skin and soft tissue infections in adults: A review of their epidemiology, pathogenesis, diagnosis, treatment and site of care. *Canadian Journal of Infectious Diseases and Medical Microbiology*, 19(2), 173–184. <https://doi.org/10.1155/2008/846453>

15. Ko, D. S. C., Lerner, R., Klose, G., & Cosimi, A. B. (1998). Effective treatment of lymphedema of the extremities. *Archives of Surgery*, 133(4), 452–458. <https://doi.org/10.1001/archsurg.133.4.452>
16. Krasagakis, K., Samonis, G., Maniatakis, P., Georgala, S., & Tosca, A. D. (2006). Bullous erysipelas: Clinical presentation, staphylococcal involvement and methicillin resistance. *Dermatology*, 212(1), 31–35. <https://doi.org/10.1159/000089019>
17. Lazzarini, L., Conti, E., Tositti, G., & de Lalla, F. (2005). Erysipelas and cellulitis: Clinical and microbiological spectrum in an Italian tertiary care hospital. *Journal of Infection*, 51(5), 383–389. <https://doi.org/10.1016/j.jinf.2004.12.010>
18. Li, D. G., Xia, F. D., Khosravi, H., Dewan, A. K., Pallin, D. J., Baugh, C. W., & Kupper, T. S. (2018). Outcomes of early dermatology consultation for inpatients diagnosed with cellulitis. *JAMA Dermatology*, 154(5), 537–543. <https://doi.org/10.1001/jamadermatol.2017.6197>
19. Mistry, R. D. (2013). Skin and soft tissue infections. *Pediatric Clinics of North America*, 60(5), 1063–1082. <https://doi.org/10.1016/j.pcl.2013.06.011>
20. Morris, A. D. (2008). Cellulitis and erysipelas. *Clinical Evidence*, 2008, 1708.
21. Nava, G., Greer, K., Patterson, J., & Lin, K. Y. (2009). Metastatic cutaneous breast carcinoma: A case report and review of the literature. *Canadian Journal of Plastic Surgery*, 17(1), 25–27. <https://doi.org/10.1177/229255030901700105>
22. Oh, C. C., Ko, H. C., Lee, H. Y., Safdar, N., Maki, D. G., & Chlebicki, M. P. (2014). Antibiotic prophylaxis for preventing recurrent cellulitis: A systematic review and meta analysis. *Journal of Infection*, 69(1), 26–34. <https://doi.org/10.1016/j.jinf.2014.02.011>
23. Ong, B. S., Dotel, R., & Tran, S. (2022). Recurrent cellulitis: Who is at risk and how effective is antibiotic prophylaxis? *International Journal of General Medicine*, 15, 6561–6572. <https://doi.org/10.2147/IJGM.S355955>
24. Phoenix, G., Das, S., & Joshi, M. (2012). Diagnosis and management of cellulitis. *BMJ*, 345, e4955. <https://doi.org/10.1136/bmj.e4955>
25. Quirke, M., Ayoub, F., McCabe, A., Boland, F., Smith, B., O'Sullivan, R., & Wakai, A. (2017). Risk factors for nonpurulent leg cellulitis: A systematic review and meta-analysis. *British Journal of Dermatology*, 177(2), 382–394. <https://doi.org/10.1111/bjd.15186>
26. Raff, A. B., & Kroshinsky, D. (2016). Cellulitis: A review. *JAMA*, 316(3), 325–337. <https://doi.org/10.1001/jama.2016.8825>
27. Roujeau, J. C., Sigurgeirsson, B., Korting, H. C., Kerl, H., & Paul, C. (2004). Chronic dermatomycoses of the foot as risk factors for acute bacterial cellulitis of the leg: A case control study. *Dermatology*, 209(4), 301–307. <https://doi.org/10.1159/000080853>
28. Sapuła, M., Krankowska, D., Wiercińska-Drapała, A., & Kuchar, E. (2020). In search of risk factors for recurrent erysipelas and cellulitis of the lower limb: A cross sectional study of epidemiological characteristics of patients hospitalized due to skin and soft tissue infections. *Interdisciplinary Perspectives on Infectious Diseases*, 2020, 1307232. <https://doi.org/10.1155/2020/1307232>
29. Shien, T., Shimizu, C., Akashi-Tanaka, S., Hojo, T., & Kinoshita, T. (2014). Clinicopathological features of erysipeloid carcinoma of the breast. *Breast Cancer*, 21(3), 297–302. <https://doi.org/10.1007/s12282-012-0382-4>
30. Stevens, D. L., Bisno, A. L., Chambers, H. F., Dellinger, E. P., Goldstein, E. J. C., Gorbach, S. L., Hirschmann, J. V., Kaplan, S. L., Montoya, J. G., & Wade, J. C. (2014). Practice guidelines for the diagnosis and management of skin and soft tissue infections: 2014 update by the infectious diseases society of america. *Clinical Infectious Diseases*, 59(2), e10–e52. <https://doi.org/10.1093/cid/ciu296>
31. Sullivan, T., & de Barra, E. (2018). Diagnosis and management of cellulitis. *Clinical Medicine*, 18(2), 160–163. <https://doi.org/10.7861/clinmedicine.18-2-160>
32. Sundheimer, C., & Henghold, W. B. (2019). Erysipelas carcinomatosum. *Dermatology Online Journal*, 25(1)
33. Thomas, K. S., Crook, A. M., Foster, K. A., Mason, J. M., Chalmers, J. R., Nasr, I. S., Brindle, R. J., Nunn, A. J., Williams, H. C., & U.K. Dermatology Clinical Trials Network PATCH Trial Team. (2012). Prophylactic antibiotics for the prevention of cellulitis of the leg: Results of the UK dermatology clinical trials network patch ii trial. *British Journal of Dermatology*, 166(1), 169–178. <https://doi.org/10.1111/j.1365-2133.2011.10586.x>

34. Thomas, K. S., Crook, A. M., Nunn, A. J., Foster, K. A., Mason, J. M., Chalmers, J. R., Nasr, I. S., Brindle, R. J., English, J., Meredith, S. K., Reynolds, N. J., de Berker, D., Mortimer, P. S., Williams, H. C., & U.K. Dermatology Clinical Trials Network PATCH I Trial Team. (2013). Penicillin to prevent recurrent leg cellulitis. *The New England Journal of Medicine*, 368(18), 1695–1703. <https://doi.org/10.1056/NEJMoa1206300>
35. Vignes, S., Poizeau, F., Dupuy, A., & Arrault, M. (2022). Cellulitis risk factors for patients with primary or secondary lymphedema. *Journal of Vascular Surgery: Venous and Lymphatic Disorders*, 10(1), 179–185. <https://doi.org/10.1016/j.jvsv.2021.06.011>
36. Walsh, T. L., Chan, L., Konopka, C. I., Burkitt, M., Moffa, M. A., Bremmer, D. N., Murillo, M. A., Watson, R., Chan-Tompkins, N. H., & Nagel, J. L. (2016). Appropriateness of antibiotic management of uncomplicated skin and soft tissue infections in hospitalized adult patients. *BMC Infectious Diseases*, 16, 721. <https://doi.org/10.1186/s12879-016-2067-0>
37. Warren, A. G., Brorson, H., Borud, L. J., & Slavin, S. A. (2007). Lymphedema: A comprehensive review. *Annals of Plastic Surgery*, 59(4), 464–472.
38. Webb, E., Neeman, T., Bowden, F. J., Gaida, J., & Mumford, V. (2020). Compression therapy to prevent recurrent cellulitis of the leg. *The New England Journal of Medicine*, 383(7), 630–639. <https://doi.org/10.1056/NEJMoa1917197>
39. Webb, E., Neeman, T., Bowden, F. J., Mumford, V., & Bissett, B. (2023). Compression therapy is cost saving in the prevention of lower limb recurrent cellulitis in patients with chronic edema. *Lymphatic Research and Biology*, 21(3), 260–267. <https://doi.org/10.1089/lrb.2022.0029>
40. Weng, Q. Y., Raff, A. B., Cohen, J. M., Gunasekera, N., Okhovat, J. P., Vedak, P., Joyce, C., Kroshinsky, D., & Mostaghimi, A. (2017). Costs and consequences associated with misdiagnosed lower extremity cellulitis. *JAMA Dermatology*, 153(2), 141–146. <https://doi.org/10.1001/jamadermatol.2016.3816>
41. Yadav, K., Suh, K. N., Eagles, D., & Perry, J. J. (2015). Predictors of oral antibiotic treatment failure for nonpurulent skin and soft tissue infections in the emergency department. *Academic Emergency Medicine*, 22(2), 163–170. <https://doi.org/10.1111/acem.12584>
42. Zervos, M. J., Freeman, K., Vo, L., Haque, N., Pokharna, H., Raut, M., & Kim, M. (2012). Epidemiology and outcomes of complicated skin and soft tissue infections in hospitalized patients. *Journal of Clinical Microbiology*, 50(2), 238–245. <https://doi.org/10.1128/JCM.05817-11>