

Erysipelas and its Management in a Tertiary Care Hospital

Mayra, Evelyn Quinones Martinez¹, Charit nayelly chang Cruz², Kim Rodriguez Relingh³

^{1,2,3}Department of internal medicine high speciality hospital of veracruz, sesver ,veracruz,Mexico Av.20 De noviembre 1074 ver,ver

ABSTRACT

Erysipelas is an acute dermo-hypodermic (non-necrotizing) infection of bacterial origin, mainly group A beta-hemolytic streptococcus. The lower limbs are affected in more than 80% of cases and the risk factors identified are skin barrier breakdown, lymphedema and obesity. Diagnosis is clinical and is based on the association of an acute inflammatory plaque with fever, lymphangitis, lymphadenopathy and leukocytosis. Bacteriology is generally not useful due to low sensitivity or late positivity. In atypical forms, erysipelas should be distinguished from necrotizing fasciitis and acute venous thrombosis. Penicillin remains the gold standard treatment, although new drugs may be used, given their pharmacodynamic profile. Recurrence is the main complication, being crucial the correct treatment of risk factors¹.

ARTICLE DETAILS

Published On:
06 May 2023

Available on:
<https://ijmscr.org/>

INTRODUCTION

Erysipelas is a cutaneous bacterial infection caused by group A beta-hemolytic streptococcus or *Streptococcus pyogenes* (*S. pyogenes*) or, more rarely, *Staphylococcus aureus* (*S. aureus*). It is characterized by the presence of acute inflammatory signs (erythema, edema, heat and local pain) in the cutaneous integument, often associated with systemic symptoms of malaise, chills and fever. It diffusely affects the dermis and the superficial part of the subcutaneous cellular tissue, being important the involvement of local lymphatic vessels. Its preferred locations are the lower extremities, centropacial area and auricular pavilions.²

EPIDEMIOLOGY

Erysipelas is a frequent pathology. The incidence rate varies from 0.2/1000 to 24.6/1000 persons per year in different populations. They affect the lower extremity in approximately 80% of cases, and can also occur on the upper extremities, face and trunk.³ Lesions of the upper extremity are associated with intravenous drug users and women undergoing axillary lymphadenectomy for breast cancer (with an incidence that can reach 24%).⁴

Facial erysipelas in children has decreased in recent decades, erysipelas of the upper extremities after breast surgery, and erysipelas of the erysipelas on the upper extremities after breast surgery are also becoming rarer because surgical procedures are becoming less and less invasive.⁵

They are more frequently seen in elderly, immunocompromised patients, and with underlying pathologies that will be mentioned throughout the article.⁶

CLINICAL MANIFESTATIONS

Erysipelas begins as a localized area of erythema and edema, painful, with raised borders and spreads rapidly with active periphery. The distinguishing features of classic erysipelas are red, edematous (orange peel-like) lesions, raised margins and clear contrast with healthy skin, which is often accompanied by systemic symptoms two to three days after inoculation, such as fever, chills, lymphangitis and regional lymphadenopathy, and often the gateway is found to be trauma (often unnoticed by the patient), abrasions, eczematous lesions or more frequently superficial mycosis. Bacteremia is present in less than 5% of cases. Sometimes it is accompanied by vesicles or blisters that may be hemorrhagic (erysipelas bullosa), especially in elderly or immunosuppressed patients, complicating the natural course. Another variant is erysipelas phlegmonosa, considered the most severe form, in which abscesses are formed and then necrosis^{7,8,9,10}.

DIAGNOSIS

The diagnosis of erysipelas is based on the association of an acute inflammatory plaque with fever, lymphagitis, adenopathy and hyperleukocytosis. These associated

Erysipelas and its Management in a Tertiary Care Hospital

symptoms are variable (20-70 p. 100 of cases). Bacteriology is not useful for the diagnosis of erysipelas due to its low sensitivity (blood culture 5 p. 100, standard tests 5-41 p. 100) or late positivity (serology). In addition, skin bacteriology is difficult to evaluate when bacteria other than streptococci are isolated. Erysipelas should be distinguished from nonnecrotizing cellulitis by peculiar clinical features (such as erysipeloid, staphylococcal facial infection, *Pasteurella*, *Haemophilus influenzae*) and necrotizing fasciitis. Some non-infectious diseases can mimic erysipelas, such as venous thrombosis, familial Mediterranean fever, prosthesis intolerance and compartment syndrome. Because the diagnostic value of clinical symptoms is unknown and no diagnostic gold standard has been established, it is impossible to be certain that nonstreptococcal (especially staphylococcal) erysipelas actually exists. Therefore, the first-line treatment for all erysipelas should be an antistreptococcal antibiotic. Before prescribing treatment, blood culture and blood count may be helpful. If antistreptococcal antibiotic therapy is ineffective, all differential diagnoses should be reviewed.¹¹

TREATMENT

The treatment for erysipelas will initially be local measures, such as immobilization and subsequent elevation of the area that is affected to reduce edema, also cold compresses with sodium chloride or sterile saline will be used, which will considerably reduce pain because they are astringent agents in the presence of blisters. If the physical treatment does not produce improvement, antibiotic treatment will be started empirically, which can be modified according to the evolution of the patient and depending on the result of cultures in case of taking them, it will be started for mild cases with cloxacillin 500 mg every 6 hours, later azithromycin 500 mg every 24H or clarithromycin 500 mg every 12H will be used.^{12,13,14,15} If methicillin-resistant *S. Aureus* has been cultivated, the treatment will be changed to cotrimoxazole at a rate of 80mg/400mg two tablets every 12 hours + rifampicin 600 mg every 24 hours or it has been shown that the use of ciprofloxacin 500 mg every 12 hours or the use of fusidic acid 500 mg every 8 hours are well tolerated by patients and the evolution tends to be positive.¹⁶ In patients with evolved erysipelas occupying a large area and with associated underlying pathologies, they should be hospitalized and treated with intravenous benzathine penicillin G at a rate of 1000000 to 2,000000 IU every 4-6 H. If the causal agent is suspected to be staphylococcal, semisynthetic penicillin resistant penicillin such as cloxacillin at a rate of 2g every 4 hours intravenous or cefazolin at a rate of 1g every 8h intravenous should be administered. And in cases of methicillin-resistant *S. aureus*, vancomycin will be used at a rate of 500 mg every 6 hours or 1 g every 12 hours¹⁷.

CONCLUSIONS

In conclusion, treatment of erysipelas is based on the severity of the disease and the age of the patient. Topical treatments are the first line of treatment for most patients with mild to moderate erysipelas. Systemic treatments are reserved for patients with severe erysipelas with systemic involvement who do not respond to topical treatments. The search for new treatments is challenging for hospital-based health care personnel.

REFERENCES

- I. Caetano M, Amarin I. Erisipela [Erysipelas]. *Acta Med Port.* 2005 SepOct;18(5):385-93. Portuguese. Epub 2005 Oct 16. PMID: 16611543
- II. Wessels MR. Choosing an Antibiotic for Skin Infections. *N Engl J Med.* 2015;372(12):1164-5.
- III. Ellis Simonsen SM, Van Orman ER, Hatch BE, Jones SS, Gren LH, Hegmann KT, et al. Cellulitis incidence in a defined population. *Epidemiol Infect* 2006;134:293-9.
- IV. Simon MS, Cody RL. Cellulitis after axillary lymph node dissection for carcinoma of the breast. *Am J Med.* 1992;93:543-8.
- V. Ferreira A, Bolland MJ, Thomas MG. Metaanalysis of randomised trials comparing a penicillin or cephalosporin with a macrolide or lincosamide in the treatment of cellulitis or erysipelas. *Infection* 2016;44:607-15.
- VI. Wessels MR. Choosing an Antibiotic for Skin Infections. *N Engl J Med.* 2015;372(12):1164-5.
- VII. Olmos E, Bonilla CE. Infección por *Streptococcus* y *Staphylococcus* en dermatología. En Olmos E: *Texto de dermatología 25 años Hospital de San José.* Bogotá: Sociedad de Cirugía de Bogotá; 2007, p. 187-203.
- VIII. Camilleri MJ, Daniel Su WP. Panniculitis. En: Freedberg IM, Eisen AZ, Wolf K, Austen KF, Goldsmith LA, Katz S: *Fitzpatrick's dermatology in general medicine*, 6th ed; 2003. p.1275-8.
- IX. Swartz MN, Pasternack MS. Skin and soft tissue infections. En Mandell GL, Bennett JE, Dolin R: *Principles and practice of infectious disease.* 6th ed. Philadelphia: Churchill Livingstone; 2005, p.1172-84.
- X. Stevens DL. Cellulitis, pyoderma, abscesses and other skin and subcutaneous infections. En: Cohen J, Powderly WG: *Infectious diseases.* 2nd ed. Philadelphia: Mosby Elsevier; 2004. p. 133-55.
- XI. Vaillant L. Critères diagnostiques de l'érysipèle [Diagnostic criteria for erysipelas]. *Ann Dermatol Venereol.* 2001 Mar;128(3 Pt 2):326-33. French. PMID: 11319359.
- XII. Wang YF, Que HF, Wang YJ, Cui XJ. Chinese herbal medicines for skin and soft tissue infections. *Cochrane Database Syst Rev.* 2013;2013(6).

Erisipelas and its Management in a Tertiary Care Hospital

- XIII. Quéré I, Nagot N, Vikkula M. Incidence of Cellulitis among Children with Primary Lymphedema. *N Engl J Med*. 2018;378(21):2047–8.
- XIV. Swartz MN. Cellulitis. 2004;904–12.
- XV. Norimatsu Y, Ohno Y. Predictors for readmission due to cellulitis among Japanese patients. *J Dermatol*. 2021;48(5):681–4.

- XVI. Raff AB, Kroshinsky D. Cellulitis a review. *JAMA - J Am Med Assoc*. 2016;316(3):325– 37
- XVII. Bd B, Kl HW. Cellulitis Pathophysiology Treatment / Management Differential Diagnosis. 2020;1–5