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## Érysipèle et fasciite nécrosante : prise en charge

Faculté de médecine de Tours – mercredi 26 janvier 2000

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L'organisation de cette conférence a été rendue possible grâce à l'aide apportée par les laboratoires suivants que la Spilf tient à remercier : Abbott, Bayer Pharma, Bristol-Myers Squibb, GlaxoWellcome, Hoechst Marion-Roussel, Institut Smithkline Beecham, Merck Sharp & Dohme-Chibret, Pfizer, Pharmacia Upjohn, Produits Roche, Laboratoires Rhône-Poulenc Rorer, Wyeth Lederle.

La Société française de dermatologie remercie les laboratoires suivants de leur soutien : Galderma, Laboratoires Leo, Pierre Fabre Dermatologie, Laboratoires Rhône-Poulenc Rorer, Produits Roche.

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## AVANT-PROPOS

Cette conférence a été organisée et s'est déroulée conformément aux règles méthodologiques préconisées par l'Agence nationale d'accréditation et d'évaluation en santé (ANAES) qui lui a attribué son label de qualité.

Les conclusions et recommandations présentées dans ce document ont été rédigées par le Jury de la Conférence, en toute indépendance. Leur teneur n'engage en aucune manière la responsabilité de l'ANAES.



## Consensus conference on Erysipelas and necrotizing fasciitis: management

### Short text \*

#### QUESTIONS 1 AND 2: ERYSIPELAS

##### Question 1 – What data is needed today to deal with erysipelas?

##### A reminder of anatomy and terminology

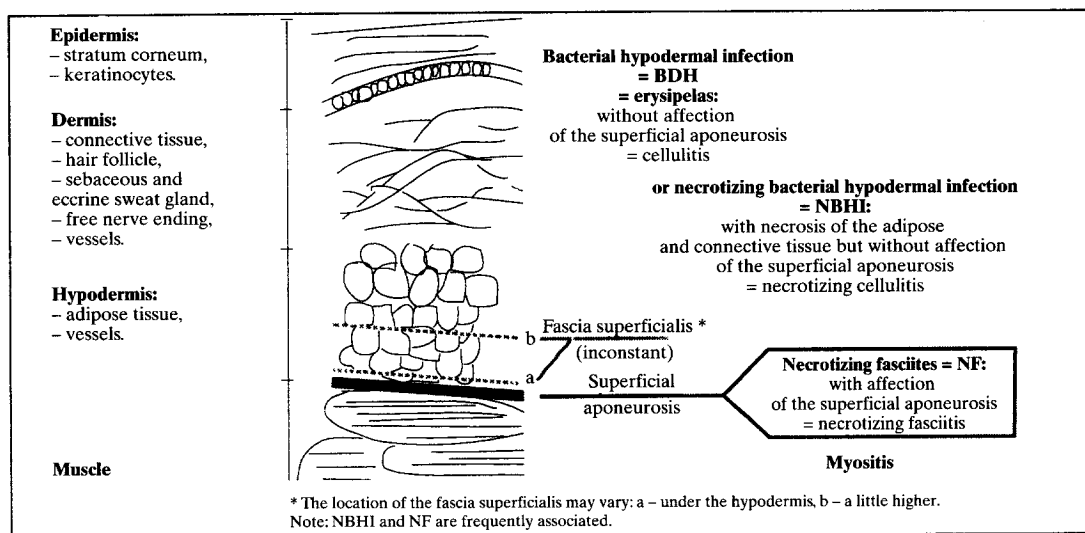
The skin is made of epidermis, dermis, hypodermis (wrongly named sub-cutaneous tissue). The hypodermis is lined in its deep part by the fascia superficialis, not well defined and inconstant, and a solid deeper structure, the superficial aponeurosis, site of necrosis in fasciitis (*cf. figure infra*).

The English term “cellulitis” referring to a non-existing sub-cutaneous cellular tissue is improper. It

is confusing because it concerns various histological entities and thus must be discarded and replaced according to the nature of the lesion and the anatomical structure affected by bacterial hypodermal infection (BDH), necrotizing BDH, or necrotizing fasciitis.

##### Epidemiological Data

Erysipelas is an acute hypodermal infection (non-necrotizing) of bacterial origin, essentially streptococcal, sometimes recurrent. A large number of patients, probably close to 50% are treated at home. It is a common pathology the incidence of which is estimated at 10 to 100 cases for 100,000 inhab/year.



\* La version française de ce texte court a déjà été publiée dans Méd Mal Infect 2000 ; 30 : 241-5.

Erysipelas located in the lower limb for more than 85% of the cases.

Risk factors have been identified: local (lymphoedema and initial infection site i.e. interdigital toe intertrigo, leg ulcer), general (obesity). Diabetes and chronic alcohol abuse do not seem to be risk factors contrary to popular belief.

### Microbiological Data

Erysipelas is a peculiar disease due to its toxic and infectious aspects and to the weak bacterial density in the lesions.

Only streptococcal etiology can be demonstrated ( $\beta$ -hemolytic A, B, C, and G streptococci). There is no convincing argument suggesting a staphylococcal etiology of erysipelas.

Bacteriological assessment is more interesting on an epidemiological level than for the diagnosis, because of a weak sensibility or of a late positivity. Bacteriological assessment is not necessary for typical presentations and in the absence of comorbidity symptoms(s).

### Clinical Data

Positive diagnostic is easy and clinical. The onset of the disease is often brutal. It combines general symptoms (fever, chills) and local symptoms (inflammatory plaque sometimes blisteric or purpuric but without necrosis). The initial infection site must be identified. A sensitive satellite adenopathy is present in 46% of the cases, lymphangitis in 26% of the cases.

No complementary examination is necessary.

Anamnesis and a clinical examination must rule out the following conditions:

- necrotizing hypodermal infection, necrotizing fasciitis,
- deep and superficial phlebitis,
- acute bacterial hypodermal infection due to other specific pathogens,
- stasis dermatitis,
- hypodermal infection on a surgical scar which may be erysipelas.

Evolution is favorable within 8 to 10 days of antibiotic treatment in over 80% of the cases. Apyrexia comes in within 72 h; it precedes the improvement of local signs usually observed in the seventh day: in 80% of the cases for edema, and 60% of the cases for erythema.

Initial severity depends on the underlying conditions and the severity of the local and systemic assessment.

Mortality is inferior to 0.5% and depends on associated pathologies. Complications are rare (abscess, general complications).

## **Question 2 – How should erysipelas be managed?**

### **Antibiotic Treatment**

Treatment for erysipelas must be antistreptococcal. Empiric antibiotic drugs belong to the  $\beta$ -lactam family. There is no consensus in the jury concerning the use of pristinamycin in first intention. Injectable G penicillin is the reference antibiotic drug. Nevertheless constraints and iatrogenic risks (repeated infusions) are linked to its use and hospitalization is required. This is why initial oral treatment is used (amoxicillin). Evaluating new modes of administration is necessary (short duration of treatment, single daily intra-muscular or intravenous direct administration of a long half-life antibiotic drug).

Choosing the antibiotherapy depends on hospitalizing or keeping the patient at home, on the severity of the local and general evaluation, on an uncertain diagnosis when confronted to an atypical assessment, on allergy to  $\beta$ -lactamines, on expected observance to an oral therapy, and on associated diseases.

Dosage should be made according to the patient's weight, especially for obese patients, and to elimination conditions, especially renal.

### **In case of initial hospitalization**

A severe initial local or initial clinical assessment justifies choosing the reference G Penicillin treatment with 4 to 6 infusions per day (10 to 20 million units [MU] per day). Apyrexia then allows switching to an oral antibiotherapy (penicillin V, 3 to 6 MU/d in 3 daily doses; amoxicillin, 3 to 4.5 g/d in 3 daily doses) until all local signs disappear, with total treatment duration lasting between 10 and 20 days.

When confronted to typical erysipelas, without severity signs, an oral treatment oral is possible. If the patient leaves hospital early, after apyrexia is secured, the community practitioner must check the absence of local complications and treatment observance.

### **In case of home treatment**

Oral treatment with amoxicilline 3 to 4.5 g/d in 3 daily doses is prescribed. Treatment usually lasts 15 days. If necessary, it must be followed by a secondary prevention.

### **In case of allergy to $\beta$ -lactamines**

Pristinamycin is chosen (2 to 3 g/d in 3 daily doses), a macrolide or clindamycin. Some of these antibiotic drugs are available under parenteral intravenous presentation.

### Unfavorable evolution under treatment

The absence of improvement or the worsening condition suggests that there are deep and/or necrotizing lesions, or resistant bacteria. Antibiotic treatment must then be reconsidered and in some cases, surgery may be required.

### Criteria for initial or secondary hospitalization

Initial hospitalization is required whenever a parenteral treatment or a close surveillance are necessary: when the diagnosis is uncertain, when there are important general signs, comorbidity or a social context making home treatment impossible.

If home treatment is selected, a daily surveillance of general and local signs is necessary. Persistence of fever after 72 hours of treatment, apparition of new local or general signs, decompensation of an associated disease must lead to hospitalization.

### Local treatment

A local or antibiotic antiseptic treatment with an etiological aim on erysipelas or on the initial site of infection is not necessary. Applying topical anti-inflammatory drugs is contra-indicated. An adapted treatment of the initial site of infection is required (eradication of an interdigital toe intertrigo, treating a leg ulcer).

### Deep venous thrombosis risk and anticoagulant drugs

There is a small risk of deep venous thrombosis in erysipelas of the lower limb (0.7 to 4.9%). It does not justify the systematic use of anticoagulant drugs with a prophylactic aim. Antiaggregant drugs are not indicated either. A preventive anticoagulant treatment may be suggested only in the case of an associated thrombo-embolic risk, as in any acute infectious disease.

Systematic echo-Doppler search for deep venous thrombosis is not necessary. Venous pressure stockings and an early bedrising, which would contribute to limiting the occurrence of deep venous thrombosis and to fight lymphoedema in risk patients, may be suggested.

### NSAID and corticosteroid drugs

Non steroidal anti-inflammatory drugs (NSAID) or corticosteroid drugs are not indicated for erysipelas.

Using NSAID for acute bacterial hypodermal infection could induce necrotizing fasciitis. Currently available data doesn't allow establishing a causal relationship. Using NSAID is thus contra-indicated.

In case of high or badly tolerated pyrexia, antipyretic and/or antalgic drugs such as acetaminophen should be chosen.

It is not necessary to change the treatment if it a patient follows a long-term NSAID or general corticosteroid therapy. Nevertheless, these treatments represent a comorbidity factor which should lead to initial hospitalization.

### Primary and secondary prevention

#### Primary prevention

A careful management of venous and lymphatic stasis and treatment of interdigital toe intertrigo are recommended in the general population.

#### Secondary prevention

Recurrent erysipelas affects about 20% of the patients. They are often multiple for a same patient. They are often induced by the persistence or recurrence of factors having induced the first episode: lymphoedema, persistence, or recurrence of the initial infection site. Preventing recurrence is mandatory from the first episode of erysipelas. This prevention relies on:

- identifying and efficiently treating the initial infection site, especially when it is chronic. This is especially the case for an adapted management of interdigital toe intertrigo and its inducing factors;
- the long-term management of a lymphoedema or an edema of venous origin by pressure stockings and/or manual lymphatic drainage.

A preventive antibiotherapy against recurrence is necessary in patients already affected by multiple recurrences or in whom inducing factors are difficult to control.

It must be prolonged and eventually definitive because its effect is only suspensive.

Penicillin is used: penicillin V, 2 to 4 MU/d b.i.d. orally or benzathine-penicillin, 2,4 MU every 2 or 3 weeks by intramuscular injections. This second treatment may warrant a good observance.

In case of allergy to  $\beta$ -lactam, a macrolide is used orally.

## QUESTIONS 3 AND 4: NECROTIZING FASCIITIS

### Question 3 – What data is needed today to deal with necrotizing bacterial hypodermal infection and necrotizing fasciitis (NBHI-NF)?

These terms are used to describe rare forms of infection lethal in close to 30% of the cases. *Streptococcus pyogenes* is a frequent causal agent. It is often associated to other bacteria. This infection causes a