SPANISH ASSOCIATION OF PæDIATRICS

SEIP–AEPAP–SEPEAP consensus document on the aetiology, diagnosis and treatment of bacterial skin infections in out-patients


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Bacterial skin infections;
Staphylococcus aureus;
Streptococcus pyogenes;
Treatment

Abstract  Skin infections are a common cause for dermatological consultations in the pædiatric setting. A review is presented of the clinical manifestations, diagnosis and treatment of the main bacterial skin infections, as well as the diagnosis and treatment of super-infected puncture and bite wounds. The most prevalent bacteria in skin infections are Staphylococcus aureus and Streptococcus pyogenes. Treatment is usually empirical, since microbiological studies are only recommended under certain circumstances or lack of improvement with common therapies. Superficial skin infections can be treated with local antiseptics or antibiotics (mupirocin or fusidic acid). Systemic treatment is usually reserved for patients with extensive or severe disease or with other risk factors. Systemic treatment depends on the suspected infecting bacteria, with penicillin, amoxicillin, amoxicillin–clavulanic acid and first- or second-generation cephalosporin being the most frequently used drugs. Due to the low incidence of community-acquired methicillin-resistant infection by S. aureus in Spain, the use of clindamycin or co-trimoxazole is only recommended after severe disease, relapses or a clear epidemiological background.

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PALABRAS CLAVE
Infecciones cutáneas bacterianas; Staphylococcus aureus; Streptococcus pyogenes; Tratamiento

Documento de consenso SEIP-AEPAP-SEPEAP sobre la etiología, el diagnóstico y el tratamiento de las infecciones cutáneas bacterianas de manejo ambulatorio

Resumen Las infecciones cutáneas constituyen un motivo de consulta frecuente en dermatología pediátrica. Se revisan las manifestaciones clínicas, el diagnóstico y el tratamiento de los principales cuadros infecciosos bacterianos de la piel, así como de la sobreinfección de las heridas punzantes y por mordedura. Las bacterias más prevalentes en las infecciones cutáneas son Staphylococcus aureus (S. aureus) y Streptococcus pyogenes. El tratamiento es generalmente empirico y solo ante determinadas circunstancias o mala evolución clínica se recomienda el estudio microbiológico. Las infecciones cutáneas superficiales pueden tratarse con antisépticos y antibióticos tópicos (mupirocina o ácido fusídico). El tratamiento sistémico se reserva para formas extensas, graves o con otros factores de riesgo del huésped. En estos casos, el antibiótico de elección dependerá, entre otros factores, de los patógenos sospechados; los más utilizados son penicilina, amoxicilina, amoxicilina-ácido clavulánico y cefalosporinas de primera o segunda generación. Considerando la baja incidencia de S. aureus resistente a la meticilina de adquisición comunitaria en nuestro país, no se recomienda modificar el tratamiento empírico salvo en circunstancias de especial gravedad, recurrencia o antecedente epidemiológico, en cuyo caso el tratamiento recomendado es clindamicina o trimetoprima-sulfametoxazol.

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Introduction

The skin is the first barrier of the organism against external agents and skin infections, the most frequent reason for paediatric dermatology consultations. The main risk factors are deterioration of the integrity of the skin, poor hygiene, overcrowding, humidity and immunodeficiency.

The most prevalent bacteria are Staphylococcus aureus (S. aureus) and Streptococcus pyogenes (S. pyogenes). Both can cause skin infections by direct inoculation, haematogenous dissemination and through the production of toxins, as in Staphylococcal scalded skin syndrome by S. aureus and staphylococcus or streptococcus toxic shock syndrome.

Based on the system of consensus documents, we will add the strength of the recommendation to our proposed measures, following the classification of the Infectious Diseases Society of America (Table 1).

Antibiotic treatment of skin infections

In general, the antibiotic treatment of skin infections is empirical. Blood or aspirate cultures, biopsies and swabs are only recommended under certain circumstances (chemotherapy, neutropenia, serious immunodeficiency, immersion wounds, animal bites, infection of the general condition, suspicion of complications, extensive infection, suspicion of ecthyma gangrenosum, necrotising fasciitis, admitted patients) or in the event of poor response to treatment. Treatment should be based on clinical analysis, possible microorganism involved, site, extension and depth of the infection, and personal history.

Asymptomatic skin infections involving the superficial layers of the skin are usually treated with antiseptics or topical antibiotics; those with the best coverage against common pathogens are mupirocin ointment and fusidic acid cream or ointment. In infections by methicillin-sensitive S. aureus with poor response to mupirocin, retapamulin ointment is a valid alternative in patients aged over 9 months. Systemic antibiotic therapy is reserved for certain clinical forms (cellulitis), extensive forms, rapid progression or Table 1 Strength of recommendation and quality of evidence.

<table>
<thead>
<tr>
<th>Strength of recommendation</th>
<th>Quality of evidence</th>
</tr>
</thead>
<tbody>
<tr>
<td>A Good evidence to support a recommendation for use</td>
<td>I Evidence from at least one properly randomised, controlled trial</td>
</tr>
<tr>
<td>B Moderate evidence to support a recommendation for use</td>
<td>II Evidence from at least 1 well-designed clinical trial, without randomization; from cohort or case-controlled analytic studies (preferably from more than 1 centre); from multiple time series; or from dramatic results from uncontrolled experiments</td>
</tr>
<tr>
<td>C Poor evidence to support a recommendation</td>
<td>III Evidence from opinions of respected authorities, based on clinical experience, descriptive studies, or reports of expert committees</td>
</tr>
<tr>
<td>D Moderate evidence to support a recommendation against use</td>
<td></td>
</tr>
<tr>
<td>E Good evidence to support a recommendation against use</td>
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</tbody>
</table>

Taken from Khan et al. ²
dissemination, serious cases or patients in generally poor condition (necrotising fasciitis), neonates, cellulitis-adenitis in patients under 3 months of age and in immunodepressed patients. Sometimes, incision and surgical drainage are essential.

If there is suspicion of *S. aureus*, the antibiotic of choice is cloxacillin, although its pharmacological characteristics (administration every 6 h, low bioavailability and bad taste) make amoxicillin–clavulanic acid and first-generation cephalexin (cefadroxil or cefalexin) a reasonable alternative. In the event of contraindication for β-lactams, the drugs of choice are clindamycin (only available in 150 and 300 mg capsules in Spain) and trimethoprim-sulfamethoxazole.

If there is suspicion of *S. pyogenes*, the antibiotic of choice is penicillin or amoxicillin. In patients who are allergic to β-lactams, 16-atom macrolides can be used (josamycin or midecamicina), clindamycin or, in the event of non-anaphylactic reaction, first-generation cephalexin.

In infections where both bacteria may be implicated, treatment with amoxicillin–clavulanic acid or a first-generation cephalexin is indicated.

At the time of drafting this document, neither cefadroxil nor cefalexin, the only first-generation cephalexin with oral presentation, are sold in Spain in suspension form, and only 500 mg capsules are available. Under these circumstances, second-generation cephalexin (cefuroxime, cefaclor), despite being slightly less active against gram-positive cocci than first-generation cephalexin, is a valid alternative.

**Main infectious conditions**

**Impetigo**

The onset of impetigo may occur at any age and is transmitted by autoinoculation, direct contact, or contaminated objects. It produces non-scarring epidermal lesions, well delimited, usually in exposed areas. It may be bullous or non-bullous. In the non-bullous form, caused by *S. pyogenes, S. aureus* (alone or in co-infection) and, occasionally, by group C and G streptococci, the lesions evolve from macula to papules, vesicles and pustules, which finally form a thick melicrneric crust. In the bullous form, caused by *S. aureus* exfoliative toxins, the lesions evolve to vesicles and then to blisters with yellow more or less turbid and even purulent fluid, and finally form a fine crust. The main complication is local dissemination (cellulitis, lymphadenitis) or even systemic complications (pneumonia, osteoarthritis, sepsis).

In non-complicated mild cases, topical disinfectants can be used, mupirocin or fusidic acid every 8 h, or retapamulin every 12 h, for 5–7 days. If the lesions are extensive or with systemic symptoms, oral antibiotic therapy with amoxicillin–clavulanic acid, a first- or second-generation cephalexin, or cloxacillin is recommended.

**Perianal bacterial dermatitis**

Perianal bacterial dermatitis consists of an erythema around the anus caused by *S. pyogenes* or less frequently by *S. aureus*, alone or associated with *S. pyogenes*. Incidence peaks at 3–5 years of age, predominantly in males (70%), although prevalence is the same in both sexes if vulvovaginitis is included, which together with balanoposthitis constitute what is known as perineal disease.

It manifests as a non-eroded erythema, measuring 2–3 cm, around the anus, with well-delimited edges, sometimes accompanied by pruritus, painful bowel movement, constipation, overflow incontinence, haematochezia and fissures. Boys may present with balanoposthitis and girls with vulvovaginitis with secretion and dysuria. There are usually no general symptoms, unlike cellulitis. In the acute phase (<6 weeks progression) the lesion is red, bright, humid and painful, and may present a whitish pseudo-membrane.

Without treatment, lesions become chronic, with painful fissures, mucous secretion or psoriasis-like plates with yellowish periphery crust. When the cause is staphylococcus, there may be pustules and the erythema extends towards the adjacent skin.

Patients or members of the household may have presented acute pharyngooaginaldilis or a skin infection in the previous 3 months or simultaneously with perianal dermatitis, or be pharyngeal or perianal carriers of *S. pyogenes*. Recurrence after treatment with penicillin or amoxicillin is frequent (up to 37%), especially if there are non-diagnosed cases in relatives.

It is believed that transmission is by contact, whether through autoinoculation (infection or pharyngeal or skin carrier) or interfamilial contagion, especially if bath water is shared or if a relative has presented acute pharyngooaginaldilis or perianal dermatitis.

Diagnosis is based on clinical analysis and confirmation is bacteriological. Samples from the perianal area must be gathered from all patients (A-II) and pharyngeal samples if there are symptoms (B-III), for a rapid antigen detection test for *S. pyogenes* (100% positive predictive value) or a culture. If the result of the test is negative, it is recommended to prepare a culture due to the possibility of false negatives or *S. aureus* as a cause of the condition (A-II). Potential cases in persons cohabiting with the patient should be investigated and a rapid diagnostic test or a culture for symptomatic patients (B-III) carried out, since this is a frequent cause of recurrences within the family.

Although good results have been reported with topical antibiotic therapy in monotherapy (B-II), the use of systemic antibiotics is recommended in all cases (A-II). If no rapid diagnostic test available or if it is positive, oral treatment with penicillin or amoxicillin for 10 days is recommended (B-II), which may be prolonged to 14–21 days based on clinical progress (C-III). If there is no response after one cycle of amoxicillin, or if the diagnostic test was negative, and while waiting for the result of the culture, amoxicillin–clavulanic acid may be used (C-III).

When choosing the initial antibiotic therapy, a familial history of streptococci or staphylococcus infection must be taken into consideration (C-II).

For recurrent infection cefuroxime is recommended for 7–10 days (C-II). The association of oral and topical treatment seems to decrease recurrences (A-II).
Infections of hair follicles: folliculitis, furuncle and carbuncle

The aetiologic agent of hair follicle infection is *S. aureus*, followed by gram-negative bacilli. Predisposing factors include obesity, diabetes, hyperhidrosis, immunosuppression and atopic dermatitis.

Clinically, patients present with lesions on hair-bearing sites on the face, neck, armpits or glutei, ranging from a papule-vesicle with a erythematous base (folliculitis, which affects the superficial hair follicle), to tender erythematous nodules (furuncle, extending to the deep dermis), to warm, tender, erythematous plaques (carbuncle, affecting the surrounding subcutaneous cellular tissue).

The diagnosis is clinical and a microbiological study is only recommended for lesions that are atypical or respond poorly to treatment.12

Treatment is initially topical with mupirocin or fusidic acid, and in recurrent or extensive forms not responding to topical treatment, a combination of amoxicillin-clavulanic acid, clindamycin, cefadroxil, cefalexin or cefuroxime. For furuncles and carbuncles, it is recommended to apply local heat and assess the possibility of making an incision and surgical drainage.

Acute bacterial lymphangitis

This is defined as the inflammation of the lymphatic vessels of the subcutaneous tissue. The organisms most frequently involved are *S. pyogenes*, *S. aureus* and *Pasteurella multocida* (*P. multocida*) (the latter after an animal bite). There is usually systemic involvement, even before the distal oedema and linear cord to regional nodes are evident.

Empirical treatment in mild to moderate forms is amoxicillin-clavulanic acid or a first- or second-generation cephalosporin.

Erysipelas

This is a superficial infection affecting the superior dermis, the subcutaneous cellular tissue and sometimes the lymphatic system, in most cases caused by *S. pyogenes* or streptococci of groups B, C or G.1,13-18

The triggering factors include abrasions, ulcers, intertriginous fungal infections of the feet, venous or lymphatic obstruction and chronic oedema. In neonates, the infection may originate in the umbilical cord and extend to the abdominal wall.

Onset is acute, characterised by an erythematous plate with well-defined and slightly elevated edges. It is more frequent in lower limbs and face, and may be associated with regional lymphadenitis. There are usually general symptoms (fever, chills, discomfort), sometimes before cutaneous signs. Systemic complications are also possible (sepsis, streptococcal toxic-shock syndrome, endocarditis, etc.), although they are rare in otherwise healthy patients. When the infection is resolved it produces scaling and hyperpigmentation.

The diagnosis is clinical (A-II); haemocultures and cultures of skin biopsies or aspirates have a low yield.

Superinfection of bite or puncture wounds

Most superinfections of the wounds produce clinical manifestations in the first 12 h, generally erythema, pain or some other type of secretion through the wound. In human bites, fever above 38 C, abscess and lymphangitis are established criteria of superinfection, as well as the presence of at least 4 of the following: erythema extending more than 3 cm from the edge of the wound, pain on palpation, inflammation, purulent drainage or leukocyte count of over 12,000 cells/ml.

In superinfections of puncture wounds in the foot, clinical symptoms usually begin during the first 5–7 days if the causative agent is *Staphylococcus*, and after 7 days if it is produced by *Pseudomonas*. It is important to note the infection of bones and deep tissues.23,24

In addition to local cleaning and debridement, prophylactic antibiotic treatment is recommended only in the cases listed in Table 5 (A-I) and according to the guidelines in Table 6.

Standard prophylaxis should always be considered in the case of rables, tetanus and other diseases transmitted through wounds.

T reatment consists of administration of oral penicillin or amoxicillin (A-III).1 Patients with lesions of more than 5 cm or generally poor condition, infants and immunodepressed patients may require hospitalisation and parenteral treatment (A-II). The care of wounds and their predisposing factors are also important aspects of the treatment (A-II).

Cellulitis

This is an acute infection affecting the dermis and subcutaneous cellular tissue. Predisposing factors include trauma, wounds and pre-existing infections. It appears as a warm, tender, poorly defined erythematous plaque, which sometimes causes phlyctena, petechial or local necrosis and may be accompanied by lymphadenitis and systemic symptoms.17

The main cause is *S. aureus* and *S. pyogenes*. Other less frequent microorganisms are *Streptococcus agalactiae* and gram-negative bacilli in neonates and enterobacteria in immunosuppressed patients.

Its location may provide an aetiological orientation: in the periauricular region and the sole of the foot with puncture wounds *P. aeruginosa* is more frequent, while in bites, *P. multocida* is common.

The diagnosis is clinical, but a microbiological study is recommended in the cases discussed in the section on general treatment. An ultrasound scan may be useful to differentiate cellulitis from non-fluctuant abscesses.

A systemic antibiotic treatment with good coverage against *S. aureus* and *S. pyogenes* must always be recommended. In mild cases and under strict surveillance, oral treatment with amoxicillin-clavulanic acid, cloxacillin, cefadroxil, cefalexin, cefuroxime or clindamycin may be considered.

Tables 2 and 3 summarise of other infectious conditions of clinical relevance, and Table 4 details the recommended dosage of the most widely used anti-infective drugs.
Table 2 Clinical forms of most frequent bacterial skin infections according to their location of preference.

<table>
<thead>
<tr>
<th>Anatomical location</th>
<th>Infection</th>
</tr>
</thead>
<tbody>
<tr>
<td>Scalp</td>
<td>Abscesses in new borns</td>
</tr>
<tr>
<td>Fingers</td>
<td>Paronychia</td>
</tr>
<tr>
<td>Lymph nodes</td>
<td>Cellulitis</td>
</tr>
<tr>
<td>Sweat glands</td>
<td>Erysipelas</td>
</tr>
<tr>
<td>Lips</td>
<td>Folliculitis by <em>P. aeruginosa</em> (hot tubs, depilation)</td>
</tr>
<tr>
<td>Cheeks</td>
<td>Adenophyllum</td>
</tr>
<tr>
<td>Umbilicus</td>
<td>Peripuritis</td>
</tr>
<tr>
<td>Outer ear</td>
<td>Hydrosadenitis</td>
</tr>
<tr>
<td>Eyelids</td>
<td>Erysipelas</td>
</tr>
<tr>
<td>Perineum</td>
<td>Bacterial cellulitis: pneumococci, <em>Haemophilus influenzae</em> type b</td>
</tr>
<tr>
<td></td>
<td>Cellulitis-adenitis syndrome by <em>S. agalactiae</em></td>
</tr>
<tr>
<td>Sole of the foot</td>
<td>Neonatal omphalitis</td>
</tr>
<tr>
<td>Skin folds</td>
<td>Auricular chondritis (piercing)</td>
</tr>
<tr>
<td></td>
<td>Periorbital cellulitis</td>
</tr>
<tr>
<td></td>
<td>Perianal bacterial disease</td>
</tr>
<tr>
<td></td>
<td>Infection by <em>P. aeruginosa</em> (puncture wound, shoes)</td>
</tr>
<tr>
<td></td>
<td>Pitted keratolysis (keratolysis plantare <em>sulcatum</em>)</td>
</tr>
<tr>
<td></td>
<td>Erythrasma by <em>Corynebacterium minutissimum</em></td>
</tr>
<tr>
<td></td>
<td>Intertrigo by <em>S. pyogenes</em></td>
</tr>
</tbody>
</table>

Taken from Moraga-Llop and Tobeña Rue[18]

Community-acquired methicillin-resistant *S. aureus*

Epidemiology

In the United States, where up to 80% of staphylococci isolated in ambulatory patients are resistant to methicillin, Community-acquired methicillin-resistant *S. aureus* (CA-MRSA) represents a public health problem.[23] In Spain, some studies in paediatric populations report a frequency of resistance to methicillin in *S. aureus* of 9–14.8%,[26,29] but there are no studies at the national level. The frequency in neonates was investigated in the Community of Madrid[30] from 2007 to 2009, and a prevalence of 3.3% was reported.

The role of methicillin resistance as an indicator of seriousness is starting to be questioned, and more relevance is being given to other virus factors, such as Panton–Valentine leukocidin (PVL),[32] although most strains of PVL are also resistant to methicillin.[31,33] Nosocomial MRSA is associated with certain multigene-resistant genotypes, but this is not the case in CA-MRSA, which only rarely presents resistance to macrolides and lincosamides.[27,31,33]

Clinical symptoms and diagnosis

Although they may cause pneumonia, sepsis and osteoarthritis, most CA-MRSA infections are limited to the skin and soft tissues.

There are no clinical criteria to differentiate skin infections produced by methicillin-sensitive *S. aureus* from those caused by MRSA,[34] although in the latter, therapeutic failure and formation of abscesses and necrosis with black superficial crust similar to a spider bite are more frequent.[5] Given the implications for treatment, any non-fluctuant lesions suggestive of abscesses detected during physical examination should be studied under ultrasound examination (C-III). If possible, it is recommended to take samples for culture, determination of LPV and genetic profiling.

Treatment

The current low incidence of AC-MRSA in the paediatric population does not justify amending empirical antibiotic therapy guidelines. However, under certain circumstances changes must be considered, such as extensive cellulitis, abscesses or necrosis, the presence of recurrent abscesses in children or in other members of their household, poor response to conventional treatment, patients living in highly endemic areas (Asia, America or Eastern Europe) or living with known carriers (C-III).

In minor, non-suppurating infections (impetigo, mild superficial infection of wounds, etc.), topical treatment with mupirocin is usually sufficient[19] (A-III). In skin abscesses, incision and early surgical drainage are essential. This has been shown to be effective with otherwise healthy patients with non-complicated infections to achieve full healing with
Table 3  Other clinical forms of bacterial skin infections.

<table>
<thead>
<tr>
<th>Disease</th>
<th>Location</th>
<th>Aetiology</th>
<th>Predisposing factors</th>
<th>Clinical analysis</th>
<th>Diagnosis</th>
<th>Treatment</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Blistering distal dactylitis\textsuperscript{16-18}</td>
<td>Soft part of the fingers&lt;br&gt;Infrequent: other surfaces of palms and soles</td>
<td>\textit{S. pyogenes}&lt;br&gt;Less frequent: \textit{S. aureus}, \textit{S. agalactiae}</td>
<td>Wounds, atopic dermatitis, humidity</td>
<td>Blister with purulent fluid, slightly tender, with erythematous base&lt;br&gt;No general symptoms</td>
<td>Clinical&lt;br&gt;Confirmation: culture\textsuperscript{a}</td>
<td>Incision and drainage + amoxicillin–clavulanic acid 10 days&lt;br&gt;If there is poor response cephalosporin, clindamycin Mupirocin: optional (not in monotherapy)</td>
<td></td>
</tr>
<tr>
<td>Ecthyma\textsuperscript{17-19}</td>
<td>Lower and upper limbs</td>
<td>\textit{S. pyogenes}, \textit{S. aureus}&lt;br&gt;copathogen?</td>
<td>Pruritic lesions: bites, scabies, pediculosis; poor hygiene; malnutrition</td>
<td>Ulcer with black crust that becomes chronic and heals leaving a scar&lt;br&gt;Complications: cellulitis lymphagitis</td>
<td>Clinical&lt;br&gt;Confirmation: Gram stain and culture of exudate or biopsy of deep tissue</td>
<td>Removal of crusts with moist dressings&lt;br&gt;Topical fusidic acid/mupirocin if the lesion is small (&lt;2 cm)&lt;br&gt;Oral 1st generation cephalosporin/amoxicillin–clavulanic acid/macrolide 7 days\textsuperscript{5}&lt;br&gt;Consider admission</td>
<td></td>
</tr>
<tr>
<td>Ecthyma gangrenosum\textsuperscript{17,18}</td>
<td>Perineum, glutei, lower limbs, apocrine areas</td>
<td>\textit{P. aeruginosa}&lt;br&gt;Other bacteria, fungi and virus (immunosuppressed)</td>
<td>Immunosuppression (serious neutropenia), healthy infants</td>
<td>Necrotic ulcer with black crust (bacterial vasculitis of small veins)&lt;br&gt;Sepsis by \textit{P. aeruginosa}</td>
<td>Clinical&lt;br&gt;Confirmation: culture of the lesions, haemocultures, skin biopsy</td>
<td>Early systemic treatment (intravenous) anti-\textit{Pseudomonas} sp.</td>
<td></td>
</tr>
<tr>
<td>Erythrasma\textsuperscript{18}</td>
<td>Humid body folds:&lt;br&gt;interdigital spaces of feet, groins, armpits, infra-mammalian and intergluteal areas</td>
<td>\textit{Corynebacterium minutissimum}, mixed flora, \textit{S. pyogenes}</td>
<td>Heat, humidity, obesity, maceration of the skin, diabetes mellitus, bad hygiene</td>
<td>Red/brown lesions with irregular well-defined, slightly scaling Mild pruritus</td>
<td>Clinical&lt;br&gt;Confirmation: Wood's lamp: reddish fluorescence\textsuperscript{a}&lt;br&gt;Confirmation: methylene/Gram blue staining\textsuperscript{a}</td>
<td>Topical with erythromycin, clindamycin, mupirocin, imidazoles or Whitfield's solution\textsuperscript{6} 2 times a day&lt;br&gt;Extensive or persistent cases: oral clarithromycin for 10–14 days</td>
<td></td>
</tr>
<tr>
<td>Hidradenitis\textsuperscript{17,22}</td>
<td>Axilla, groins, perineum</td>
<td>\textit{S. aureus}, Oropharyngeal anaerobes</td>
<td>Heat, humidity, hormonal changes, poor hygiene</td>
<td>Painful nodules causing abscesses leaving hypertrophic scars or fistulas&lt;br&gt;Very frequent recurrence</td>
<td>Clinical&lt;br&gt;Confirmation: culture\textsuperscript{a}</td>
<td>Strict hygiene + topical antiseptics/antibiotics + early oral amoxicillin–clavulanic acid/clindamycin</td>
<td></td>
</tr>
<tr>
<td>Disease</td>
<td>Location</td>
<td>Aetiology</td>
<td>Predisposing factors</td>
<td>Clinical analysis</td>
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</tbody>
</table>
| **Neonatal omphalitis**<sup>20</sup> | Umbilicus or umbilical cord        | Gram-negative bacilli                 | Delivery at home or areas with poor health resources, improper care of cord rupture of membranes, etc. | Erythema, oedema and bad periumbilical odour, sometimes accompanied by secretion | Clinical Confirmation: exudate culture<sup>c</sup> | Mild: —chlorhexidine + mupirocin (Moderate—serious: —admission, intravenous antibiotic therapy)
| **Acute paronychia**<sup>18</sup>   | Nail folds                        | *S. aureus* or mixed aerobe flora (*S. pyogenes, Pseudomonas sp.*) or anaerobe | Microtrauma, humidity, irritants (onychophagia, finger suction), contact dermatitis | Warn fold, red, swollen, painful and frequently with pus | Clinical Confirmation: culture<sup>a</sup> | Superficial: warm, moist dressings, 2–3 times a day Deep: incision and drainage + oral amoxicillin–clavulanic acid or cefadroxil 7 days
| **Periporitis**<sup>22,24</sup>    | Occipital region, big folds, back and glutei | *S. aureus*                          | Humidity, lack of hygiene, malnutrition, chronic diseases | Mild form: miliaria pustulosa Serious form: nodules, abscesses | Clinical Confirmation: culture<sup>a</sup> | Avoid hyperhidrosis Mild form: antiseptics (zinc sulfate)/mupirocin Serious form: incision and drainage + oral amoxicillin–clavulanic acid + topical mupirocin
| **Pitted keratolysis**<sup>21</sup> | Soles of the feet                  | *Corynebacterium* sp. Others: *Dermatophilus congolensis* and *Kytococcus sedentarius* | Humidity: hyperhidrosis, closed shoes, prolonged submersion in water | Superficial depressions of the epidermis (pitted), sometimes with linear pattern (*sulcatum*), bad odour, sometimes burning, stinging | Clinical Wood’s lamp coral red fluorescence<sup>a</sup> Confirmation: culture<sup>a</sup> | Treatment of hyperhidrosis + wash with antiseptic soap + topical erythromycin, clindamycin, fusidic acid or mupirocin Imidazoles + topical urea or Whitfield solution 2 times a day
| **Keratolysis plantare sulcatum**<sup>16,24</sup> | Soles of the feet Rare: palms of the hands | *S. agalactiae* | Colonisation of mucosa? | New-borns (2–6 weeks) Facial/submandibular cellulitis Fever, irritability, refusal to feed Ipsilateral acute otitis media Bacteraemia: sepsis/meningitis | Clinical Aspirate culture Haemoculture Lumbar puncture: Gram staining, culture |

<sup>a</sup> Not necessary in typical cases.
<sup>b</sup> Whitfield’s solution: 6–12% benzoic acid and 3–6% Vaseline-based salicylic acid.
<sup>c</sup> Not so useful due to high risk of contamination. Always assess clinical context.
no adjuvant antibiotic therapy\textsuperscript{35-38} (A-II). Systemic antibiotic treatment is recommended in other localised infections, as adjuvant if drainage has been incomplete, if there is intense local infection (abscesses of more than 5 cm) and in patients under 2 years of age or with other risk factors (immunosuppression, moderate-serious atopic dermatitis, etc.) (A-III).

The drug of first choice is oral clindamycin, which also inhibits the production of LPV\textsuperscript{35} (A-II). Trimethoprim–sulfamethoxazole is an effective alternative in areas where there is high resistance to clindamycin (A-II), in the event of intolerance, or when capsules are not suitable for the patient. Linezolid must be avoided in mild-moderate infections, because it is expensive, has potential adverse effects and leads to development of resistances. The presence of LPV does not require changes in the therapeutic approach although it is associated with a greater need for incision and surgical drainage.\textsuperscript{35,37}

If empirical treatment is initiated with any of these drugs, changing to a betalactam is recommended as soon as its sensitivity can be proved.

**Study and treatment of carriers**

It is estimated that 19.3% of the Spanish population is colonised by S. aureus, of which only 1.3% are MRSA.\textsuperscript{39} The most frequent areas of colonisation are the nostrils, but axilla inguinal and intestinal carriers have also been described.\textsuperscript{40} Considering the low incidence of MRSA in paediatrics, the systematic study of persons living with the patient is not recommended, and should be limited to outbreaks in limited communities (families, institutions, etc.) and recurrent infections (C-III). In these cases, decolonisation in the child and carriers living in their household should consist of nasal mupirocin every 12 h and washing the body and hair with 4% chlorhexidine soap for 5–10 days (C-III). In the event of therapeutic failure with mupirocin, topical fusidic acid every 12 h along with oral trimethoprim–sulfamethoxazole for 7 days should be used (C-III).

**Prevention**

The main transmission pathway seems to be intimate skin-to-skin contact.\textsuperscript{40} General prevention measures consist in minimising the risk of skin trauma (use of protection during sports activities, etc.) (C-III), keeping wounds clean and covered (A-III), frequent hand washing and body hygiene (A-III), avoiding sharing towels and clothes (A-III) and proper disposal of contaminated objects (A-III).
**Conflict of interest**

The authors declare that there are no conflicts of interest.

**References**


