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

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Abstract Skin infections are a common cause for dermatological consultations in the paediatric 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
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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 empírico 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.

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,[1] 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).[2]

Antibiotic treatment of skin infections

In general, the antibiotic treatment of skin infections is empirical.[3] Blood or aspirate cultures, biopsies and swabs are only recommended under certain circumstances (chemotherapy, neutropoenia, 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.[4]

Systemic antibiotic therapy is reserved for certain clinical forms (cellulitis), extensive forms, rapid progression or

Table 1   Strength of recommendation and quality of evidence.

Strength of recommendation	
A	Good evidence to support a recommendation for use
B	Moderate evidence to support a recommendation for use
C	Poor evidence to support a recommendation
D	Moderate evidence to support a recommendation against use
E	Good evidence to support a recommendation against use
Quality of the evidence	
I	Evidence from at least one properly randomised, controlled trial
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
III	Evidence from opinions of respected authorities, based on clinical experience, descriptive studies, or reports of expert committees

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 cloxacilin, although its pharmacological characteristics (administration every 6 h, low bioavailability and bad taste) make amoxicillin–clavulanic acid and first-generation cephalosporin (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 cephalosporin.

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

At the time of drafting this document, neither cefadroxil nor cefalexin, the only first-generation cephalosporin with oral presentation, are sold in Spain in suspension form, and only 500 mg capsules are available. Under these circumstances, second-generation cephalosporin (cefuroxime, cefaclor), despite being slightly less active against gram-positive cocci than first-generation cephalosporin, 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 meliceric 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 cephalosporin, or cloxacilin is recommended.[5]

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*.[6] Incidence peaks at 3–5 years of age, predominantly in males (70%), although prevalence is the same in both sexes if vulvovaginitis is included,[8] which together with balanoposthitis constitute what is known as perineal disease.

It manifests as a non-elevated 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.[9] Boys may present with balanoposthitis and girls with vulvovaginitis with secretion and dysuria.[10] 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.[6]

Patients or members of the household may have presented acute pharyngoamigdalitis or a skin infection[7] in the previous 3 months or simultaneously with perianal dermatitis, or be pharyngeal or perianal carriers of *S. pyogenes*.[7,8] Recurrence after treatment with penicillin or amoxicillin is frequent (up to 37%),[11] 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[10] or if a relative has presented acute pharyngoamigdalitis or perianal dermatitis.[7]

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)[7] 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).[10] 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[11] for 7–10 days (C-II). The association of oral and topic treatment seems to decrease recurrences (A-II).[6]

Infections of hair follicles: folliculitis, furuncle and carbuncle

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

Clinically, patients present with lesions on hair-bearing sites on the face, neck, armpits or glutei, ranging from a papule-vesicle with a erythematosus base (folliculitis, which affects the superficial hair follicle), to tender erythematosus 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 erythematosus 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.

Treatment 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, cloxacilin, 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.

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 lymphagitis 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 rabies, tetanus and other diseases transmitted through wounds.

Table 2 Clinical forms of most frequent bacterial skin infections according to their location of preference.

Anatomical location		Infection
Scalp		Abscesses in new borns
Fingers		Paronychia
		Blistering distal dactylitis
Lower limbs		Cellulitis
		Erysipelas
		Folliculitis by *P. aeruginosa* (hot tubs, depilation)
Lymph nodes		Adenophyllum
Sweat glands	Eccrine	Periporitis
	Apocrine	Hydrosadenitis
Lips		Angular cheilitis
Cheeks		Erysipelas
		Bacterial cellulitis: pneumococci, *Haemophilus influenzae* type b
		Cellulitis-adenitis syndrome by *S. agalactiae*
Umbilicus		Neonatal omphalitis
Outer ear		Auricular chondritis (piercing)
Eyelids		Periorbital cellulitis
Perineum	Perianal	
	Balanopreputial	Perianal bacterial disease
	Vulvovaginal	
Sole of the foot		Infection by *P. aeruginosa* (puncture wound, shoes)
		Pitted keratolysis (keratolysis plantare *sulcatum*)
Skin folds		Erythrasma by *Corynebacterium minutissimum*
		Intertrigo by *S. pyogenes*

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.[25] 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 multidrug-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.[3] 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 superinfection of wounds, etc.), topical treatment with mupirocin is usually sufficient[35] (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.

Disease	Location	Aetiology	Predisposing factors	Clinical analysis	Diagnosis	Treatment
Blistering distal dactylitis[16–18]	Soft part of the fingers Infrequent: other surfaces of palms and soles	*S. pyogenes* Less frequent: *S. aureus, S. agalactiae*	Wounds, atopic dermatitis, humidity	Blister with purulent fluid, slightly tender, with erythematous base No general symptoms	Clinical Confirmation: culture[a]	Incision and drainage + amoxicillin–clavulanic acid 10 days If there is poor response cephalosporin, clindamycin Mupirocin: optional (not in monotherapy)
Ecthyma[17–19]	Lower and upper limbs	*S. pyogenes, S. aureus* copathogen?	Pruritic lesions: bites, scabies, pediculosis; poor hygiene; malnutrition	Ulcer with black crust that becomes chronic and heals leaving a scar Complications: cellulitis lymphagitis	Clinical Confirmation: Gram stain and culture of exudate or biopsy of deep tissue	Removal of crusts with moist dressings Topical fusidic acid/mupirocin if the lesion is small (<2 cm) Oral 1st generation cephalosporin/amoxicillin–clavulanic acid/macrolide 7 days[35] Consider admission
Ecthyma gangrenosum[17,18]	Perineum, glutei, lower limbs, apocrine areas	*P. aeruginosa* Other bacteria, fungi and virus (*immunosuppressed*)	Immunosuppression (serious neutropenia), healthy infants	Necrotic ulcer with black crust (bacterial vasculitis of small veins) Sepsis by *P. aeruginosa*	Clinical Confirmation: culture of the lesions, haemocultures, skin biopsy	Early systemic treatment (intravenous) anti-*Pseudomonas* sp.
Erythrasma[18]	Humid body folds: interdigital spaces of feet, groins, armpits, infra-mammalian and interglutei areas	*Corynebacterium minutissimun*, mixed flora, *S. pyogenes*	Heat, humidity, obesity, maceration of the skin, diabetes mellitus, bad hygiene	Red/brown lesions with irregular well-defined, slightly scaling Mild pruritus	Clinical Wood's lamp: reddish fluorescence[a] Confirmation: methylene/Gram blue staining[a]	Topical with erythromycin, clindamycin, mupirocin, imidazoles or Whitfield's solution[b] 2 times a day Extensive or persistent cases: oral clarithromycin for 10–14 days
Hidradenitis[17,22]	Axilla, groins, perineum	*S. aureus*, Oropharyngeal anaerobes	Heat, humidity, hormonal changes, poor hygiene	Painful nodules causing abscesses leaving hypertrophic scars or fistulas Very frequent recurrence	Clinical Confirmation: culture[a]	Strict hygiene + topical antiseptics/antibiotics + early oral amoxicillin–clavulanic acid/clindamycin

Table 3 (*Continued*)

Disease	Location	Aetiology	Predisposing factors	Clinical analysis	Diagnosis	Treatment
Neonatal omphalitis[20]	Umbilicus or umbilical cord	Gram-negative bacilli *S. aureus* *S. pyogenes* Less frequent: anaerobes, mixed infections	Delivery at home or areas with poor health resources, improper care of cord prematurity, chorioamnionitis, premature rupture of membranes, etc.	Erythema, oedema and bad periumbilical odour, sometimes accompanied by secretion	Clinical Confirmation: exudate culture[c]	Mild: –chlorhexidine + mupirocin Moderate–serious: –admission, intravenous antibiotic therapy
Acute paronychia[18]	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[a]	Superficial: warm, moist dressings, 2–3 times a day Deep: incision and drainage + oral amoxicillin–clavulanic acid or cefadroxil 7 days
Periporitis[22,24]	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	Avoid hyperhidrosis Mild form: antiseptics (zinc sulfate)/mupirocin Serious form: incision and drainage + oral amoxicillin–clavulanic acid + topical mupirocin
Pitted *keratolysis*[21] Keratolysis plantare *sulcatum*[16,24]	Soles of the feet Rare: palms of the hands	*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[a] Confirmation: culture[a]	Treatment of hyperhidrosis + wash with antiseptic soap + topical erythromycin, clindamycin, fusidic acid or mupirocin Imidazoles + topical urea or Whitfield solution[c] 2 times a day
Cellulitis-adenitis syndrome[25]	Face and neck: cheeks, submandibular region	*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	Admission and intravenous treatment

[a] Not necessary in typical cases.
[b] Whitfield's solution: 6–12% benzoic acid and 3–6% Vaseline-based salicylic acid.
[c] Not so useful due to high risk of contamination. Always assess clinical context.

Table 4 Posology of most used oral antibiotics in bacterial skin infections.[a]

Drugs	Dosage	Maximum permissible dose
Penicillin V	25–50 mg/kg/d every 6 h	3 g/d
Cloxacilin	50 mg/kg/d every 6 h	4 g/d
Amoxicillin (±clavulanic acid)	50–60 mg/kg/d every 8–12 h	3 g/d
Cefadroxil	25–50 mg/kg/d every 12 h	1 g/d
Cefalexin	20–25 mg/kg/d every 6 h	2 g
Cefuroxime	3 months–5 years: 15 mg/kg/d every 12 h 5–12 years: 125 mg/12 h >12 years: 250 mg/12 h	4 g/d
Cefaclor	20 mg/kg/d every 8 h	1 g/d
Clarithromycin	15 mg/kg/d every 12 h	1 g/d
Josamycin	30–50 mg/kg/d every 12 h	1 g/d
Midecamicin	35–50 mg/kg/d every 8–12 h	1.8 g/d
Clindamycin	10–30 mg/kg/d every 6 h	1.8 g/d
Trimethoprim–sulfamethoxazole (TMP–SMX)	6–12 mg TMP/kg/d every 12 h 30–60 mg SMX/kg/d every 12 h	TMP: 320 mg/d SMX: 1600 mg/d

[a] General duration of the treatment: 7–10 days, except for perianal bacterial dermatitis treated with penicillin or amoxicillin (10–21 days) and erysipelas (10–14 days).

no adjuvant antibiotic therapy[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 atopical dermatitis, etc.) (A-III).

The drug of first choice is oral clindamycin, which also inhibits the production of LPV[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.[3,31]

If empirical treatment is initiated with any of these drugs, changing to a betalactamic 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.[39] The most frequent areas of colonisation are the nostrils, but axilla inguinal and intestinal carriers have also been described.[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[3] (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[3,35] (C-III).

Prevention

The main transmission pathway seems to be intimate skin-to-skin contact.[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[35,40] (C-III).

Table 5 Indications of antibiotic prophylaxis in bites and puncture wounds.[25]

Cat and human bites
Dog bites with delay in assistance of >8 h
Dog bites in hands, feet and face
Deep puncture wounds
Wounds with crushing, oedema or requiring debridement
Wounds with infection of tendons, muscles or joints
Wounds with underlying fractures
Puncture wounds in the sole of the foot with delay in assistance of >24 h, with extensive involvement or devitalised tissue
Underlying disease: immunodepression, risk of endocarditis, chronic pneumopathy, chronic hepatopathy, diabetes mellitus

Taken from Bangert et al.[25].

Table 6 Infections secondary to bites and puncture wounds.[23]

	Microorganisms	Treatment	Allergy to penicillin.
Dog bite	*Pasteurella canis* *Capnocytophaga canimorsus*	Amoxicillin–clavulanic acid	No anaphylaxis: – 2nd or 3rd generation cephalosporin + clindamycin Anaphylaxis: – Trimethoprim–sulfamethoxazole + clindamycin
Cat bite	*Pasteurella multocida* *Pasteurella septica* *Corynebacterium* *Bartonella henselae*		
Dog and cat bites	*Staphylococcus* *Streptococcus* *Enterococcus* Anaerobes		
Human bite	*Streptococcus anginosus* *Staphylococus aureus* *Eikenella corrodens* *Fusobacterium* *Peptostreptococcus* *Prevotella*		
Puncture wound	*Staphylococcus aureus* Beta-haemolytic Streptococcus Anaerobes *Pseudomonas aeruginosa*	Wound for <7 days Cefadroxil Amoxicillin–clavulanic acid Wound on foot for >7 days Hospital admission Intravenous antibiotic therapy antipseudomonic	Wound for <7 days No anaphylaxis: –cefadroxil Anaphylaxis: –clindamycin

Taken from Álvez González.[23]

Conflict of interest

The authors declare that there are no conflicts of interest.

References

1. Stevens DL, Bisno AL, Chambers HF, Patchen E, Goldstein AJ, Gorbach SL, et al. Practice guidelines for the diagnosis and management of skin and soft tissue infections: 2014 update by the Infectious Diseases Society of America. Clin Infect Dis. 2014;59:e10–52.
2. Khan AR, Khan S, Zimmerman V, Baddour LM, Tleyjeh IM. Quality and strength of evidence of the Infectious Diseases Society of America clinical practice guidelines. Clin Infect Dis. 2010;51:1147–56.
3. Frick MA, Moraga-Llop FA, Bartolomé R, Larrosa N, Campins M, Roman Y, et al. Infecciones por *Staphylococcus aureus* resistente a meticilina adquirido en la comunidad en niños. Enferm Infecc Microbiol Clin. 2010;28:675–9.
4. Rojo Conejo P. Infecciones por estafilococo. In: Moro M, Málaga S, Madero L, editors. Tratado de pediatría Cruz. Madrid: Ed. Panamericana; 2014. p. 771–6.
5. Rodríguez Fernández R. Infecciones por estreptococo del grupo A. In: Moro M, Málaga S, Madero L, editors. Tratado de pediatría Cruz. Madrid: Ed. Panamericana; 2014. p. 776–80.
6. Heath C, Desai N, Silverberg NB. Recent microbiological shifts in perianal bacterial dermatitis: *Staphylococcus aureus* predominance. Pediatr Dermatol. 2009;26:696–700.
7. Echeverría Fernández M, López-Menchero Oliva JC, Marañón Pardillo R, Mínguez Navarro C, Sánchez Sánchez C, Vázquez López P. Aislamiento del estreptococo beta hemolítico del grupo A en niños con dermatitis perianal. An Pediatr (Barc). 2006;64:153–7.
8. Olson D, Edmonson MB. Outcomes in children treated for perineal group A beta-hemolytic streptococcal dermatitis. Pediatr Infect Dis J. 2011;30:933–6.
9. Lehman R, Pinder S. Streptococcal perianal infection in children. BMJ. 2009;338:b1517.
10. Herbst R. Perineal streptococcal dermatitis/disease: recognition and management. Am J Clin Dermatol. 2003;4: 555–60.
11. Meury SN, Erb T, Schaad UB, Heininger U. Randomized, comparative efficacy trial of oral penicillin versus cefuroxime for perianal streptococcal dermatitis in children. J Pediatr. 2008;153:799–802.
12. Ensinck GN, Casanueva E, Sguassero Y. Comité Nacional de Infectología de la SAP. Infecciones de la piel y partes blandas en pediatría: consenso sobre diagnóstico y tratamiento. Arch Argent Pediatr. 2014;112:e96–102.
13. Lawrence HS, Nopper AJ. Superficial bacterial skin infections and cellulitis. In: Long S, Pickering LK, Prober CG, editors.

Principles and practice of pediatric infectious diseases. 4th ed. Philadelphia: Churchill Livingstone; 2012. p. 427–35.

14. Jackson MA. Skin infections bacterial skin infections. In: Cherry JD, Harrison GJ, Kaplan SL, editors. Feigin and Cherry's textbook of pediatric infectious diseases. 7th ed. Philadelphia: Elsevier; 2014. p. 772–81.

15. Millett CR, Halpern AV, Reboli AC, Heymann WR. Bacterial diseases. In: Bolognia JL, Jorizzo JL, Schaffer JV, editors. Dermatology. 3rd ed. Philadelphia: Elsevier; 2012. p. 1187–220.

16. Larru B, Gerber JS. Cutaneous bacterial infections caused by *Staphylococcus aureus* and *Streptococcus pyogenes* in infants and children. Psychiatr Clin N Am. 2014;61:457–78.

17. Santos Sebastián M. Infecciones cutáneas y de partes blandas. In: Moro M, Málaga S, Madero L, editors. Tratado de pediatría Cruz. Madrid: Ed. Panamericana; 2014. p. 761–7.

18. Moraga Llop FA, Tobeña Rue M. Infecciones bacterianas cutáneas y de partes blandas. In: Delgado Rubio A, editor. Enfermedades infecciosas en pediatría. Madrid: McGraw Hill; 2008. p. 651–62.

19. Matiz C, Friedlander SF. Subcutaneous tissue infections and abscesses. In: Long S, Pickering LK, Prober CG, editors. Principles and practice of pediatric infectious diseases. 4th ed. Philadelphia: Churchill Livingstone; 2012. p. 454–62.

20. Darmstadt GL, Zaidi AKM, Stoll BJ. Neonatal infections: a global perspective. Neonatal infections: omphalitis. In: Remington JS, Klein JO, Wilson CB, Nizet V, Maldonado YA, editors. Infectious diseases of the fetus and newborn. 7th ed. Philadelphia: W.B. Saunders; 2011. p. 30–1.

21. García-Cuadros R, Figueroa-Núñez del Prado Y. Abanico clínico de la queratólisis punctata. Dermatol Peru. 2006;16:233–8.

22. Thorell EA. Cervical lymphadenitis and neck infections. In: Long S, Pickering LK, Prober CG, editors. Principles and practice of pediatric infectious diseases. 4th ed. Philadelphia: Churchill Livingstone; 2012. p. 135–47.

23. Álvez González F. Infecciones por mordeduras y heridas punzantes. Protocolos de infectología de la Asociación Española de Pediatría; 2011. Disponible en: http://www.aeped.es/sites/default/files/documentos/mordeduras.pdf [consultado December 2014].

24. Chaudhry MA, MacNamara AF, Clark S. Is the management of dog bite wounds evidence based? A postal survey and review of the literature. Eur J Emerg Med. 2004;11:313–7.

25. Bangert S, Levy M, Hebert AA. Bacterial resistance and impetigo treatment trends: a review. Pediatr Dermatol. 2012;29:243–8.

26. Broseta A, Chaves F, Rojo P, Otero JR. Emergencia de un clon de *Staphylococcus aureus* resistente a meticilina de origen comunitario en la población pediátrica del sur de Madrid. Enferm Infecc Microbiol Clin. 2006;24:31–5.

27. Daskalaki M, Rojo P, Marín-Ferrer M, Barrios M, Otero JR, Chaves F. Panton–Valentine leukocidin-positive *Staphylococcus aureus* skin and soft issue infections among children in an emergency department in Madrid, Spain. Clin Microbiol Infect. 2010;16:74–7.

28. Chaves F. Emergencia de infecciones pediátricas por *Staphylococcus aureus* resistente a meticilina asociadas a la comunidad: ¿debemos dar la alerta? Enferm Infecc Microbiol Clin. 2010;28:672–4.

29. Casado-Verrier B, Gómez-Fernández C, Paño-Pardo JR, Gómez-Gil R, Mingorace-Cruz J, Moreno-Alonso de Celada R, et al. Prevalencia de infecciones de piel y tejidos blandos producidas por *Staphylococcus aureus* resistentes a meticilina comunitario en Madrid. Enferm Infecc Microbiol Clin. 2012;30:300–6.

30. Barrios-López M, Rojo-Conejo P, Gómez-González C, Cháves-Sánchez F. Infecciones neonatales por *Staphylococcus aureus* de inicio en la comunidad. Enferm Infecc Microbiol Clin. 2013;31:316–8.

31. Rojo P, Barrios M, Palacios A, Gómez C, Chaves F. Community-associated *Staphylococcus aureus* in children. Expert Rev Anti Infect Ther. 2010;8:541–54.

32. Barrios López M, Gómez González C, Orellana MA, Chaves F, Rojo P. *Staphylococcus aureus* abscesses: methicillin-resistance or Panton–Valentine leukocidin presence? Arch Dis Child. 2013;98:608–10.

33. Odell CA. Community-associated methicillin-resistant *Staphylococcus aureus* (CA-MARSA) skin infections. Curr Opin Pediatr. 2010;22:273–7.

34. Jiménez JN, Ocampo AM, Vanegas JM, Rodríguez EA, Mediavilla JR, Chen L, et al. A comparison of methicillin-resistant and methicillin-susceptible *Staphylococcus aureus* reveals no clinical and epidemiological but molecular differences. Int J Med Microbiol. 2013;303:76–83.

35. Liu C, Bayer A, Cosgrove SE, Daum RS, Fridkin SK, Gorwitz RJ, et al. Clinical practice guidelines for the treatment of methicillin-resistant *Staphylococcus aureus* infections in adults and children. Clin Infect Dis. 2011;52:285–92.

36. Forcade NA, Wiederhold NP, Ryan L, Talbert RL, Frei CR. Antibacterial as adjuncts to incision and drainage for adults with purulent methicillin-resistant *Staphylococcus aureus* (MRSA) skin infections. Drugs. 2012;72:339–51.

37. Hurley HJ, Knepper BC, Price CS, Mehler PS, Burman WJ, Jenkins TC. Avoidable antibiotic exposure for uncomplicated skin and soft tissue infections in the ambulatory care setting. Am J Med. 2013;126:1099–106.

38. Lee MC, Ríos AM, Fonseca Aten M, Mejías A, Cavouti D, McCracken GH Jr, et al. Management and outcome of children with skin and soft tissue abscesses caused by community-acquired methicillin-resistant *Staphylococcus aureus*. Pediatr Infect Dis J. 2004;23:123–7.

39. Den Heijer CDJ, van Bijnen EME, Pajet WJ, Pringle M, Goossens H, Bruggeman CA, et al. Prevalence and resistance of commensal *Staphylococcus aureus*, including methicillin-resistant *S. aureus*, in nine European countries: a cross-sectional study. Lancet Infect Dis. 2013;13:409–15.

40. Cercenado E, Ruiz de Gopegui E. *Staphylococcus aureus* resistente a la meticilina de origen comunitario. Enferm Infecc Microbiol Clin. 2008;26 Suppl. 13:19–24.