Title

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Journal

The Journal of clinical and aesthetic dermatology, 9(6)

ISSN

1941-2789

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Publication Date

2016-06-01

Peer reviewed

Status Report from the Scientific Panel on Antibiotic Use in Dermatology of the American Acne and Rosacea Society

Part 3: Current Perspectives on Skin and Soft Tissue Infections with Emphasis on Methicillin-resistant *Staphylococcus aureus*, Commonly Encountered Scenarios when Antibiotic Use May Not Be Needed, and Concluding Remarks on Rational Use of Antibiotics in Dermatology

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ABSTRACT

In this third article of the three-part series, management of skin and soft tissue infections is reviewed with emphasis on new information on methicillin-resistant *Staphylococcus aureus*. Due to changes in the evolution of methicillin-resistant *Staphylococcus aureus* clones, previous distinctions between healthcare-acquired methicillin-resistant *Staphylococcus aureus* and community-acquired methicillin-resistant *Staphylococcus aureus* are currently much less clinically relevant. Many nosocomial cases of methicillin-resistant *Staphylococcus aureus* infection are now caused by community-acquired methicillin-resistant *Staphylococcus aureus*, with changing patterns of antibiotic susceptibility and resistance. Also reviewed are clinical scenarios where antibiotics may not be needed and suggestions for optimal use of antibiotic therapy for dermatologic conditions, including recommendations on perioperative antibiotic use.
(*J Clin Aesthet Dermatol.* 2016;9(6):17–24.)

A broad range of topics were reviewed in the first two parts of this three-part article series.[1,2] Examples of information discussed in the first two articles include overall patterns of antibiotic exposure, antibiotic prescribing practices, sequelae of antibiotic consumption that may be clinically relevant, reported effects of antibiotic use on the human microbiome, and data on microbiologic effects associated with specific drugs commonly used in dermatology. This third and final article of the series opens with a discussion on bacterial skin and soft tissue infections (SSTIs) commonly encountered in ambulatory dermatology practice, focusing primarily on methicillin-

DISCLOSURE: This article was written solely by the authors. Prior to journal submission, the article was reviewed by the AARS Board of Directors and the AARS Education Committee. There was no review or contribution to the authorship of this article by individuals from any company or agency of any company. The subjects included in the content of this article were presented at the third SPAUD Meeting held in Las Vegas, Nevada in September 2014 by the authors of this article series. The content of the presentations at the SPAUD meeting were developed solely by the authors. Physician Resources provided support with literature searches and article procurement, audiovisual needs, and logistical support for the meeting, which was funded by educational grants from Galderma, Allergan, Valeant, Bayer, and Promius.
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resistant *Staphylococcus aureus* (MRSA). This is followed by a summary of clinical scenarios where antibiotics may not be needed. The article concludes with suggestions on how dermatologists may rationally incorporate antibiotic therapy when their use is deemed to be necessary and methods to limit or avoid antibiotic use to help sustain their efficacy and reduce the emergence of antibiotic-resistant bacteria.

The most common bacterial SSTI encountered in outpatient dermatology practices in the United States are impetigo, folliculitis, furunculosis, cutaneous abscess, cellulitis, and infected eczematous dermatitis.[3] The latter is sometimes referred to as "crusted eczema" or "secondary impetiginization," a consequence of high density colonization of eczematous skin with *S. aureus* in individuals with atopic dermatitis.[4,5] Most SSTIs seen in outpatient clinical practice are caused by methicillin-susceptible *S. aureus* (MSSA), or by MRSA, including folliculitis, furunculosis, cutaneous abscess, and eczematous dermatitis with secondary infection; streptococcal infection is often present in cases of impetigo or cellulitis, and in some cases of folliculitis.[5–7] Although less common, SSTIs caused by Gram-negative bacteria may be seen in clinical practice, such as Gram-negative folliculitis, hot tub folliculitis, cellulitis, and infected wounds.[8,9] The causative organism in perioperative and traumatic wound infections is highly dependent on the type of skin injury and the anatomic location.

Clinical evaluation and thoughtful judgment are important components when selecting treatment for SSTIs. Management of uncomplicated SSTIs is usually successful with properly selected oral antibiotic therapy and/or appropriate physical care when indicated (i.e., wound cleansing, incision and drainage [I&D] of an abscess).[6,10,11] In some cases of small abscesses that are not associated with cellulitis or systemic symptoms, I&D alone without antibiotic therapy may be curative. Localized cases of impetigo or folliculitis may respond to topical antibiotic therapy alone.[11,12] Whenever possible, bacterial culture and susceptibility testing is recommended to identify the specific bacterial pathogen and confirm the diagnosis of infection.[10,11,13,14] One can never be faulted for confirming the diagnosis and identifying the causative bacterium. However, failure to do so may confound cases where there is poor response to the empiric antibiotic therapy that was initially prescribed.

WHAT IS THE RELEVANCE OF HEALTHCARE-ACQUIRED MRSA VERSUS COMMUNITY-ACQUIRED MRSA?

MRSA was first identified in 1961 and for the ensuing three decades, a limited number of healthcare-acquired clonal MRSA strains (HA-MRSA) circulated outside the United States, primarily in Europe.[15,16] For the purpose of clarification for the reader, in this paper, the designation of HA-MRSA refers to what in older literature was called "hospital-acquired MRSA," but more accurately refers to clones of MRSA that have their origin (onset) from within hospitals and healthcare facilities. Strains of HA-MRSA were reported in the United States during late 1970s, were endemic in hospital and long-term care facilities within less than a decade, and spread to become a global pandemic, which remains through current times.[16] The limited availability of antibiotics effective for HA-MRSA led to a marked increase in parenteral vancomycin use, followed by emergence of *S. aureus* strains that were less responsive or nonresponsive to vancomycin.[16]

Strains of community-acquired MRSA (CA-MRSA) that were genetically distinct from HA-MRSA first emerged in Australia in the early 1990s, and appeared within the United States by 1995, primarily in children.[15,16] Unlike HA-MRSA, CA-MRSA lacked the epidemiologic and clinical risk factors associated with HA-MRSA, especially exposures within hospitals and healthcare facilities. Over time, CA-MRSA strains continued to become widespread globally and share similar epidemiologic features and parallel microbiologic evolutionary characteristics. However, some individual clones varied among geographic locations, and CA-MRSA genotypes and antibiotic susceptibilities differed from HA-MRSA strains.[15,16]

The numerous genetic lineages of CA-MRSA strains that have emerged from different countries globally have steadily swept across multiple continents, with the USA300 becoming the predominant strain in the USA.[17] Coupled with the increase in CA-MRSA strains inter-mingling from various geographic sources, traditional distinctions between HA-MRSA and CA-MRSA based on clinical epidemiology and antibiotic susceptibility are currently much less relevant, as there has been a marked and continued increase in nosocomial CA-MRSA infections and multi-drug resistant CA-MRSA strains.[16–27] Authorities in microbiology and infectious diseases now favor defining CA-MRSA through genotypic-based descriptions and strain clustering.[24,28] Laboratory methodologies used to classify MRSA are pulsed-field gel electrophoresis (PFGE), multilocus sequence typing (MLST; 7 genes [ST types]), spa typing (protein A gene), and SCCmec typing (1-11).[17]

The important message is that it is no longer relevant to distinguish MRSA as CA-MRSA versus HA-MRSA based on epidemiologic parameters. This major change in distinction between CA-MRSA and HA-MRSA is not surprising given the dynamic landscape of continuously emerging "waves" of evolution of MRSA clones, antibiotic resistance patterns, and clinical presentations of infection.[16,19,20] In fact, CA-MRSA clones are increasingly causing infections that have their onset within hospitals and healthcare facilities, and have been associated with invasive infections, such as pneumonia, septic arthritis, osteomyelitis, sepsis, endocarditis, and SSTIs.[15–17,26]

WHAT ARE THE IMPLICATIONS OF CA-MRSA CLONES SUCH AS USA 300 COMMONLY CAUSING NOSOCOMIAL INFECTIONS?

In addition to healthcare-onset infections, CA-MRSA infections (including USA 300 clones) have been reported in many patient populations, including American Indians,

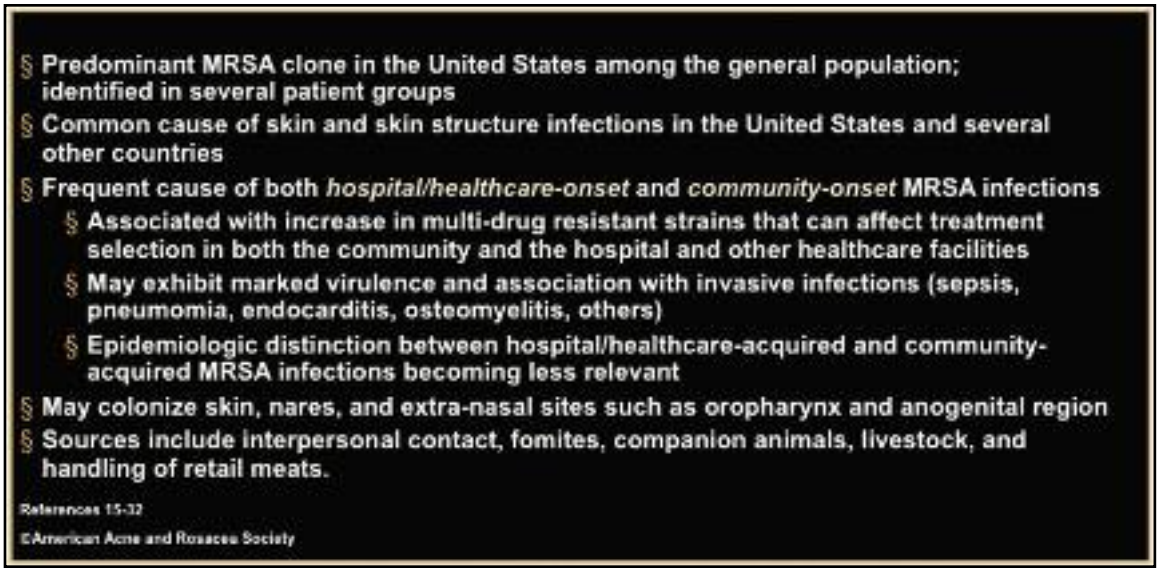

Figure 1. Clinically relevant characteristics of USA 300 MRSA

Pacific Islanders, Alaskan natives, athletes, prison inmates, men who have sex with men, adult emergency room patients, military personnel, children in day care centers, and contacts of individuals with known CA-MRSA infection.[16] However, as multidrug-resistance and increased virulence are now more common among conventional CA-MRSA clones, empiric antibiotic use may be less effective than in the past, supporting the importance of bacterial culture and susceptibility testing before initiating antibiotic therapy.[11,15,15–19,21–26] The emergence of CA-MRSA as a cause of infection in hospitals expands the risk of MRSA infections among patients and healthcare workers within hospitals and long-term care facilities.[19,20] In addition, the common presence of CA-MRSA strains within the hospital setting exposes these bacteria to selection pressure induced by a broader range of antibiotic classes and with greater antibiotic intensity and frequency of exposure, resulting in increased antibiotic resistance to multiple agents.[19] As these same strains also gain and maintain their presence in the outpatient community, changes in antibiotic susceptibility become more prevalent in both the hospital and outpatient settings. This leads to therapeutic challenges for the clinician when selecting antibiotic therapy as new antibiotic-resistant strains become more prevalent over time.[15–21] For example, some USA 300 MRSA isolates have become resistant to a variety of antibiotics, including macrolides (i.e., erythromycin), clindamycin, fluoroquinolones (i.e., levofloxacin), tetracyclines (i.e., doxycycline), mupirocin, and trimethoprim-sulfamethoxazole.[22,23] Conjugative transfer of resistance has been observed between bacterial clones with horizontal plasmid transfer suggested between USA 300 isolates and also between USA 300 and USA 100 MRSA clones.[22]

The predominant MRSA clone in the United States, USA 300, and some other clones, have gained a major foothold in many countries around the world.[15–17,21,29] USA300 and a closely related variant (Latin America) have been identified in multiple countries on five continents, and is the dominant CA-MRSA clone in at least five countries, including the United States.[21] USA 300 MRSA clones are a major cause of SSTIs (i.e., furunculosis, abscess, cellulitis), exhibit the ability to colonize both nares and extra-nasal sites (i.e., oropharynx, perineum), and are capable of surviving on fomites.[22,23,29] Potential sources of exposure to MRSA include interpersonal contact, fomites, livestock, retail meats, and bidirectional transmission via companion animals.[17,24–26,30–32] Clonal transmission of methicillin-resistant coagulase-negative staphylococci isolated from retail meats (beef, chicken, turkey) that serve as a resistance gene (mecA) reservoir, and horizontal resistance gene (SCCmec) transfer among staphylococcal species, have both been documented.[31] Clinically relevant characteristics of USA 300 MRSA are outlined in Figure 1.

WHAT ARE CURRENT RECOMMENDATIONS FOR THE OUTPATIENT MANAGEMENT OF SSTIs AND MRSA?

There is no doubt that the high prevalence of MRSA causing SSTIs encountered in ambulatory practice has changed antibiotic practices.[33] The implications of changing antibiotic susceptibility patterns of MRSA that cause infections within the community are not limited to oral antibiotics, as mupirocin resistance has been identified and continues to persist.[22,34] Consecutive, non-duplicate, clinical isolates of *S. aureus* (n=98), and coagulase-negative staphylococci (CoNS) (n=45) obtained over a six-month period in 2014 from SSTIs were studied; high-level mupirocin resistance was noted in 8.2 percent of *S. aureus* isolates and in 15.6 percent of CoNS isolates, while low-level mupirocin resistance was found in 17 percent of *S. aureus*

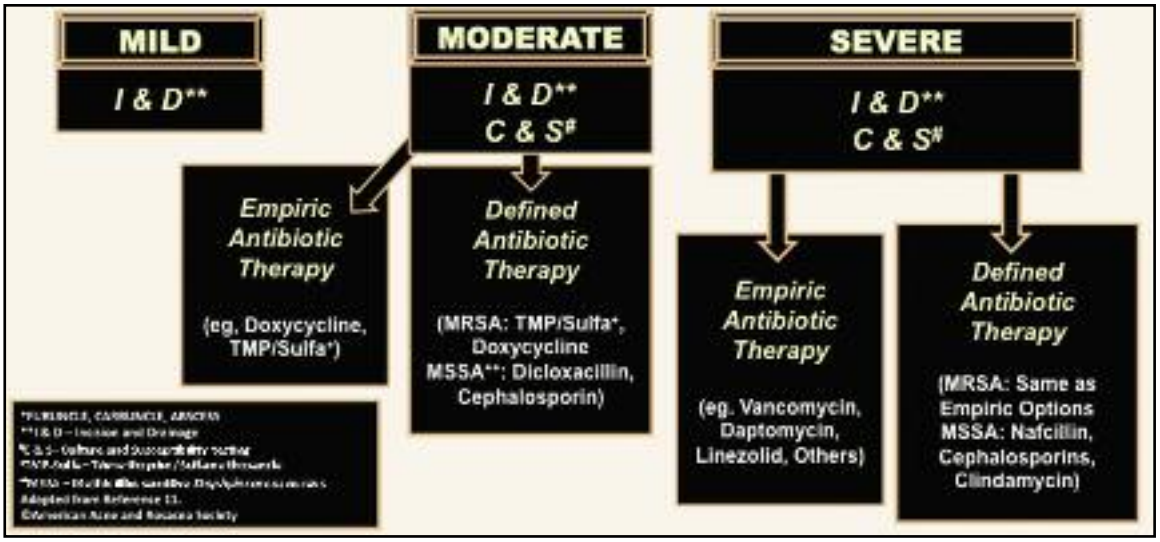

Figure 2. Management of PURULENT skin and soft tissue infections*

isolates and 8.9 percent of CoNS isolates.[34] The bottom line is that MRSA has increased awareness among many clinicians about thoughtful antibiotic prescribing and concerns about antibiotic resistance.

A complete review of management of SSTIs including MRSA infections is beyond the scope of this article; however, a detail review of more current recommendations follows below.

Over the past decade, multiple publications have presented recommendations for the management of SSTIs, and articles on guidelines for the management of MRSA infections, including SSTIs, have also appeared in the literature.[35] Evaluation of these publications identify both important principles to guide clinicians and also raise questions on applicability depending on individual patient circumstances and clinical judgment. It is suggested that prescribers incorporate a pragmatic approach to empirical antibiotic therapy for SSTI, taking into account patient-related medical risk factors, severity and extent of infection, and consideration of regional patterns of antibiotic resistance.[33,35]

At the Scientific Panel on Antibiotic Use in Dermatology of the American Acne and Rosacea Society (SPAUD) meeting in 2014, the group elected to summarize guidelines from the Infectious Disease Society of America (IDSA) published in 2014 in this article to outline a rational approach to management of the more commonly encountered SSTIs.[11] The reader is encouraged to be aware of antibiotic susceptibility patterns in their locale and to incorporate clinical judgement based on their knowledge and examination of the patient in each case.

The 2014, IDSA guidelines for management of SSTIs caused by MRSA defined objective criteria that may be used to guide clinicians in treatment selection.[11] The grading of severity of MRSA infection that can be used to guide management is based on factoring in the presence or absence of the following specific criteria:

Mild: Purulence; absence of systemic signs; normotensive, immunocompetent

Moderate: Purulence; systemic signs present (fever; tachycardia; tachypnea, leukocytosis); normotensive; immunocompetent

Severe: Purulence; systemic signs present (fever >38ºC, tachycardia >90, tachypnea >24, leukocytosis >12,000 WBC); hypotensive; immunocompromised/immunosuppressed; failure of previous therapy (I&D, antibiotics).

It is interesting to note that previously recognized severity criteria, such as extremes of age, >5cm of perilesional erythema, large abscess size, presence of cellulitis, and an anatomic location that is hard to drain, are not included in the categorization of infection severity.[11] These factors are likely to affect how the individual clinician will determine approach to treatment in addition to the IDSA criteria outlined above as guidelines remain secondary to thoughtful clinical judgement and careful examination of the patient. Figures 2 and 3 depict algorithmic guidelines for the management of purulent and nonpurulent SSTIs, respectively, that incorporate the severity grading described above. The reader is referred to IDSA publications for more comprehensive discussions of SSTI management guidelines, and for reviews of various SSTIs, such as those related to human bites, animal bites, surgical site infections, necrotizing fasciitis, and several others.[11,36]

WHAT ARE SOME COMMON CLINICAL SITUATIONS WHERE ANTIBIOTICS MAY NOT BE NEEDED IN DERMATOLOGIC PRACTICE?

There is no question that when antibiotics are needed to treat an infection, use of the proper antibiotic is usually vital to clearance of infection, with more rapid improvement and reduction in morbidity, and in some cases mortality. On the other hand, unnecessary antibiotic use causes emergence of resistant bacterial strains, can be associated with adverse

Figure 3. Management of NONPURULENT skin and soft tissue infections*

reactions, and increases the cost of therapy. The following is a selected list of clinical situations where use of topical or oral antibiotic therapy may not be needed, with additional information available in other sources.[37,38]

Routine postoperative topical antibiotic use. Available evidence supports the overall recommendation that topical antibiotics are not routinely indicated for postsurgical wound infection prophylaxis after clean and clean-contaminated dermatologic surgeries.[37–41]

Meta-analysis of pooled data from four trials did not show a statistically significant difference between topical antibiotics and petrolatum/paraffin in preventing postsurgical wound infections after dermatologic procedures, with a pooled odds ratio of 0.71 (95% CI, 0.42–1.19) for development of an infection.[39] In a large study (N=1207 wounds) comparing white petrolatum or bacitracin ointment applied daily over 7 to 10 days, contact dermatitis was noted in 0.9 percent of bacitracin-treated patients as compared to none of the patients applying white petrolatum.[40] Wounds that have been reported to be at higher risk for postsurgical infection after dermatologic surgery are those located below the knee or in the groin region, excisional surgeries that invade nasal or oral mucosa, wedge excisions of the lip and ears, nasal skin flaps, some Mohs surgery cases with subsequent repair procedures, and wounds in diabetics or immuno-compromised patients.[37,39,42] In many of these cases, it may be more prudent to utilize an oral antibiotic for prophylaxis if the risk of postsurgical infection is judged to be high and avoidance of infection is a significant priority due to patient-related risk factors; topical therapy alone is less likely to provide adequate prophylaxis in such cases.[39]

Preoperative oral antibiotic prophylaxis. Based on more recent guidelines on perioperative antibiotic prophylaxis and studies evaluating the risk of postsurgical infection after dermatologic surgical procedures, there has been a definite shift away from routine administration of prophylactic antibiotics.[42–45] Survey results obtained from members of the American College of Mohs Surgery in 2012, a group comprised of primarily dermatologists, suggested possible overuse of perioperative antibiotics for prevention of surgical site infections, prosthetic joint infections (PJI), and infective endocarditis (IE), based on current authoritative guidelines from the American Heart Association, American Dental Association, and the American Academy of Orthopedic Surgeons; a high percentage of survey respondents prescribed perioperative antibiotics in patients at potential risk of PJI or IE, even though the surgery did not breach mucosa or involve clinically infected skin.[44] Clinicians are encouraged to utilize current guidelines, however, it is important that they individualize their approach in each case after taking into account all relevant clinical considerations.[42] Figure 4 depicts a decision model for antibiotic prophylaxis in dermatologic surgery, with more comprehensive discussion available in references that are highly clinically relevant.[37,42–45]

Atopic dermatitis. Staphylococcal colonization with *S. aureus* is very common in individuals with atopic dermatitis (AD), with presence on eczematous skin, uninvolved skin, and within anterior nares in 85, 60, and 60 percent of cases, respectively.[4,5,37] Certain strains of *S. aureus* are believed to intiate and/or prolong flares of AD through production of specific toxins and exoproducts, with some suggestion that a threshold for bacterial density correlates directly with AD exacerbation.[5,37] In cases of clinical infection, antibiotic therapy is therapeutically beneficial; however, chronic topical or oral antibiotic therapy is not beneficial in AD and serves only to increase antibiotic resistance.[37,38] When clinical infection (such as cellulitis or "secondary impetigo") is not present, topical corticosteroid therapy and epidermal barrier repair can reduce *S. aureus* density associated with eczematous dermatitis by both reducing cutaneous inflammation and decreasing impaired permeability and antimicrobial barrier dysfunctions seen in AD.[37,46–48]

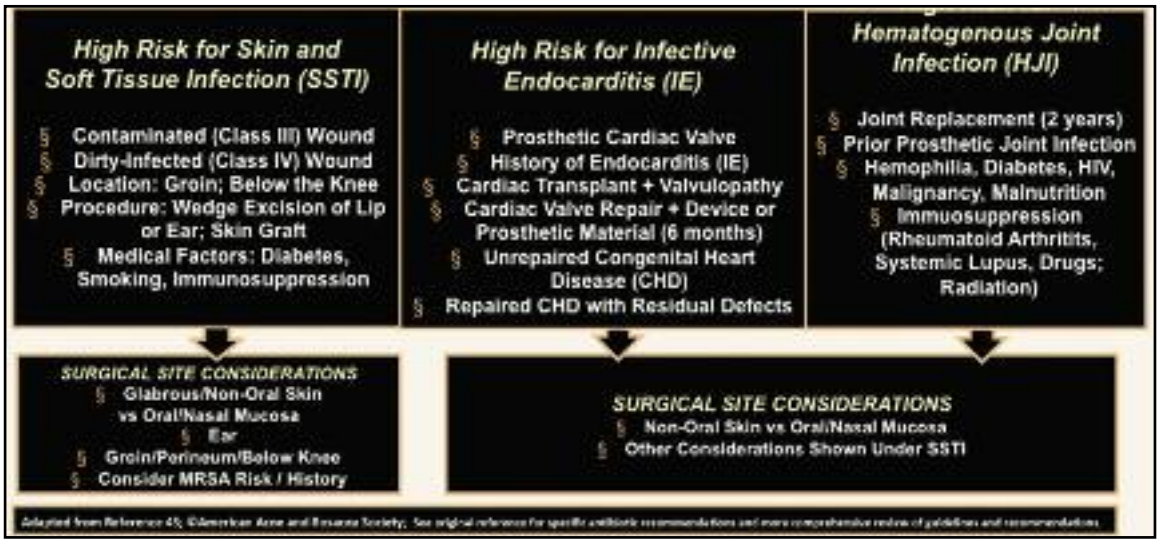

Figure 4. Systemic antibiotic surgical prophylaxis: Assessment of risk status and management

Other skin disorders. Other dermatologic conditions where oral antibiotic use is often not needed are inflamed epidermal cysts and chronic venous leg ulcers; available data support that systemic antibiotic therapy does not accelerate healing of noninfected venous ulcers, but does promote colonization with drug-resistant bacteria.[37,49]

WHAT MEASURES CAN DERMATOLOGISTS INCORPORATE TO IMPROVE RATIONAL USE OF ANTIBIOTIC THERAPY AND LIMIT POTENTIAL FOR ANTIBIOTIC RESISTANCE?

In this three-part article series, a large body of information was covered on antibiotic exposure, prescribing characteristics, patterns of antibiotic resistance, and suggested management principles. Within all three articles, suggestions were given which clinicians can incorporate in order to use antibiotics more rationally and reduce antibiotic resistance.

The following are some important questions to ask oneself and principles to consider when prescribing antibiotic therapy.

- In any given case, is antibiotic therapy needed? Why is it needed? What is the regimen to be used including dosing and duration of use?[49]
- If infection is considered, has culture and susceptibility testing been obtained (when applicable) prior to initiation of antibiotic therapy?[11]
- When treating acne vulgaris, utilize topical antibiotic therapy when clinically indicated and avoid its use as monotherapy. Concomitant use with benzoyl peroxide is recommended to reduce the emergence of antibiotic-resistant *Propionibacterium acnes*.[49–51]
- Use oral antibiotic therapy for acne vulgaris only when felt to be definitively needed and in combination with a topical regimen that preferably contains benzoyl peroxide and a topical retinoid.[49–51] It is important that an exit strategy for discontinuation of oral antibiotic therapy be planned up front and also discussed with the patient (i.e., after 3–4 months).[49–51]
- When treating acne vulgaris, consider non-antibiotic options when selecting initial treatment or adjusting therapy. This may include a variety of topical agents and/or physical modalities.[49–52]
- Antibiotic therapy may be avoided in many cases of rosacea. Non-antibiotic topical therapies and subantimicrobial-dose doxycycline are frequently effective for treatment of papulopustular rosacea. Erythematotelangiectatic rosacea and persistent non-transient facial erythema present in patients with papulopustular rosacea may be treated with physical modalities and/or topical alpha-adrenergic agonist therapy (i.e., brimonidine, oxymetazoline).[49,53,54]
- In atopic dermatitis, limit antibiotic therapy to intermittent use for treatment of clinical infection and avoid use as chronic suppressive therapy.[4,5,37] The latter has not been shown to be effective and increases emergence of antibiotic-resistant bacterial strains.
- Routine use of antibiotic therapy is not suggested in cases of inflamed epidermal cysts and chronic venous leg ulcers.[37,49]
- Avoid routine use of perioperative oral antibiotic prophylaxis for dermatologic surgical procedures.[42–45] Use antibiotic prophylaxis only when indicated based on current guideline recommendations or when clinical judgment determines necessity for a specific patient.[42] Reasons to use perioperative oral antibiotic therapy included cases at high risk of postoperative surgical site infection and conditions recognized to favor development of infective endocarditis or hematogenous joint infection.[45]
- Avoid routine application of a topical antibiotic to postoperative wound sites after common dermatologic procedures (e.g., curettage, biopsy,

sutured incision line).[37,39–41] White petrolatum is sufficient to maintain a moist wound environment, reduces risk of contact dermatitis, and avoids antibiotic resistance.[37–40] Overall, the use of a topical antibiotic has not been shown to reduce the risk of postsurgical wound infection.[39–41]

ACKNOWLEDGMENT

The American Acne and Rosacea Society and SPAUD thank Guillermo Sanchez, PA-C, MPH, Public Health Scientist from the Centers for Disease Control in Atlanta, Georgia, for his presentation and participation at the SPAUD meeting in September 2014.

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