

August 1, 2017

Robert Cybulski, PhD

Marisa D'Angeli, MD

Rupali Jain, PharmD

John Lynch, MD

Natalia Martinez-Paz

Paul Pottinger, MD

Agenda

- Didactic: Skin and Soft Tissue Infections 2
- Case Discussion
- Open Discussion

URL: <http://rwpoll.com>

Code: uwecho

Skin and Soft Tissue Infections Part 2

John Lynch, MD, MPH
Associate Professor
Harborview Medical Center &
The University of Washington School of Medicine

August 1, 2017

URL: <http://rwpoll.com>
Code: uwecho

Types of SSTI

- Erysipelas
- Impetigo
- Cellulitis
- Necrotizing skin and soft tissue infection (and Fournier's gangrene)
- Furuncle and carbuncle (diff than folliculitis)
- Cutaneous abscess
- Pyomyositis
- Gas gangrene
- Wound infections....

SSTI: Most Common Organisms

- Aerobic Gram-positive bacteria
- *Staphylococcus aureus* (MSSA and MRSA)
 - Purulent SSTI like abscesses and wound infections
- *Streptococcus pyogenes* (aka Group A strep)
 - Non-purulent SSTIs like cellulitis
- *Vibrio vulnificis*, aeromonas, peptostreptococcus, clostridia species
- Polymicrobial infections

subcutis/hypodermis Dermis

Epidermis

Superficial
arteriovenous
plexus

Papillary dermis

Reticular dermis

Meissner's corpuscle

Sweat duct

Deep
arteriovenous
plexus

Subcutaneous fat

Dermal nerve fibres

Eccrine sweat gland

Pacinian corpuscle

Hair shaft

Opening of sweat duct

Dermal
papillae

Arrector pil
muscle

Sebaceous
gland

Hair follicle

Eccrine sweat duct

Eccrine sweat gland



subcutis/hypodermis Dermis

Epidermis



Hair shaft

Opening of sweat duct

Dermal papillae

Meissner's corpuscle

Sweat duct

Deep arteriovenous plexus

Subcutaneous fat

Arrector pil muscle

Sebaceous gland

Dermal nerve fibres

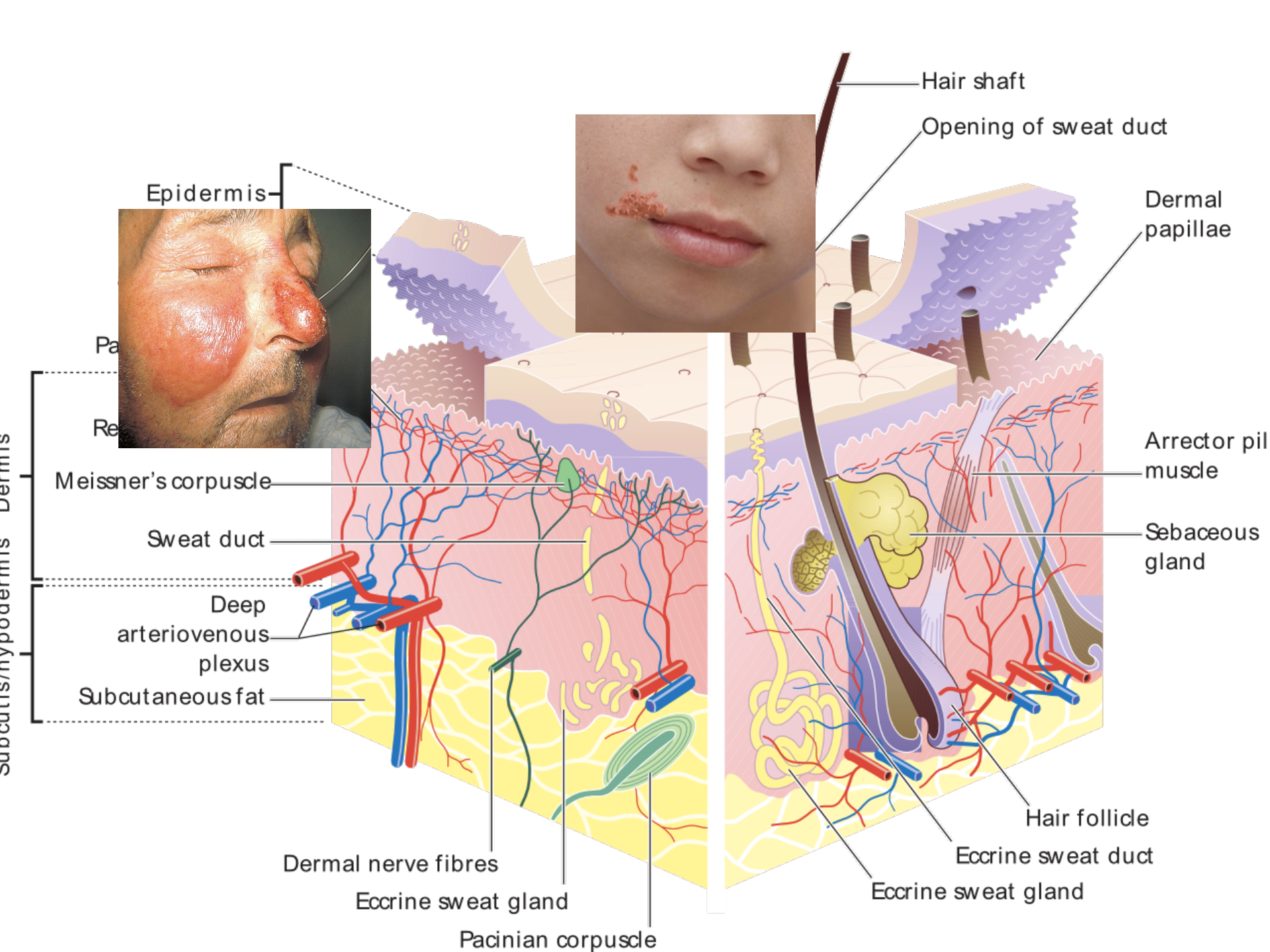
Eccrine sweat gland

Pacinian corpuscle

Hair follicle

Eccrine sweat duct

Eccrine sweat gland



subcutis
hypodermis
Dermis

Epidermis

Hair shaft

Opening of sweat duct

Dermal papillae

Arrector pil muscle

Sebaceous gland

Hair follicle

Coiled sweat duct

Sebaceous gland

Meissner's corpuscle

Sweat duct

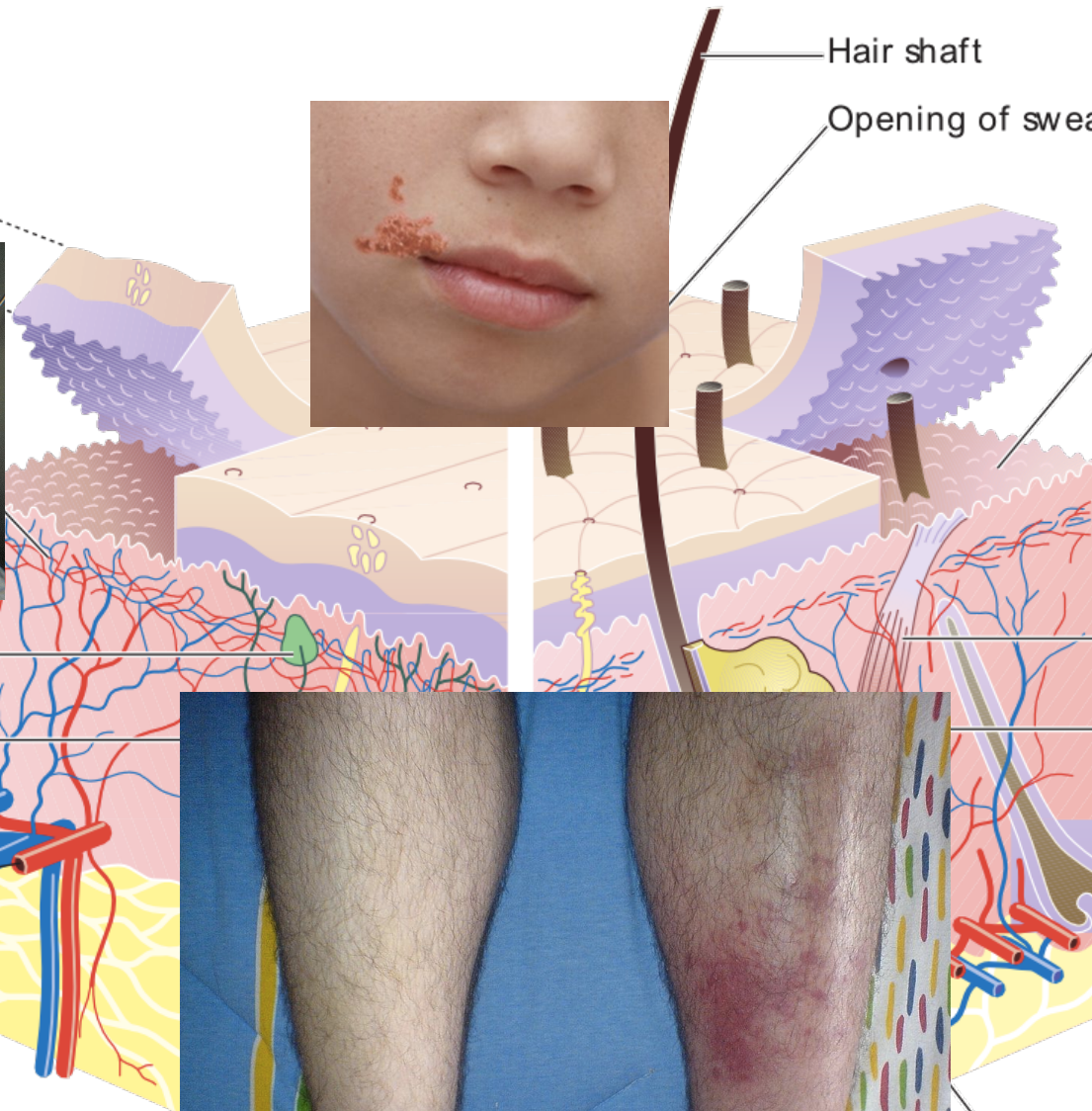
Deep arteriovenous plexus

Subcutaneous fat

Dermal nerve fibre

Eccrine sweat gland

Pacinian corpuscle



10 Cellulitis Myths

- Myth 1: Skin that is red and swollen is definitely cellulitis.
- Lesson 1: Local presentation of edema, erythema, warmth, hyperemia, tenderness, “orange peel” appearance, vesicles, bullae, petechiae, and pain may lead to a diagnosis of ABSSSI.
- Diagnoses of deep venous thrombosis (DVT), venous stasis dermatitis, venous insufficiency, lymphedema, contact dermatitis, gout, herpes zoster, acute lipodermatosclerosis, noninfectious phlebitis, insect bite hypersensitivity, Sweet's syndrome, and fixed drug reaction should also be considered
- Straight leg raise test

10 Cellulitis Myths

- Myth 2: My patient has bilateral lower-extremity swelling and redness; my patient has bilateral cellulitis.
- Lesson 2: Bilateral lower-extremity cellulitis is exceedingly rare. If bilateral swelling is present, noninfectious etiologies should be considered first, including but not limited to chronic stasis dermatitis, DVT, heart failure, venous stasis, and lymphedema.

10 Cellulitis Myths

- Myth 4: With the increased prevalence of methicillin-resistant *Staphylococcus aureus*(MRSA) in the community, all clinically stable, community-dwelling patients presenting to the ED with cellulitis should be treated with an antibiotic that has activity against MRSA.
- Lesson 4: The antibiotic spectrum decision should be based on several factors, including presence or absence of purulence, severity of illness, patient-specific risk factors for MRSA, and local bacteria ecology.

10 Cellulitis Myths

- Lesson 4: The antibiotic spectrum decision should be based on several factors, including presence or absence of purulence, severity of illness, patient-specific risk factors for MRSA, and local bacteria ecology.
- Impetigo, erysipelas, and cellulitis that is diffuse, erythematous, nonpurulent with extensive lymphangitic spread, or unassociated with a defined portal is more commonly caused by Group A or other β -hemolytic streptococci rather than *Staphylococcus* spp., however, *S. aureus* may also be present. In nonpurulent cellulitis, the addition of MRSA coverage (with trimethoprim-sulfamethoxazole [TMP-SMX]) to cephalexin does not improve patient outcomes, including clinical cure or progression to abscess

10 Cellulitis Myths

- Myth 5: My patient requires hospitalization for cellulitis, therefore, my patient has a MRSA infection and requires MRSA targeted anti-infective therapy.
- Lesson 5: Similar to lesson 4, the presence or absence of purulence, severity of illness, patient-specific risk factors for MRSA, and local bacteria ecology should guide the provider in determining the causative pathogen of an ABSSSI, irrespective of the location of the patient.

10 Cellulitis Myths

- Lesson 5: Similar to lesson 4, the presence or absence of purulence, severity of illness, patient-specific risk factors for MRSA, and local bacteria ecology should guide the provider in determining the causative pathogen of an ABSSSI, irrespective of the location of the patient.
- In patients who are sick enough to be hospitalized, it is reasonable to begin an antibiotic with activity against MRSA, such as vancomycin, and culture the lesion. If the lesion is not culturable, it is reasonable to obtain MRSA swabs of the nares and pooled axilla/groin to guide definitive antibiotic therapy.

10 Cellulitis Myths

- Myth 7: Because one cannot tell whether cellulitis is caused by *Streptococcus* spp., MSSA, MRSA, Gram-negative or anaerobic pathogens, each patient needs to be treated with broad-spectrum antibiotic therapy.
- Lesson 7: Antibiotic therapy should be selected based on the characteristics of the infection, severity of illness, and patient-specific risk factors for different organisms. Most cases of uncomplicated cellulitis without abscess or purulence will not need combination therapy with a β -lactam and anti-MRSA antibiotic. Gram-negative and anaerobic coverage is generally unnecessary.
- NOTE: TMP-SMX?

10 Cellulitis Myths

- Myth 8: If the redness extends beyond the drawn wound margin in a patient with cellulitis, the patient is getting worse.
- Lesson 8: Because of the subacute spread of redness, edema, or induration in some patients at the time of presentation with cellulitis, the lesion may continue to spread for the first 48 h after administration of antibacterial drug therapy.

10 Cellulitis Myths

- Lesson 8 continued...
- Erythema may extend beyond documented margins during the first 48 h without necessarily representing treatment failure. The intensity of the erythema is often a more important variable, with improving cases resulting in less intensely red inflammation.
- If erythema and fever continue beyond 48 to 72 h, treatment failure should be considered and antibiotic therapy should be reassessed.
- The Infectious Diseases Society of America recommends 5 days of treatment for erysipelas and cellulitis, with the option to extend treatment duration in the absence of clinical improvement within this time period.

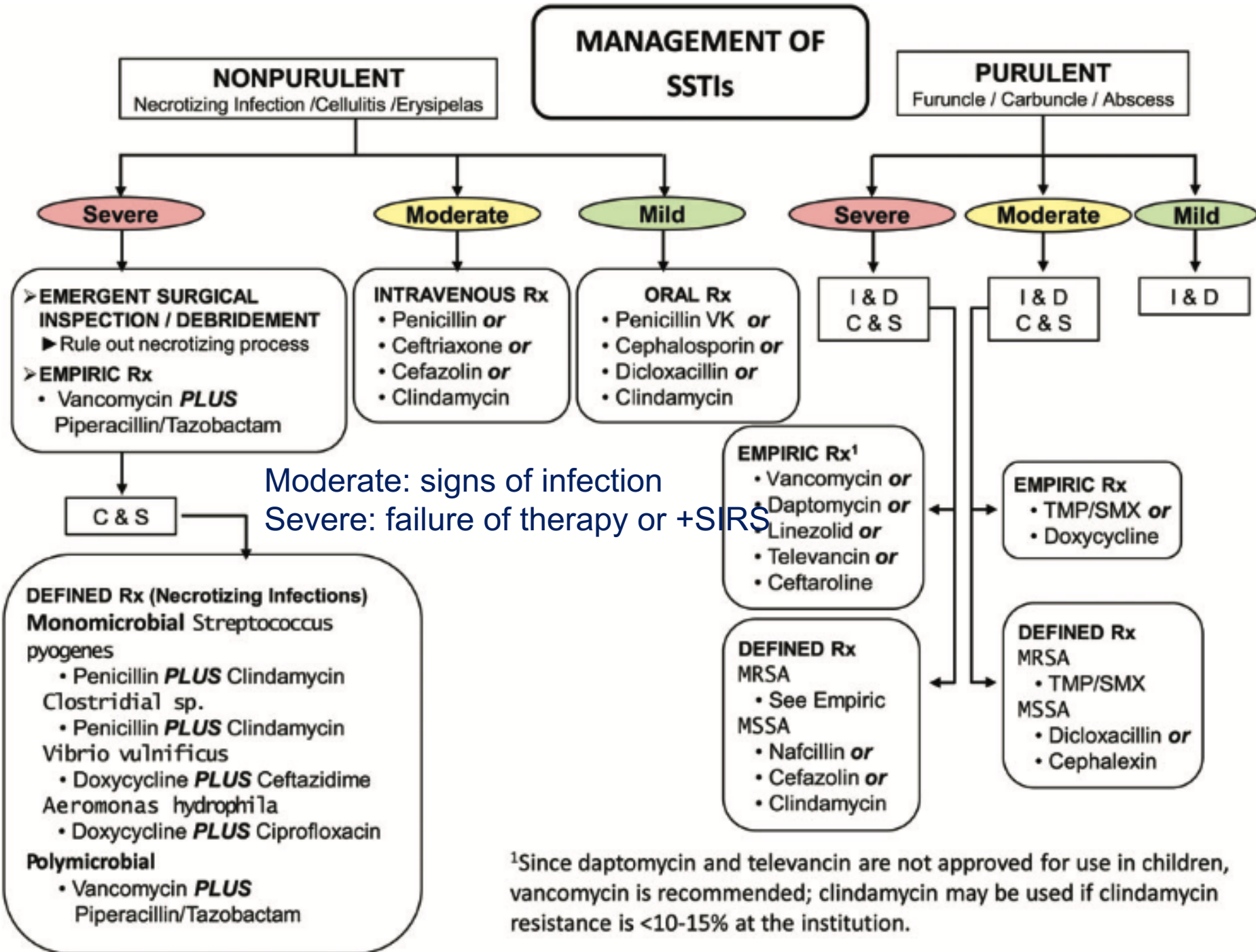
10 Cellulitis Myths

- Myth 10: All patients with tick bites and surrounding redness have cellulitis.
- Lesson 10: Local tick bite reactions are predictable and do not indicate that a patient has cellulitis. These reactions are usually no more than a few centimeters in size.

Practice Guidelines for the Diagnosis and Management of Skin and Soft Tissue Infections: 2014 Update by the Infectious Diseases Society of America

Dennis L. Stevens,¹ Alan L. Bisno,² Henry F. Chambers,³ E. Patchen Dellinger,⁴ Ellie J. C. Goldstein,⁵ Sherwood L. Gorbach,⁶ Jan V. Hirschmann,⁷ Sheldon L. Kaplan,⁸ Jose G. Montoya,⁹ and James C. Wade¹⁰

¹Division of Infectious Diseases, Department of Veterans Affairs, Boise, Idaho; ²Medical Service, Miami Veterans Affairs Health Care System, Florida; ³San Francisco General Hospital, University of California; ⁴Division of General Surgery, University of Washington, Seattle; ⁵University of California, Los Angeles, School of Medicine, and R. M. Alden Research Laboratory, Santa Monica, California; ⁶Department of Community Health, Tufts University, Boston, Massachusetts; ⁷Medical Service, Puget Sound Veterans Affairs Medical Center, Seattle, Washington; ⁸Department of Pediatrics, Baylor College of Medicine, Houston, Texas; ⁹Department of Medicine, Stanford University, California; and ¹⁰Geisinger Health System, Geisinger Cancer Institute, Danville, Pennsylvania



Moderate: signs of infection
Severe: failure of therapy or +SIRS

¹Since daptomycin and televancin are not approved for use in children, vancomycin is recommended; clindamycin may be used if clindamycin resistance is <10-15% at the institution.

References

McCreary, E. K. *et al.* Top 10 Myths Regarding the Diagnosis and Treatment of Cellulitis. *J. Emerg. Med.* (2017). doi:10.1016/j.jemermed.2017.05.007