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REVIEW ARTICLE OPEN ACCESS

Rising Drug Resistance in Skin and Soft Tissue Infecting Pathogens: An Integrative Analysis

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Abstract

Bacterial skin and soft-tissue infections (SSTIs) are a major health concern worldwide because of their high prevalence, frequent recurrence, and increasing antibiotic resistance. Recent research indicates that SSTI incidence can reach up to 77.5 cases per-year in high-income settings, while prevalence rates may be as high as 66% among hospitalised patients in resource-limited regions. Recurrence rates range from 7%-45% depending on comorbidities, treatment adequacy, and host variables. Methicillin-resistant *Staphylococcus aureus* (MRSA) and other resistant bacteria reduce the efficacy of traditional treatments. Novel antibiotics such as dalbavancin, tedizolid, oritavancin, and delafloxacin are effective against resistant SSTIs, but their use varies regionally. This review summarises recent research on SSTI epidemiology, recurrence, and resistance patterns, emphasising diagnostic gaps, treatment problems, and potential therapeutic methods. Enhanced surveillance, uniform reporting, and newer antimicrobials are critical for reducing the SSTI burden and directing future research.

Keywords: Pyogenic Infections, Soft Tissue Infections, Antibiotic Resistance, Bacterial Infection

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INTRODUCTION

The global increase in bacterial infections resistant to numerous antibiotics threatens to return contemporary medicine to the pre-antibiotic era. Although new antibiotics are still being produced, the cost of developing them is high, and bacteria will eventually develop resistance to them. Breaking the cycle of bacterial infection requires non-antibiotic techniques.[1] Recent reviews of experimental antibiotics indicate that medication development is substantially slower than the emergence and spread of resistant bacteria. Only two new antibiotic classes, dalbavancin and oritavancin, have entered the market over the past 30 years. This standstill has prompted research into new chemicals and anti-infective treatments.

Antimicrobial peptides, antivirulence drugs, bacteriophages, and antibodies show promise as replacements or adjuncts to standard antibiotics. Although the pipeline for phase II and III antibacterial drugs is still restricted, progress is possible through the combination of computational techniques with chemical and biological innovation.[2,3] Antibiotics have saved lives since their introduction in the 1940s, but rising resistance has swung the advantage back to bacteria. With high rates of emergent resistance and limited progress in antibiotic discovery, the demand for therapeutic methods other than conventional chemotherapy is increasing.

Human skin infections

The skin is the body's primary defence against microbial invasion, acting as a physical barrier and releasing low-pH secretions and fatty acids that prevent pathogen growth. Its indigenous microbiota further limits the colonisation of dangerous pathogens.[4,5] The skin not only protects against diseases but also controls temperature, prevents fluid loss, and aids in sensory awareness. Pathogens that break this barrier cause tissue injury and an inflammatory response.[6,7]

Types of human skin infections

Primary infections

Primary skin infections appear in normally healthy skin, are caused by a single pathogen, and exhibit a specific clinical pattern (Figure 1). Examples include impetigo, folliculitis, and boils.[8]

Secondary infections

Secondary infections develop in diseased skin. Their clinical course varies depending on underlying diseases (Figure 1). Examples include intertrigo and toe-web infections.[9]

Bacterial skin infections

Bacterial skin infections are among the most common illnesses in hospitals and are sometimes referred to as skin and soft tissue infections (SSTIs) or skin and soft structure infections (SSSIs). Erythema, warmth, induration, and discomfort are examples of local or systemic inflammatory reactions caused by microbial invasion of the epidermal and subcutaneous tissue.[10] SSTIs can range from severe, potentially fatal necrotising fasciitis to minor, self-limiting illnesses.[11,12] According to Mngqibisa et al.,[13] SSTIs are classified as deep (affecting the subcutaneous tissue, fascia, and muscle) or superficial (affecting the epidermis and dermis). Deeper infections are typically polymicrobial, whereas superficial infections often include a single pathogen. Cellulitis penetrates the dermis, whereas conditions such as folliculitis, erysipelas, and impetigo affect the epidermis. Fasciitis or myonecrosis may develop when the infection spreads to deeper tissues.[14,15]

Fungal skin infections

Dermatophytosis and cutaneous candidiasis are examples of prevalent superficial fungal diseases. Dermatophytic infections on different body sites lead to tinea capitis, tinea corporis, tinea cruris, tinea manuum, and tinea pedis. Other dermatophytes (tinea unguium) and fungal species (onychomycosis) cause fungal nail infections.[16] Another common fungal illness is candidiasis, which is primarily caused by *Candida albicans*.[17]

Viral skin infections

Childhood chickenpox is caused by primary infection with the varicella-zoster virus (VZV), and the reactivation of latent VZV causes herpes zoster. Herpes simplex virus (HSV)-2 mostly causes genital herpes, whereas HSV-1 causes cold

sores, stomatitis, and ocular infections. Human papillomavirus (HPV) often cause warts in children, teenagers, and older adults.[18,19] A poxvirus illness called molluscum contagiosum is prevalent throughout the world, particularly in tropical regions and in children.[20]

Parasitic skin infections

Parasitic skin infections continue to contribute substantially to the global dermatologic disease burden especially in tropical, subtropical and resource†limited regions. Scabies, caused by the mite *Sarcoptes scabiei,* remains one of the most common and burdensome skin diseases worldwide. Current estimates indicate that hundreds of millions of people are affected each year globally. Treatment challenges and emerging resistance remain a concern. For instance, a 2025 report documented a case of CLM refractory to conventional therapy with Ivermectin.[21]

Factors influencing human skin infections

The prevalence of pyoderma is greatly influenced by various socioeconomic and environmental factors, including poverty, poor hygiene, overcrowding, malnutrition, and

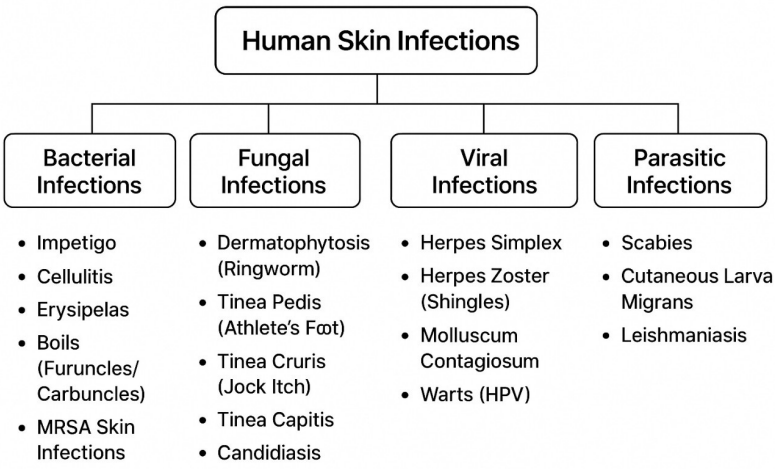

Figure 1. Types of skin infections caused by different microorganisms

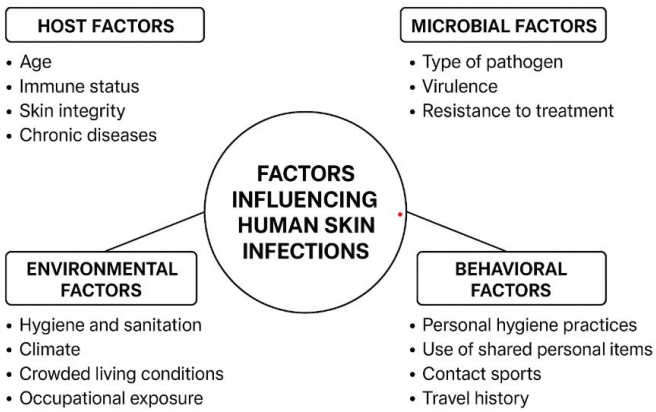

Figure 2. Factors influencing human skin infections

Table. Epidemiology and recurrence of skin and soft tissue infections (SSTIs)

Measure/ Outcome	Estimate	Sample/Numerator & CI	Study Context	Ref.
Incidence of SSTI episodes (USA)	77.5 per 1,000 person-years	9.1 million SSTI episodes in 5.4 million patients; 95% CI 77.4-77.5 per 1,000 PYO. (OUP Academic)	Retrospective claims-data cohort (Optum Clinformatics), 2010-2020, USA. (PMC)	37
Proportion of recurrent SSTI (USA cohort)	26.3% of index cases	Among ~2.98 million index SSTI cases, 783,963 had a recurrence (26.3%). (OUP Academic)	Same Optum cohort (2010-2020). (PubMed)	37
Prevalence of SSTI among suspected hospitalized cases (Uganda)	66.4%	268 randomly selected reports; prevalence 66.4% (95% CI ~60.7-72.1). (PubMed)	Retrospective study at Jinja Regional Referral Hospital, Uganda (2019-2021). (makir.mak.ac.ug)	38
Recurrent skin and soft tissue infection (RSSTI) rate	7%-45%	Reported recurrence range in recent literature; risk depends on comorbidities/ host factors. (Lippincott Journals)	Review of risk factors & management for recurrent SSTIs. (Lippincott Journals)	39
Recurrence risk (by risk-score stratification)	High-risk group: ~16.6% recurrence	Based on BRRISC score stratification: high-risk (score 6–15) had recurrence of 16.6% (95% CI 13.3-20.4%). (PMC)	Model/risk-stratification in cellulitis recurrence - part of the recurrence review. (PMC)	39

climate.[22,23] Climate, genetics, age, sex, stress, diet, and hospitalisation affect the microbial makeup of skin infections.[24] Because of physiological skin changes and lowered immunity, older adults are especially vulnerable. Approximately 70% of adults over 70 have at least one skin condition, and skin and soft tissue infections cause greater morbidity and death in this age range.[25]

Age-related skin thinning, decreased sebaceous gland activity, and dehydration increase infection susceptibility and hinder recovery (Figure 2). Skin fragility worsens with prolonged UV exposure.[26,27] *Proteus mirabilis* and *Pseudomonas aeruginosa* colonise older adults more frequently, partially because of their weakened immune systems.[28] The risk of recurring or severe SSTIs is further increased by comorbid conditions such as diabetes, renal disease, obesity, HIV infection, and immunosuppressive medication.[29] The likelihood of infection also increases due to lymphedema and surgical lymph node excision.[30]

Occurrence of human skin infections

In India, bacterial skin infections are common and are a major reason for dermatology consultations. Because of widespread and improper antibiotic usage, illnesses that were previously treatable are becoming less responsive to widely used antibiotics (Table). Of the 178 lab-confirmed cases in an analysis of clinically suspected SSTIs, 66.4% of samples tested positive for SSTI pathogens.[31]

According to a major health system dataset, the annual incidence of SSTIs is approximately 77.5 per 1,000 people. A 2024 epidemiological investigation identified 77.5 SSTI episodes per 1,000 people during the study period (5.4 million patients with 9.1 million SSTI episodes). This estimate reflects healthcare utilisation based on a large dataset.

An estimated 14 million instances of cellulitis, a prevalent bacterial SSTI, occur in the United States each year.[32] SSTIs are among the

most prevalent hospital/healthcare-associated infections (HCAIs). A national systematic review and meta-analysis in Nigeria reported an overall HCAI prevalence of approximately 15.8% (pooled data). The most common HCAIs in the included studies were surgical site infections and SSTIs.[33]

MRSA prevalence among SSTI isolates varies greatly by region. In Asia, documented regional prevalence ranged from 7.3% to 74% depending on country, environment, and year, highlighting regional variations in the burden of resistance.[34]

According to recent evaluations of recurrence risk, cellulitis has a recurrence rate of approximately 14% within one year and 45% within three years, which greatly increases the burden on the healthcare system.[35]

According to several recent regional studies (2023-2024), children living in high-burden environments show a high prevalence of impetigo, with prevalence estimates varying by context, season, and monitoring technique.[36]

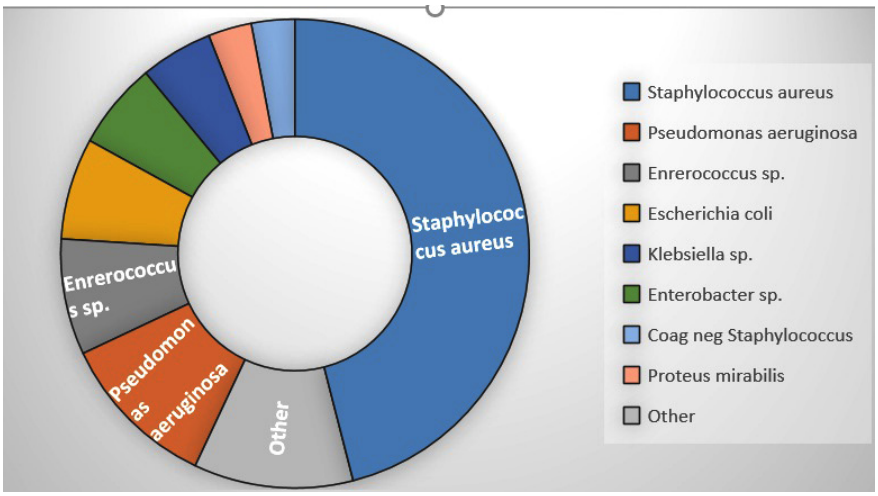

Figure 3. Microbial diversity showing the incidence of bacterial infection

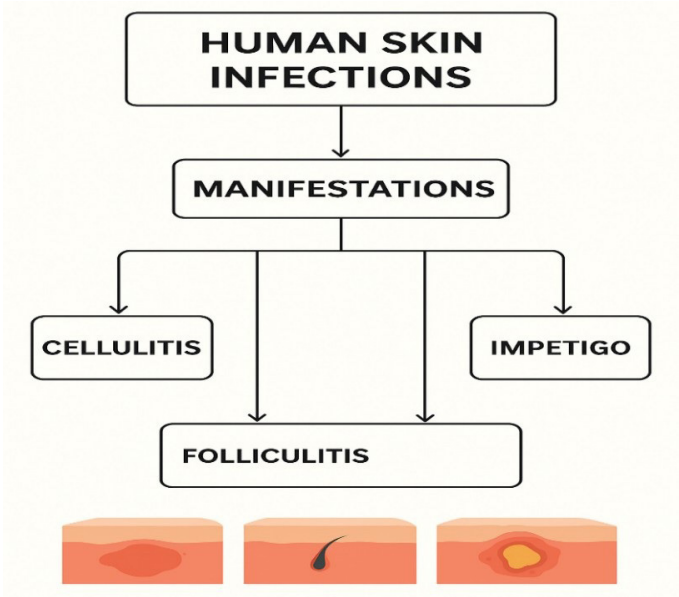

Figure 4. Manifestations of human skin infections

Causative agents of human skin infections

Bacteria, fungi, viruses, and parasites cause skin infections. Most pyogenic skin infections are caused by bacterial pathogens, including *Staphylococcus aureus* and *Streptococcus pyogenes*.

Agents of bacterial causation

Because the skin is constantly exposed to the environment, it hosts a variety of microbiota, primarily bacteria (Figure 3). Gram-positive bacteria such as *S. aureus, S. pyogenes, S. epidermidis,* and *Corynebacterium* spp. are more common above the waist.[40] Both Gram-positive and Gram-negative bacteria, including *Enterobacteriaceae* species, are more prevalent below the waist.[41] The following figure illustrates the overall occurrence of bacterial causal agents of human skin diseases, as suggested by Maraki et al.[42]

Gram-positive organisms are the most prevalent pathogens in complicated skin and skin structure infections (CSSSIs) and uncomplicated skin and skin structure infections (USSSIs). Figure 4 illustrates the most common Gram-positive and Gram-negative microorganisms that cause skin diseases. *S. aureus* was the most common (46%), followed by *P. aeruginosa* (11%), *Enterococcus* sp. (8%), *E. coli* (7%), *Enterobacter* sp. (6%), *Klebsiella* sp. (5%), *P. mirabilis* (3%), coagulase-negative

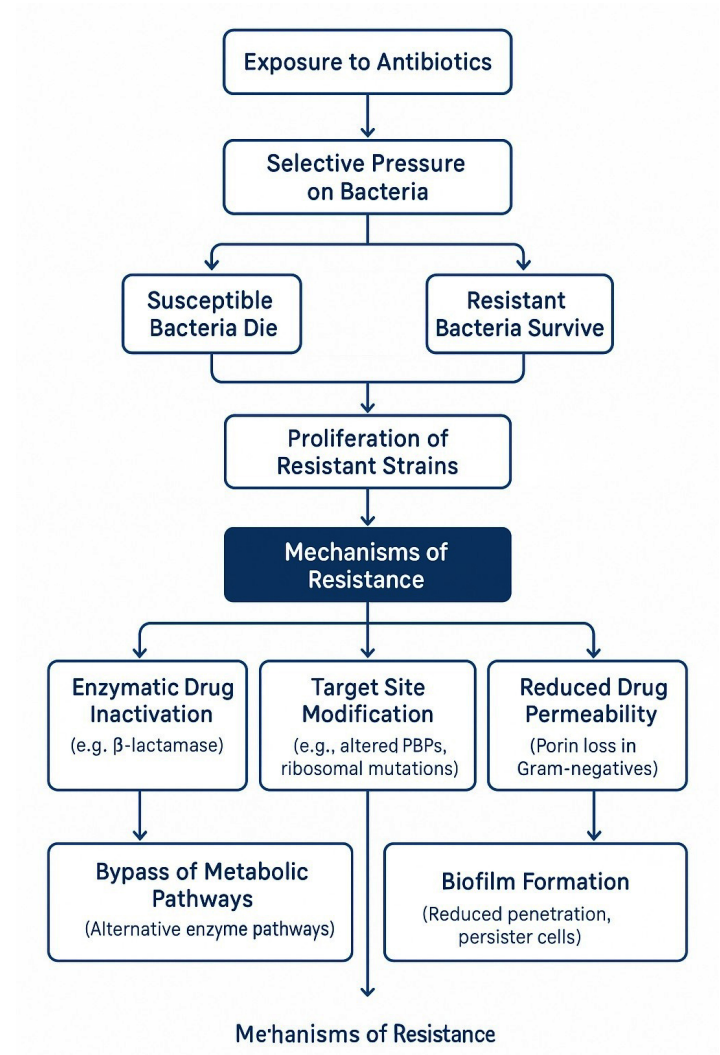

Figure 5. Mechanism of antibiotic resistance pattern in bacteria

Staphylococcus sp. (3%), and miscellaneous species (11%). *S. aureus* and *S. pyogenes* are the usual causes of USSSIs, and historically, empirical treatment has primarily targeted these Gram-positive bacteria.[43] However, anaerobic and Gram-negative bacteria are becoming more common.[44]

SSTIs are characterised by different microbiology depending on how they enter the skin.[45,46] According to a 2024 study conducted in northern India, 48.36% of pus samples from SSTIs were culture-positive during the study period, whereas 36.33% of the samples were culture-negative.[47] Among the positive clinical isolates, 54.04% were Gram-negative bacilli and 45.96% were Gram-positive cocci. The top six microbes accounted for approximately 90% of all infections: *S. aureus* (38.05%), *E. coli* (17.39%), *Pseudomonas* sp. (11.82%), *Acinetobacter* sp. (10.16%), *Klebsiella* sp. (6.72%), and Coagulase-negative *Staphylococcus* species (5.50%). Other isolates included Proteus species (2.26%), *Enterococcus* species (2.19%), β-haemolytic streptococci (Group A) (0.18%), and *S. pneumoniae* (0.03%). *S. aureus* was a frequent pathogen in over 70% of all SSTIs and in 50% of community cellulitis cases. MRSA was present in 10.9% of patients with bacterial skin infections in South India.[48] In a hospital-based prospective investigation conducted among children in north India in 2024, the incidence of community-acquired (CA) MRSA was 6.9%.[49]

Methicillin-resistant *S. aureus* (MRSA)

MRSA initially appeared in hospitals in the 1960s before spreading to the general public. Prevalence differs greatly by country (1%-74%).[50] CA-MRSA is a leading cause of SSTIs worldwide, especially in children.[51] *S. aureus* was responsible for 76% of SSTIs in a major emergency department research, with MRSA accounting for 59% of these infections.[52] The Panton-Valentine leukocidin (PVL) toxin, which causes tissue necrosis and leukocyte death, is commonly present in CA-MRSA strains.[53]

Manifestations of human skin infections

The ability of an organism to infiltrate tissues, proliferate, and elude host defences is known as pathogenicity. Adhesins, enzymes, and toxins are among the many variables that affect microbial virulence, which differs between species.[54]

The epidermis, dermis, subcutaneous and adipose tissues, muscular fascia, and other layers of the skin architecture are colonised by bacteria, initially in small quantities. When the integumentary barrier is compromised, bacteria proliferate, leading to invasion and

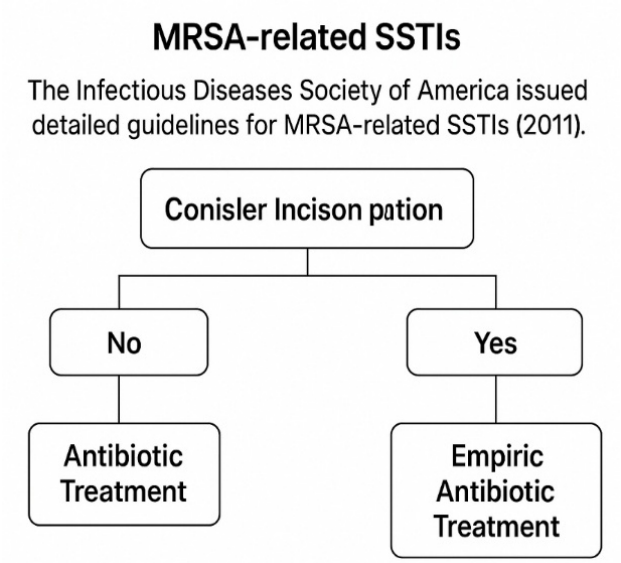

Figure 6. A guideline for the usage of antibiotics for MRSA-related SSTIs

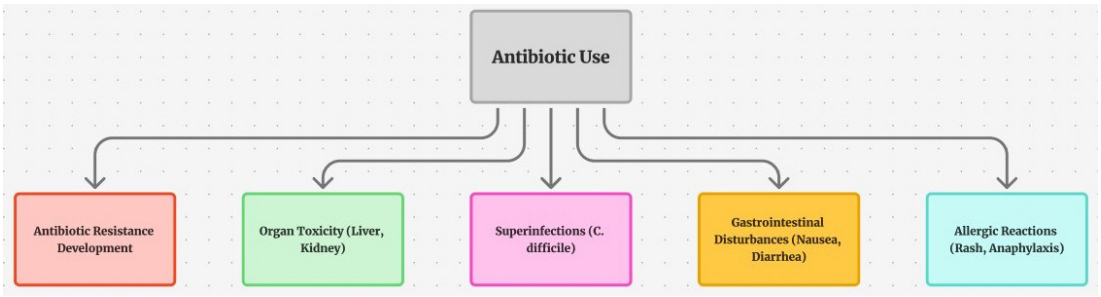

Figure 7. Adverse effect of antibiotic usage

the development of SSTIs. (Figure 4). Finally, involvement of deeper skin structures may lead to fasciitis and myositis.[55] These pathways allow native flora and normal skin flora introduced by the penetrating tool to enter.[56-59]

Bacterial infection

Three processes are necessary for the establishment of an SSTI: bacterial adhesion to host cells, tissue invasion with host defence evasion, and toxin generation. Most pathogenic bacteria have virulence genes that encode proteins providing these characteristics. Toxins are the strongest contributors to clinical illness among the bacterial virulence factors.[60-62] Endotoxins and exotoxins are the two primary categories of toxins. Gram-negative bacterial cell walls are rich in lipopolysaccharide chains known as endotoxins. Lipopolysaccharides may be advantageous in small amounts because they stimulate the immune system.

By stimulating the production of costimulatory molecules, they increase T-lymphocyte activation and the release of chemoattractants. However, excessive lipopolysaccharide elaboration may cause harmful overstimulation of the host's inflammatory and immunological systems.[63] Conversely, exotoxins are actively released proteins that cause tissue injury or dysfunction via several pathways.

Inflammation

The host's reaction to tissue invasion and damage is the other part of the infection process. The body uses inflammation as a defensive response to begin tissue repair and eliminate the causative organisms. Microbial invasion and damage to the skin or soft tissues alter vascular tone, increasing blood flow to the affected area. Further alterations in the microvasculature facilitate the extravasation of leukocytes and plasma proteins. At the damaged site, these cells and proteins move, gather, and become active. When cells are activated, they phagocytose and eliminate foreign objects, dead tissue, and microorganisms. The fever response is caused by certain exotoxins or pyrogenic cytokines. Ultimately, the injured area is cleansed, quarantined, and restored.[64]

Depth of infection

The potential depths of SSTI involvement and the corresponding diagnoses are shown in the following figure. Cellulitis extends into the dermis, whereas superficial infections such as erysipelas, impetigo, folliculitis, furuncles, and carbuncles are found in the epidermis. Deeper infections progress to fasciitis or myonecrosis after passing through the subcutaneous tissue.[65] Determining the level of infection through examination can be challenging, but laboratory testing can aid this evaluation.[66]

Coagulase, staphylokinase, lipase, and β-lactamase are among the virulence factors found in 90% of *S. aureus* strains. Factors that prevent phagocytosis include the polysaccharide slime layer and protein A on the cell surface. Certain strains produce cytolytic toxins, leukocidins, exfoliative toxins, epidermal cell differentiation inhibitors, and toxic shock syndrome toxins.[67] Panton-Valentine leukocidin (PVL) is frequently linked to enhanced virulence in CA-MRSA.[68] By producing the powerful chemotactic proteins interleukin-8 and leukotriene B4, this cytotoxin destroys leukocytes and promotes tissue necrosis.[69,70]

Additionally, *S. aureus* secretes enterotoxins, which are superantigens that activate T cells non-specifically and circumvent normal immune system pathways, causing a large release of cytokines. Research has shown that enterotoxins are produced by up to 50% of *S. aureus* isolates recovered from SSTIs.[71,72]

Antibiotic Treatment and Development of Drug Resistance

Antibiotic therapy

Antimicrobial medications can be either broad-spectrum or narrow-spectrum and act through various mechanisms: inhibition of cell wall synthesis (e.g., β-lactams, vancomycin), cell membrane disruption (e.g., polymyxins), inhibition of protein synthesis (e.g., tetracyclines, macrolides, aminoglycosides), inhibition of nucleic acid production (e.g., rifampin, quinolones), and suppression of metabolic pathways (e.g., trimethoprim, sulfonamides).[73,74]

Antibiotic policy

β-Lactams are first-line therapy for streptococcal and methicillin-sensitive *S. aureus* infections, with benzylpenicillin preferred for susceptible patients. Clindamycin is used for rapidly progressing infections, while newer fluoroquinolones (levofloxacin, moxifloxacin) and other systemic agents including cephalosporins, carbapenems, macrolides, linezolid, glycopeptides, glycylcyclines, and topical drugs like retapamulin broaden treatment options. Susceptibility patterns of skin isolates have been extensively studied.[75]

Development of drug resistance

Drug resistance occurs when initially susceptible microbes stop responding to antibiotics (Figure 5). Resistance may be non-genetic, for example, a temporary change into L-forms. R-plasmid acquisition and chromosomal mutations are examples of genetic resistance. To withstand antibiotics, microorganisms employ five primary strategies: modified enzymes, drug-inactivating enzymes, reduced membrane permeability, changed drug targets, and avoidance of metabolic processes.[76] Shortly after penicillin became widely used, *S. aureus* strains that produced penicillinase appeared.[77] MRSA emerged by 1961, despite the introduction of methicillin to combat β-lactamase.[78] The *mecA* gene, which codes for PBP2a and decreases β-lactam binding, is the primary resistance mechanism of MRSA.[79]

Current resistance trends

Studies conducted in India have revealed high rates of resistance to co-trimoxazole, erythromycin, and penicillins.[80-83] *E. coli* and *Klebsiella* frequently produce extended-spectrum β-lactamase (ESBL).[84-86] Common pathogens are becoming more resistant to aminoglycosides, macrolides, and fluoroquinolones. Treatment is becoming more difficult in many areas because MRSA strains exhibit multidrug-resistance and possess the PVL toxin.

Current treatment guidelines

The Infectious Diseases Society of America published comprehensive guidelines for treating MRSA-related SSTIs.[87-89] (Figure 6). These guidelines recommend using topical mupirocin (2%) for mild infections and incision and drainage alone for simple abscesses. Clindamycin, trimethoprim-sulfamethoxazole, doxycycline/minocycline, and linezolid are recommended as empirical treatments for purulent cellulitis caused by community-acquired MRSA. If non-purulent cellulitis does not improve after 48-72 hours, it is recommended to start with β-lactams and add MRSA coverage. Options for severe SSTIs that require hospitalisation include ceftaroline (active against MRSA and Gram-negative bacteria), vancomycin, linezolid, daptomycin, tigecycline, and telavancin. Although vancomycin has long been the main treatment for MRSA, other drugs such as linezolid, daptomycin, and glycopeptide derivatives have been developed because of delayed bacterial clearance and toxicity concerns.[90-93]

Adverse Effects of Antibiotic Treatment

Several adverse events are linked to antibiotic therapy (Figure 7). Clindamycin is linked to diarrhoea (particularly colitis caused by *C. difficile*); trimethoprim-sulfamethoxazole is associated with hyperkalaemia risk, particularly in older adults; long-term linezolid use can cause hematologic toxicity, neuropathy, and lactic acidosis; daptomycin is linked to GI problems,

renal impairment, and muscle soreness; telavancin causes GI symptoms, taste abnormalities, and nephrotoxicity; ceftaroline is associated with electrolyte imbalance and increased liver enzymes; and fluoroquinolones are linked to GI distress and glycaemic abnormalities. Certain MRSA strains are less susceptible to vancomycin, which increases morbidity and causes treatment failure.[94-100]

CONCLUSION

Skin and soft tissue infections (SSTIs) continue to pose a significant clinical and public health burden worldwide. The prevalence of SSTIs varies widely, ranging from 77.5 per 1,000 person-years in the USA to 66% among hospitalized patients in resource-limited settings, highlighting the need for region-specific surveillance and management strategies. Methicillin-resistant *Staphylococcus aureus* (MRSA), particularly community-associated strains, remains a major contributor to morbidity, with prevalence rates ranging from 1%-74% across different countries.

Future research should focus on combating antimicrobial resistance (AMR) by investigating alternative therapeutic strategies, including novel antimicrobials, phage therapy, antimicrobial peptides, and immunomodulatory approaches. Additionally, studies evaluating the efficacy of preventive measures, rapid diagnostics, and stewardship interventions are crucial. Maintaining updated epidemiological data with standardized reporting of prevalence and resistance patterns will be essential to guide evidence-based clinical management and reduce the global burden of SSTIs.

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DATA AVAILABILITY

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ETHICS STATEMENT

This article does not contain any studies on human participants or animals performed by any of the authors.

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