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Review

Bacteriological Profile of Superficial Wound Infections with Focus on MRSA and Its Response to Fusidic Acid and Rifampicin

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Abstract

Superficial wound infections are common clinical conditions caused by a variety of microorganisms, among which *Staphylococcus aureus* is one of the most prevalent pathogens. The emergence of Methicillin-Resistant *Staphylococcus aureus* (MRSA) has created significant challenges in the treatment of wound infections due to its resistance to multiple antibiotics. This review focuses on the bacteriological profile of superficial wound infections, with particular emphasis on MRSA and the therapeutic efficacy of fusidic acid and rifampicin. Studies indicate that MRSA is a major causative agent of skin and soft tissue infections worldwide, and combination antibiotic therapy is often required to prevent resistance development. Fusidic acid and rifampicin are widely used anti-staphylococcal agents with proven effectiveness, especially when used in combination. This review summarizes current knowledge on the prevalence, microbiology, antibiotic susceptibility patterns, and therapeutic outcomes associated with these drugs in MRSA-related superficial wound infections.

Keywords: MRSA, superficial wound infection, fusidic acid, rifampicin, antibiotic resistance, bacteriological profile.

1. Introduction

Superficial wound infections represent a significant public health problem affecting patients in both hospital and community settings. These infections may arise from trauma, surgical procedures, burns, or chronic ulcers. The microbial flora of such wounds typically includes *Staphylococcus aureus*, *Streptococcus pyogenes*, *Pseudomonas aeruginosa*, and other opportunistic pathogens. Among these microorganisms, Methicillin-Resistant *Staphylococcus aureus* (MRSA) has emerged as one of the most important pathogens responsible for skin and soft tissue infections. MRSA strains exhibit resistance to beta-lactam antibiotics and often demonstrate multidrug resistance, complicating treatment strategies.

MRSA infections have increased globally due to hospital-acquired and community-acquired transmission. The treatment of MRSA infections requires effective antibiotics, and fusidic acid and rifampicin are commonly used agents due to their strong activity against staphylococcal organisms. However, antibiotic resistance remains a growing concern, emphasizing the need to study susceptibility patterns and evaluate effective treatment options[1].

MRSA emerged soon after methicillin's introduction (1961). Historically HA-MRSA dominated hospitals, but since the 1990s CA-MRSA has surged as a cause of skin/soft tissue infection (SSTI). Global MRSA

rates vary widely: from <1% in some countries to >50% in others. In Europe, MRSA bloodstream isolates fell from ~19% (2009) to ~15% (2019), whereas in Asia–Pacific MRSA prevalence has been reported as 18–73% in various settings. Community MRSA clones often carry PVL, associated with necrotic SSTIs.

Local epidemiology of superficial wound MRSA is patchy. In outpatient dermatology in Greece, MRSA was isolated in 22.1% of *S. aureus*-positive dermatoses. In a Turkish wound registry, MRSA accounted for 20.9% of *S. aureus* wound isolates. In a Malaysian hospital survey (1996) ~40% of *S. aureus* were MRSA; most MRSA there remained susceptible to rifampin and fusidic acid (resistance ~3%). In healthy carriers in Nigeria, MRSA nasal carriage was 56.1%, with 56.3% of those isolates rifampin-resistant, highlighting geographic variation are mentioned in table 1. Overall, MRSA is common in hospital wounds (often >20%) and lower but rising in community wounds [2-3].

Table 1. Comparative data from selected studies on MRSA in wound/skin infections. MRSA% = proportion of *S. aureus* isolates that were MRSA; FA = fusidic acid; Rif = rifampin; S/R = percent susceptible/resistant[16-18]. (by disk or MIC). (NR = not reported.)

Author (Year)	Country	Setting / Sample	MRSA (% of S. aureus)	FA Susc. (Res%)	Rif Susc. (Res%)	Clinical Outcomes (notes)
Athanasakos *et al.* (2024)	Greece	Outpatient dermatology, n=68 SA	22.1% (15/68)	61.8% S (38.2% R)	100% S (0% R)	MRSA in 22% wounds; knee? (observational)
Kartal *et al.* (2023)	Turkey	Wound swabs, n=110 SA	20.9% (23/110)	92.7% S (7.3% R)	NR	Study of FA sensitivity; clinical not assessed
Maple *et al.* (1999)	Malaysia	Hospital isolates, n=390 SA	39.7% (155/390)	96.4% S (3.6% R)	96.7% S (3.3% R)	Fusidic & rifampin remain >95% active

2. Epidemiology of MRSA in Wound Infections

MRSA is a major cause of hospital-acquired infections and is frequently isolated from superficial wound infections. It is responsible for various conditions such as cellulitis, impetigo, abscesses, and infected surgical wounds. Studies indicate that MRSA infections are increasingly reported in both hospital and community settings. MRSA has become a dominant pathogen in skin and soft tissue infections worldwide, contributing significantly to morbidity and healthcare costs. The prevalence of MRSA varies across regions and healthcare facilities. Factors contributing to its spread include prolonged hospitalization, indiscriminate antibiotic use, invasive procedures, and poor infection control practices.

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3. Bacteriological Profile of Superficial Wound Infections

The bacteriological profile of superficial wound infections usually includes both Gram-positive and

Gram-negative bacteria. Common pathogens isolated from wound infections include:

- *Staphylococcus aureus*
- Methicillin-Resistant *Staphylococcus aureus* (MRSA)
- *Streptococcus pyogenes*
- *Pseudomonas aeruginosa*
- *Escherichia coli*
- *Klebsiella pneumoniae*

Among these organisms, *Staphylococcus aureus* is the most commonly isolated pathogen. MRSA strains have developed resistance mechanisms such as altered penicillin-binding proteins, which reduce the effectiveness of beta-lactam antibiotics. The ability of MRSA to form biofilms on tissues and medical devices further enhances its survival and resistance to antimicrobial therapy [5-6].

4. Fusidic Acid and Its Role in MRSA Treatment

Fusidic acid is a steroidal antibiotic primarily active against Gram-positive bacteria, particularly *Staphylococcus aureus*. It works by inhibiting bacterial protein synthesis through interference with elongation factor-G during translation. Fusidic acid is commonly used in topical and systemic forms for treating skin infections. Clinical studies show that many MRSA isolates remain susceptible to fusidic acid, making it an effective option for treating superficial infections.

However, resistance may develop when fusidic acid is used alone for prolonged periods. Some studies have shown that treatment with fusidic acid monotherapy may lead to the emergence of resistant MRSA strains. Therefore, fusidic acid is often recommended in combination with other antibiotics to reduce resistance[7].

5. Rifampicin and Its Antibacterial Activity

Rifampin is a bactericidal rifamycin that inhibits bacterial RNA polymerase (rpoB). It penetrates well into tissues and cells, making it valuable for complicated staph infections. In vitro, MRSA is usually highly rifampin-susceptible (MICs often <0.06 µg/mL). In the Malaysian survey, only ~3.3% of *S. aureus* were rifampin-resistant, and the Greek series reported 0% rifampin resistance.

However, rifampin resistance emerges extremely rapidly during therapy. Single-step rpoB mutations can confer high-level resistance. Clinical guidelines universally forbid rifampin monotherapy in MRSA because of this risk. Consequently, rifampin is always used in combination (e.g. with fusidic acid, vancomycin, linezolid) for device-associated or deep infections. Resistance rates in surveillance data are often low because rifampin is reserved for special cases, but studies have documented high resistance in heavily pre-treated settings. For example, a Nigerian cohort found 56.3% of MRSA nasal isolates resistant to rifampin, reflecting selection pressure.

Rifampin resistance in MRSA is typically due to rpoB point mutations (e.g. S464P, H481N/Y). Unlike FA, there is no known transferable plasmid-mediated rifampin resistance in *S. aureus*. Cross-resistance to other drugs is not an issue, but because rifampin is also used in TB therapy, resistance testing in MRSA is standard practice

Rifampicin is a broad-spectrum antibiotic that inhibits bacterial RNA synthesis by binding to the DNA-dependent RNA polymerase enzyme. It has excellent activity against *Staphylococcus aureus*, including MRSA strains. Due to its strong bactericidal activity and ability to penetrate biofilms, rifampicin is frequently used in combination therapy for chronic infections and device-related infections. Studies have demonstrated that rifampicin combined with fusidic acid can significantly reduce MRSA colonization and infection rates in clinical settings. However, rifampicin should not be used alone because rapid resistance can develop8[8-9].

6. Synergistic Effect of Fusidic Acid and Rifampicin

Combination therapy with fusidic acid and rifampicin has shown synergistic antibacterial effects against MRSA. This combination improves treatment outcomes by:

- Enhancing antibacterial activity
- Reducing the development of resistance
- Improving penetration into infected tissues
- Increasing bactericidal effectiveness against biofilm-forming MRSA

Research indicates that antibiotic combinations involving fusidic acid and rifampicin demonstrate improved activity against MRSA isolates compared to monotherapy. Therefore, this combination is widely recommended in clinical practice for difficult-to-treat staphylococcal infections[10].

7. Antibiotic Resistance Trends

Antibiotic resistance in MRSA is an increasing global concern. Resistance to commonly used antibiotics such as clindamycin, tetracycline, and cotrimoxazole has been reported in several studies. Although resistance to fusidic acid and rifampicin remains relatively low in many regions, improper usage can lead to resistance development. Therefore, antibiotic stewardship and susceptibility testing are essential for selecting appropriate therapy [11-12]. Understanding the bacteriological profile of wound infections and antibiotic susceptibility patterns helps clinicians select effective treatment regimens. Early identification of MRSA and appropriate antibiotic therapy can reduce complications, hospital stay, and treatment costs.

Healthcare institutions should implement strict infection control measures, antimicrobial stewardship programs, and routine surveillance to prevent the spread of MRSA.

8. Clinical Use and Outcomes of Fusidic Acid and Rifampicin

8.1 Fusidic Acid Use

Topical fusidic acid (cream or ointment) is widely used for impetigo, minor wounds, or folliculitis caused by *S. aureus*. It achieves high local concentrations and minimal systemic side effects. Oral fusidic acid is available in some countries (UK, Australia) and has been used for staph infections (e.g. bone/joint). In MRSA superficial infections, topical FA alone often suffices, with cure rates generally high if susceptibility is confirmed. For example, among MRSA skin infections, topical FA monotherapy led to clinical improvement in 70–90% of patients in small series (references were not given in sources found; further controlled trials are lacking). However, due to rising resistance in some areas, culture and susceptibility testing are important before relying on FA monotherapy[13].

Systemic FA (oral or IV) has been studied mainly in deep or device infections. A recent RCT in prosthetic joint infection (Beck et al. 2023) compared oral fusidic acid plus rifampin vs standard care; outcomes were promising but 26% of relapsing MRSA developed rifampin resistance. No large trials have examined fusidic acid monotherapy for skin MRSA beyond case reports.

8.2 Rifampin Use

Rifampin is used orally or IV (depending on formulation) and indicated in combination therapy for serious staph infections (e.g. osteomyelitis, prosthetic joint infections, deep abscesses). In practice it is not used for isolated simple superficial wounds or cellulitis but may be considered if other agents fail. When used (always with another anti-MRSA drug), rifampin improves bactericidal activity, especially against biofilms. For example, rifampin plus fusidic acid has been used in osteomyelitis or foreign-body infections, and rifampin plus vancomycin is standard for prosthetic joint MRSA (IDSA 2013 guidelines).

There are few data on rifampin specifically in skin MRSA. Some reports of skin CA-MRSA treatment (usually clindamycin, TMP/SMX or doxycycline) include rifampin as an adjunct for complications. But rifampin doses vary; common regimens are 600–900 mg/day (divided). No studies directly compare rifampin regimens for superficial MRSA. Toxicity (hepatotoxicity) and drug interactions (induces CYP450) limit rifampin use. Clinical outcomes are best when rifampin is given for ≥10–14 days in combination, but no standard exists for skin infections[13].

8.3. Synergy and Resistance Emergence

In vitro and some clinical data support synergy between fusidic acid and rifampin against MRSA. Checkerboard and time-kill studies show that FA+rip often yield greater killing than either alone. In biofilm models, rifampin markedly enhances FA's activity and vice versa. French and IDSA experts note that FA+rip is an effective combination, particularly for fluoroquinolone-resistant staphylococci. The rationale is complementary mechanisms: FA inhibits elongation factor, rifampin inhibits RNA polymerase. Clinically, combination therapy may allow higher cure rates and slower resistance selection. For example, a series of prosthetic joint infections treated with FA+rip had good outcomes, though some still relapsed with rifampin-resistant MRSA[14-15].

The major caution is rifampin resistance. Any rifampin monotherapy almost inevitably selects rpoB mutants quickly. Even in combination, inadequate dosing or poor adherence can lead to breakthrough resistance. FA resistance can also emerge if FA is used alone,

especially topically over large areas. Thus, guidelines recommend against FA monotherapy for widespread infection. If rifampin is used, it must be with at least one other active drug (e.g. fusidic acid or vancomycin). Clinical trials of combination therapy are few; most data come from device infections. Stewardship reports indicate that rifampin resistance arose in ~10–20% of MRSA when rifampin was improperly dosed or not paired. In summary, FA+rip combos are often synergistic, but both drugs must be used carefully to prevent resistance.

9. Conclusion

Superficial wound infections remain a major clinical challenge, particularly with the increasing prevalence of MRSA. The bacteriological profile of these infections commonly includes *Staphylococcus aureus*, with MRSA being a dominant pathogen. Fusidic acid and rifampicin play a significant role in the treatment of MRSA infections due to their strong anti-staphylococcal activity. However, monotherapy may lead to resistance, and combination therapy is often recommended for better clinical outcomes. Continuous surveillance of antibiotic susceptibility patterns and rational use of antibiotics are essential for controlling MRSA infections and improving patient care.

MRSA is a major pathogen in superficial wound infections worldwide. Control requires awareness of its epidemiology, co-pathogens, and resistance mechanisms. Fusidic acid and rifampicin remain valuable in therapy: fusidic acid is often effective topically or systemically, while rifampin is useful only in combination therapy. Clinicians should obtain cultures for MRSA and perform susceptibility testing for fusidic acid and rifampin. Empiric use of FA should be guided by local resistance rates; rifampin should never be used alone. When MRSA is confirmed, consider combination therapy (e.g. fusidic acid plus rifampin) in complicated cases, monitoring for adverse effects. Strict infection-control (contact precautions) and antibiotic stewardship (avoid inappropriate FA use, restrict rifampin to combined regimens) are essential. Finally, further research should fill gaps in clinical outcomes for fusidic/rifampin regimens and track emerging resistance.

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