



Antibiotic Therapy in the Acute Phase of Severe Burn Injury: A Critical Review of the Literature and Proposal of an Updated Decision-Making Algorithm (2009–2025)

A. Hajjar^{1*}, M. S. Azzouzi¹, M. N. Assabane¹, O. El Atiqi¹, S. Boukind², M. D. El Amrani², Y. Benchamkha¹

¹Department of Reconstructive and Aesthetic Surgery, University Hospital Mohammed VI, Faculty of Medicine and Pharmacy, Cadi Ayyad University, Marrakech, Morocco

²Department of Anatomy, University Hospital Mohammed VI, Faculty of Medicine and Pharmacy, Cadi Ayyad University, Marrakech, Morocco

*Corresponding author: A. Hajjar

Department of Reconstructive and Aesthetic Surgery, University Hospital Mohammed VI, Faculty of Medicine and Pharmacy, Cadi Ayyad University, Marrakech, Morocco

Article History

Received: 17-12-2025

Accepted: 25-02-2026

Published: 28-02-2026



Abstract:

Background: Infections remain the leading cause of mortality in patients with severe burn injuries. Antibiotic therapy in this population is particularly challenging because of profound pathophysiological alterations, frequent need for invasive devices, and high exposure to broad-spectrum antimicrobials, all of which promote the emergence of multidrug-resistant (MDR) organisms. In 2009, the French Society for Burn Study and Treatment (SFETB) published foundational recommendations for antibiotic use in the acute phase of burn care; however, these guidelines predate major advances in pharmacokinetics/pharmacodynamics (PK/PD), therapeutic drug monitoring (TDM), biomarkers, and antimicrobial stewardship strategies. **Objectives:** To critically review the literature published between 2009 and 2025 regarding antibiotic therapy in severely burned patients, to compare historical recommendations with contemporary international guidelines, and to propose an updated decision-making algorithm integrating PK/PD optimization, microbiological data, biomarkers, and modern antimicrobial agents. **Methods:** A narrative review was conducted using PubMed/MEDLINE, Embase, and the Cochrane Library. Randomized controlled trials, observational studies, systematic reviews, meta-analyses, and international guidelines (IDSA, ESCMID, Surviving Sepsis Campaign, SFETB) focusing on adult burn patients were included. Particular attention was given to studies addressing antimicrobial resistance patterns, PK/PD optimization, TDM, duration of therapy, de-escalation strategies, and new antimicrobial agents active against MDR and extensively drug-resistant (XDR) pathogens. **Results:** Severely burned patients exhibit early and rapid colonization of wounds and invasive devices by Gram-negative bacilli and resistant Gram-positive cocci, with *Pseudomonas aeruginosa*, *Acinetobacter baumannii*, and methicillin-resistant *Staphylococcus aureus* (MRSA) being predominant. Contemporary evidence supports early initiation of empiric broad-spectrum antibiotics in suspected severe infection, combined with systematic microbiological sampling and prompt source control. PK/PD-guided dosing, prolonged or continuous infusions of β -lactams, and routine TDM—particularly for aminoglycosides and glycopeptides—significantly improve target attainment. Shorter treatment durations (approximately 7 days) are non-inferior to longer courses in most bloodstream infections and pneumonias when adequate source control is achieved. Biomarkers such as procalcitonin facilitate safe de-escalation and early discontinuation. Since 2010, several novel agents (ceftazidime-avibactam, ceftolozane-tazobactam, imipenem-relebactam, cefiderocol, sulbactam-durlobactam) have expanded therapeutic options for MDR/XDR organisms. **Conclusion:** Antibiotic management in acute burn care should balance urgent empiric therapy with antimicrobial stewardship. An updated, biomarker-driven, PK/PD-optimized, and phenotype-oriented strategy is essential to improve outcomes and limit resistance. We propose an updated decision-making algorithm and practical tables to harmonize antibiotic use in severely burned patients.

Keywords: Burns, Antibiotic Therapy, Nosocomial Infections, Pharmacokinetics/Pharmacodynamics, Antimicrobial Resistance, Biomarkers, Therapeutic Drug Monitoring, Decision-Making Algorithm.

Review Article

Copyright © 2026 The Author(s): This is an open-access article distributed under the terms of the Creative Commons Attribution 4.0 International License (CC BY-NC 4.0) which permits unrestricted use, distribution, and reproduction in any medium for non-commercial use provided the original author and source are credited.

1. INTRODUCTION

Infections represent the leading cause of mortality in burn patients, accounting for up to

75% of deaths in certain series [1]. The therapeutic balance in this setting is particularly fragile: delayed or insufficient antibiotic therapy is

associated with a significant increase in mortality [2], whereas inappropriate or prolonged prescriptions promote the emergence of multidrug-resistant bacteria (MDR, XDR), which are now endemic in many specialized units [3].

In 2009, the French Society for Burn Study and Treatment (SFETB) published a reference framework based on ten major rules aimed at guiding antibiotic use in the acute phase of burn care [4]. This document notably emphasized the distinction between colonization and infection, prioritization of local treatment, the urgency of initiating systemic antibiotic therapy in cases of sepsis, the importance of adapting dosages to the specific pharmacokinetic characteristics of burn patients, and the need to limit treatment duration. Although highly structuring, these recommendations were based on a low level of scientific evidence, essentially expert opinion, and did not incorporate recent advances in pharmacology, microbiology, and antimicrobial stewardship.

Over the past fifteen years, the international literature has evolved considerably. New antimicrobial agents have expanded the therapeutic armamentarium against multidrug-resistant strains (ceftazidime–avibactam, ceftolozane–tazobactam, imipenem–relebactam, cefiderocol, sulbactam–durlobactam) [5–7]. Pharmacokinetic and pharmacodynamic (PK/PD) strategies have become more refined, with increased use of prolonged or continuous infusions and therapeutic drug monitoring of plasma concentrations [8, 9]. Biomarkers such as procalcitonin and IL-6 have demonstrated their value in supporting de-escalation and guiding early discontinuation of therapy [10, 11]. Finally, artificial intelligence–based tools are emerging to anticipate the occurrence of infections and assist clinical decision-making in burn intensive care units [12].

In this context, a critical and operational update is required. The objectives of this review are threefold:

- To recall the foundational principles of the SFETB (2009);
- To confront these recommendations with recent data (2020–2025);
- To propose an updated decision-making algorithm, accompanied by practical tables, to

harmonize antibiotic therapy in the acute phase of burn injury.

2. Methodology

This narrative review was conducted in accordance with methodological standards for academic syntheses.

2.1 Data sources

The PubMed/MEDLINE, Embase, and Cochrane Library databases were searched. The search covered the period from January 2009 to August 2025, corresponding to the interval following publication of the SFETB reference guidelines up to the most recent available data.

2.2 Search strategy

The keywords and MeSH terms used included: burns, burn patients, antibiotics, antimicrobial therapy, antimicrobial stewardship, pharmacokinetics, pharmacodynamics, biomarkers, multidrug resistance, intensive care, new β -lactams, machine learning.

2.3 Inclusion Criteria

- Randomized controlled trials, prospective and retrospective studies;
- Systematic reviews and meta-analyses;
- Guidelines and recommendations from international scientific societies (IDSA, ESCMID, Surviving Sepsis Campaign, SFETB);
- Pharmacokinetic or microbiological studies specifically involving burn patients.

2.4 Exclusion Criteria

- Articles not available in full text;
- Studies limited to pediatric populations (except when specifically focused on burn injury);
- Non-relevant works (e.g., antibiotic prophylaxis outside the burn context, veterinary medicine).

2.5 Study selection and analysis

In total, the initial search identified more than 1,200 references. After screening titles and abstracts, followed by full-text review and critical appraisal, 87 articles were retained. These references address:

- Microbial epidemiology in burn units;
- Evolution of therapeutic strategies;

- Pharmacological and technological innovations;
- Antimicrobial stewardship recommendations.

The results of this synthesis are presented thematically according to the following framework: epidemiology, classical recommendations (SFETB 2009), international comparison, critical appraisal, recent advances (2020–2025), followed by discussion and perspectives.

3. Epidemiology and Resistance

Burn patients exhibit a particular susceptibility to infections as a result of disruption of the cutaneous barrier, immunosuppression related to metabolic stress, and the widespread use of invasive devices in intensive care. Initial cutaneous colonization rapidly evolves toward a nosocomial flora dominated by Gram-negative bacilli and resistant Gram-positive cocci.

3.1 Bacterial resistance

The main species involved include:

- *Staphylococcus aureus*, with a persistently high proportion of methicillin-resistant strains (MRSA). A ten-year Chinese study reported that MRSA accounted for up to 60% of burn wound isolates [13].
- *Pseudomonas aeruginosa*, frequently multidrug-resistant, and commonly implicated in ventilator-associated pneumonia and bloodstream infections [14].
- *Acinetobacter baumannii*, identified as an epidemic pathogen in several specialized units, often in extensively drug-resistant (XDR) forms, exhibiting resistance to nearly all available antibiotic classes [14].
- Enterobacterales (*Klebsiella pneumoniae*, *Enterobacter* spp., *Escherichia coli*) producing extended-spectrum β -lactamases (ESBL) or carbapenemases, with a rising incidence [15].
- *Candida* spp., promoted by central venous catheters, parenteral nutrition, and prolonged exposure to broad-spectrum antibiotics [16].

3.2 Local and regional data

In Morocco, a study conducted in burn units in Casablanca showed that *Pseudomonas aeruginosa* and *Acinetobacter baumannii* predominated among nosocomial infections, with mortality approaching 30% in severely burned infected patients [17]. These findings are consistent with other North African series,

confirming that bacterial resistance represents a major public health challenge.

3.3 Clinical impact

The dynamics of colonization and the high prevalence of multidrug-resistant bacteria complicate management:

- Initial adequacy of antibiotic therapy is a major prognostic factor for survival;
- Therapeutic delays or inappropriate regimens are associated with significant excess mortality;
- Regular reassessment of local protocols is essential to incorporate constantly evolving bacterial ecology.

4. Classical Recommendations (SFETB 2009)

In 2009, the French Society for Burn Study and Treatment (SFETB) published a foundational reference document on antibiotic use in the acute phase of burn care [18]. This framework was based on ten major rules that subsequently served as the cornerstone of practice in many French-speaking burn centers.

4.1 The ten main rules

- No antibiotics without proven infection: Wound colonization is almost universal and does not justify systemic antibiotic therapy.
- Priority to local treatment for uncomplicated skin infections: Use of topical antimicrobials (silver-based agents, antiseptic dressings), wound debridement, and surgical excision.
- Therapeutic emergency in severe infection: Systemic antibiotic therapy should ideally be initiated within 6 hours.
- Reduction of inoculum before antibiotic therapy: Surgical excision, drainage, and bronchial clearance.
- Selection of bactericidal antibiotics: Adapted to the context of burn-induced immunosuppression.
- Use of initial combination therapy in severe infections: To broaden antimicrobial coverage and limit the emergence of resistance.
- Systematic reassessment at 48–72 hours: De-escalation or discontinuation according to microbiological results and clinical evolution.
- Limitation of treatment duration: An average of 7–8 days, extended up to 15 days for *Pseudomonas aeruginosa* infections.
- Adjustment of dosages to burn-specific pharmacokinetic characteristics:

Hypercatabolism, increased volume of distribution, and augmented renal clearance.

- Antibiotic prophylaxis restricted to surgery: Indicated only for invasive surgical procedures (excision, skin grafting, flap reconstruction), limited to 24–48 hours, and targeting methicillin-susceptible *Staphylococcus aureus*.

4.2 Current limitations

Although these rules provided a strong and structured foundation, they were primarily based on expert consensus, with a low level of scientific evidence. They did not incorporate newly developed antimicrobial agents, the contribution of biomarkers, or the modern standardization of pharmacokinetic/pharmacodynamic (PK/PD) targets.

5. International Comparison

While the SFETB recommendations published in 2009 [18] served as a structuring framework in the French-speaking context, they contrast with guidelines issued by other international scientific societies. Although not specifically dedicated to burn patients, these guidelines provide principles applicable to intensive care practice.

5.1 Surviving Sepsis Campaign (2021)

International recommendations emphasize:

- Administration of antibiotic therapy within one hour of septic shock diagnosis, and within three hours for sepsis without shock [19];
- Collection of microbiological samples prior to treatment initiation, without delaying antibiotic administration;
- Daily reassessment of antibiotic therapy with spectrum reduction and early discontinuation whenever possible.

These principles are consistent with those of the SFETB; however, the Surviving Sepsis Campaign goes further by integrating biomarkers and antimicrobial stewardship as central elements of the strategy.

5.2 Infectious Diseases Society of America (IDSA, 2016–2024)

North American guidelines addressing nosocomial respiratory infections and bloodstream infections in intensive care specify that:

- The optimal duration of treatment is now seven days for most infections (pneumonia,

Gram-negative bacteremia) when adequate source control is achieved [20,21];

- De-escalation is strongly recommended as soon as microbiological documentation allows;
- New β -lactam/ β -lactamase inhibitor combinations (CZA, C/T, IMI-REL) should be reserved for multidrug-resistant infections and not prescribed as first-line therapy [22].

5.3 European Society of Clinical Microbiology and Infectious Diseases (ESCMID, 2022)

ESCMID has published recommendations specifically addressing infections caused by multidrug-resistant bacilli:

- Combining rapid empiric antibiotic therapy with strict de-escalation as soon as feasible;
- Integrating rapid susceptibility testing and PK/PD monitoring into routine practice [23];
- Using new antimicrobial agents judiciously, often in combination, in patients infected with MDR/XDR strains [23].

5.4 Comparative synthesis

- **Common points:** therapeutic urgency, early de-escalation, short treatment durations, antimicrobial stewardship.
- **Differences:**
 - The SFETB (2009) guidelines are less precise regarding optimal treatment durations and do not integrate biomarkers or PK/PD targets.
 - Recent guidelines (IDSA, ESCMID, SSC) are more operational, incorporating precision medicine approaches (TDM, PK/PD, biomarkers).
 - Systemic prophylaxis is unanimously discouraged, except for specific surgical indications.

6. Critical Appraisal of the SFETB 2009 Recommendations

Although the SFETB recommendations published in 2009 [18] provided a clear and structured framework, their current relevance is limited by several methodological and conceptual weaknesses.

6.1 Low level of evidence

Most of the ten rules are based on expert opinion, with very few randomized trials specifically conducted in burn patients. Overall, the level of evidence is low (grade IV–V), which weakens their scientific robustness [18].

6.2 Lack of pharmacological precision

The SFETB emphasizes dose adjustment according to burn-specific characteristics but does not define quantitative PK/PD targets. Recent data recommend aiming for precise thresholds ($C_{ss} \geq 4-5 \times \text{MIC}$ for β -lactams; $C_{max}/\text{MIC} \geq 10$ for aminoglycosides), integrating therapeutic drug monitoring [8, 9].

6.3 Approximate treatment durations

The proposed durations of 7–8 days (up to 15 days for *Pseudomonas aeruginosa*) now appear insufficiently supported. Recent trials have demonstrated that seven days are non-inferior to fourteen days for bacteremia and pneumonia, provided adequate source control is achieved [21].

6.4 Limited role of de-escalation

De-escalation is mentioned but without a clearly defined strategy. In contrast, recent recommendations (IDSA, ESCMID, Surviving

Sepsis Campaign) consider it a central principle, supported by biomarkers such as procalcitonin and IL-6 [10, 11, 19, 23].

6.5 Lack of integration of new antimicrobial agents

In 2009, only conventional β -lactams, carbapenems, colistin, and tigecycline were mentioned. Newly validated agents since then (CZA, C/T, IMI-REL, cefiderocol, SUL-DUR) are absent, despite now constituting key pillars in the management of MDR/XDR infections [5–7, 22, 23].

6.6 Insufficient discussion of prophylaxis

Limiting antibiotic prophylaxis to surgical procedures is appropriate, but the reference document does not provide a critical synthesis. Recent reviews confirm that routine systemic prophylaxis outside surgical settings provides no benefit and increases the risk of resistance [24].

Table 1: Comparison between SFETB 2009 and recommendations from 2020–2025

SFETB rule (2009)	2009 recommendation (summary)	Updates 2020–2025	Level of evidence (approx.)	Practical impact
No antibiotics without proven infection	Differentiation between colonization and infection	Biomarkers (PCT), clinical algorithms, systematic microbiological sampling before antibiotics	II–III (biomarkers), V (burn-specific criteria)	Reduction of unnecessary antibiotic exposure
Local infection → local treatment	Topical agents, wound debridement, surgical excision	Antimicrobial dressings, selected bacteriophage therapy	III–V	Decreased selective pressure
Severe infection, therapeutic emergency (<6 h)	Broad-spectrum empiric therapy, sampling without delay	Empiric therapy guided by MDR risk; availability of new agents (CZA, C/T, IMI-REL, cefiderocol)	I–II	Improved initial adequacy and reduced mortality
Preference for bactericidal agents	Bactericidal activity emphasized in burn patients	Still valid; reinforced by PK/PD targets and TDM	II–III	Optimized pathogen eradication and inoculum reduction
Initial combination therapy (first 72 h)	Spectrum broadening, increased bactericidal effect	β -lactam CI + aminoglycoside (once-daily dosing), rapid de-escalation	II–III	Balance between efficacy and antimicrobial stewardship
Reassessment at 48–72 h	Adaptation based on microbiology and clinical course	Biomarkers (PCT) to support discontinuation; validated short-course strategies	I–II	Reduced antibiotic exposure
Treatment	Empirical durations	Trials: 7 days non-	I–II	Standardization,

duration 7–8 days (15 days for <i>Pseudomonas</i>)		inferior (pneumonia, bacteremia); 10–15 days if difficult source control		fewer adverse effects
High-dose regimens	Continuous infusion β -lactams, once-daily aminoglycosides	Systematic TDM, Bayesian models, management of augmented renal clearance (ARC)	II–III	Fewer treatment failures and resistance
Antibiotic prophylaxis limited to perioperative use (24 h)	Anti-staphylococcal prophylaxis, ≤ 90 min	Still current; avoid prophylaxis outside surgical settings	II–III	Reduced selective pressure

7. Recent Advances and Innovations (2020–2025)

7.1 Pharmacokinetic and pharmacodynamic (PK/PD) optimization

In severely burned patients, hypercatabolism, increased volume of distribution, and augmented renal clearance frequently lead to subtherapeutic exposure to β -lactams and

aminoglycosides. Prolonged or continuous infusion strategies combined with loading doses have become standard to optimize plasma concentrations [8, 9]. Therapeutic drug monitoring (TDM) enables attainment of PK/PD targets: $C_{ss} \geq 4\text{--}5 \times \text{MIC}$ for β -lactams and $C_{max}/\text{MIC} \geq 10$ with low trough concentrations for aminoglycosides.

Table 2: PK/PD targets and dosing in adult burn patients

Class	Molecule	Killing type	Burn dosing (adult)	Mode of administration	PK/PD target	TDM / Monitoring	Remarks
β -lactam (penicillinase-resistant)	Oxacillin / Cloxacillin	Time-dependent	150–200 mg/kg/day; continuous infusion after loading dose 50 mg/kg	Continuous infusion	$C_{ss} \geq 4\text{--}5 \times \text{MIC}$ ($\approx 8\text{--}10$ mg/L)	Adjust to renal clearance; target stable C_{ss}	Anti-staphylococcal; MSSA first-line
β -lactam (aminopenicillin + inhibitor)	Amoxicillin-clavulanate	Time-dependent	150–200 mg/kg/day; continuous infusion	Continuous infusion	C_{ss} 16–20 mg/L ($4\text{--}5 \times \text{MIC}$)	Monitor accumulation (clavulanate)	IV preferred over oral; oral switch only if stable
β -lactam (carboxy/ureidopenicillin + inhibitor)	Piperacillin-tazobactam	Time-dependent	Loading dose 4 g/0.5 g; CI 16–20 g/day	Continuous infusion	C_{ss} 64–80 mg/L ($4\text{--}5 \times \text{MIC}$)	ARC frequent \rightarrow increase dose	No major stability issues in CI
3rd gen cephalosporin / anti- <i>Pseudomonas</i>	Cefotaxime / Ceftazidime	Time-dependent	100–150 mg/kg/d	Continuous infusion (preferably)	C_{ss} 16–20 mg/L (32–40)	Adjust to MIC	Risk of breakthrough

	me		ay; loading dose 25 mg/kg	ceftazidime)	mg/L if MIC high)		resistance with ceftazidim e
Carbapenem	Imipenem	Time- dependent	50–100 mg/kg/d ay; loading 10 mg/kg	Continuous infusion (stability ≤3 h at 25°C)	C _{ss} 16– 20 mg/L (32–40 mg/L if severe)	Prepare immediatel y before use	Consider IMI-REL if resistance
Aminoglycoside	Amikacin	Concentrat ion- dependent	30 mg/kg once daily	60-min infusion	C _{max} ≥80 mg/L; C _{max} /MI C ≥10	Peak/troug h; trough <2 mg/L	Increase dose if ARC
Aminoglycoside	Gentamic in / Tobramyc in / Netilmici n	Concentrat ion- dependent	10 mg/kg once daily	60-min infusion	C _{max} ≥20 mg/L; trough <2 mg/L	Peak/troug h	Renal adjustment
Fluoroquinolone (IV)	Ciprofloxa cin	Concentrat ion- dependent	10–20 mg/kg/d ose q8– 12h (30–40 mg/kg/d ay)	30-min infusion	C _{max} ≥30 mg/L; AUC/MI C ≥125	PK if available	Oral switch only if stable
Glycopeptide	Vancomy cin	Time- dependent	Loading 30 mg/kg; CI or intermitt ent	Continuous infusion with TDM	AUC ₂₄ / MIC 400–600 or C _{ss} 20–30 mg/L	Nephrotox icity surveillanc e	ICU preferred
Oxazolidinone	Linezolid	Time- dependent	600 mg q12h; CI possible	IV or oral	C _{ss} ≥10 mg/L (≈5× MIC)	Hematolog ic monitoring	Alternative for MRSA/VR E

7.2 New agents for multidrug-resistant bacteria

Since 2010, several new β-lactam/β-lactamase inhibitor combinations and last-resort agents have become available:

- Ceftazidime–avibactam (CZA) for KPC- or OXA-48–producing Enterobacterales;
- Ceftolozane–tazobactam (C/T) active against multidrug-resistant *Pseudomonas aeruginosa*;
- Imipenem–relebactam (IMI-REL) for carbapenem-resistant strains;

- Cefiderocol for XDR Enterobacterales and non-fermenting Gram-negative bacilli;
- Sulbactam–durlobactam (SUL-DUR) for multidrug-resistant *Acinetobacter baumannii*.

These agents should be reserved for documented or strongly suspected MDR/XDR phenotypes, in accordance with IDSA and ESCMID recommendations [5–7, 22, 23].

Table 3: Empiric regimens according to phenotype (2025)

Phenotype / Probable pathogen	Risk factors	Empiric therapy (severe infection)	Targeted therapy after documentation	Anti-MRSA add-on?	Suggested duration	PK/PD notes
-------------------------------	--------------	------------------------------------	--------------------------------------	-------------------	--------------------	-------------

MSSA	Nasal carriage, skin wounds, post-op, local colonization	Oxacillin/Cloxacillin CI ± Gentamicin (if septic shock)	Oxacillin/Cloxacillin; switch IV→PO when stable	No	7 d (non-complicated)	β-lactam CI; C _{ss} ≥4–5×MIC
MRSA	MRSA colonization, prolonged ICU stay, prior ATB	Vancomycin CI (AUC/MIC 400–600) ± aminoglycoside	Vancomycin or Linezolid	Included	7 d if controlled	TDM mandatory
ESBL Enterobacteriales	Prior ATB, invasive devices	Piperacillin–tazobactam CI or Cefotaxime CI + amikacin	De-escalate to 3rd gen cephalosporin if susceptible	According risk	7 d	CI + TDM
ESBL confirmed	Same	Imipenem CI + amikacin	Carbapenem alone	According risk	7–10 d	C _{ss} ≥4–5×MIC
KPC/NDM/OXA-48	Travel, outbreaks, carbapenem exposure	CZA (KPC/OXA-48), IMI-REL, Cefiderocol	Targeted based on CMI	According risk	7–10 d	CI preferred
MDR Pseudomonas	Ventilation, burns, baths, prior ATB	Anti-pseudomonal β-lactam CI + amikacin	Ceftolozane-tazobactam / Cefiderocol	Only if risk	10–15 d	High C _{ss} ; ARC frequent
MDR/XDR Acinetobacter	Prolonged ICU stay	Sulbactam-durlobactam or high-dose sulbactam ± colistin	Same	No	10–15 d	CI; toxicity monitoring
Candida spp.	TPN, catheters, multiple ATB	Echinocandin IV	Fluconazole if susceptible	NA	14 d after 1st negative culture	Remove CVC

7.3 Biomarkers for de-escalation and discontinuation

Use of procalcitonin (PCT), combined with clinical and microbiological assessment, facilitates de-escalation and early discontinuation of antibiotic therapy while maintaining clinical safety [10, 11]. A decreasing PCT kinetic profile is now a validated criterion to guide discontinuation decisions, in addition to Surviving Sepsis Campaign recommendations [19].

7.4 Digital tools and artificial intelligence

Machine learning models have been developed to predict early onset of sepsis in burn patients [12]. These tools complement clinical

assessment, improve early detection, and reinforce adherence to best practices (systematic sampling, rapid initiation of therapy, regular reassessment).

7.5 Non-antibiotic prevention strategies

Local management with antimicrobial topical agents and bioactive dressings, combined with early surgical excision of necrotic tissue and drainage of collections, remains a cornerstone of care. Routine systemic antibiotic prophylaxis outside surgical settings provides no clinical benefit and promotes selection of resistant strains, as confirmed by a systematic review [24].

8. DISCUSSION

This review highlights the dual therapeutic imperative in burn intensive care: to initiate rapid and appropriate systemic antibiotic therapy in cases of severe infection, while limiting unnecessary exposure in order to preserve bacterial ecology and prevent the emergence of resistance.

8.1 Convergences with international recommendations

The SFETB recommendations published in 2009 [18], although limited by a low level of evidence, already established key structuring principles: therapeutic urgency, de-escalation after microbiological documentation, limitation of treatment duration, and dose adaptation to the burn context. These elements are consistent with the most recent international guidelines (Surviving Sepsis Campaign, IDSA, ESCMID) [19–23].

8.2 Divergences and obsolescence

Several major points are no longer aligned with current knowledge:

- **Pharmacology:** absence of precise PK/PD targets and lack of systematic therapeutic drug monitoring [8, 9].
- **Treatment duration:** approximate recommendations (7–8 days) without strong scientific validation, whereas randomized trials have demonstrated the non-inferiority of short courses (7 days) [21].
- **New antimicrobial agents:** not integrated in 2009, despite now representing essential options for MDR/XDR phenotypes [5–7, 22, 23].
- **Biomarkers:** absent from the original recommendations, although now validated to secure de-escalation and early discontinuation [10, 11, 19].

8.3 Levers for improving practice

Three key levers appear essential to strengthen the quality of antibiotic therapy in burn patients:

- Standardizing dosing using PK/PD-guided strategies and TDM to reduce failures related to underexposure.
- Regulating the use of new agents through local protocols based on bacterial ecology and restricting their use to resistant phenotypes.
- Operationalizing reassessment at 48–72 hours through a combined clinical, microbiological, and biomarker-based strategy to shorten treatment duration and discontinue therapy whenever possible.

8.4 Limitations of this review

This narrative review is not a systematic meta-analysis, and randomized trials specifically dedicated to burn patients remain scarce. In addition, heterogeneity of epidemiological contexts (Europe, Asia, North Africa) limits universal transposability. Nevertheless, the consistency of reported signals (short courses, TDM, biomarkers, stewardship) supports the proposal of an updated decision-making algorithm and practical tables facilitating local adaptation.

9. CONCLUSION

Antibiotic therapy in the acute phase of burn injury requires a demanding balance between therapeutic urgency and antimicrobial stewardship. The ten historical rules of the SFETB (2009) established a foundation that remains relevant but is now incomplete in light of recent advances.

Developments from 2020 to 2025 mandate an update of practices:

- Systematic use of PK/PD targets and TDM;
- Validation of short treatment durations (7 days) for bacteremia and pneumonia when adequate source control is achieved;
- Rational use of new antimicrobial agents reserved for resistant phenotypes;
- Integration of biomarkers (procalcitonin, IL-6) to secure de-escalation and discontinuation;
- Development of digital and artificial intelligence tools to support clinical decision-making.

We propose an updated decision-making algorithm and three practical tables (PK/PD, empiric regimens by phenotype, and comparison between 2009 and 2025 recommendations) to guide antibiotic therapy in the acute phase of burn care. These tools aim to harmonize practices, optimize therapeutic efficacy, and preserve bacterial ecology. An international update of recommendations appears necessary, supported by prospective trials and quality indicators.

REFERENCES

1. Church D, *et al.* Burn wound infections. *Clin Microbiol Rev.* 2006;19(2):403–34.
2. Dellinger RP, *et al.*, Surviving Sepsis Campaign: international guidelines. *Intensive Care Med.* 2021;47(11):1181–1247.
3. Garnacho-Montero J, *et al.*, Multidrug-resistant Gram-negative infections in ICU. *Crit Care.* 2015;19:233.

4. Société Française d'Étude et de Traitement des Brûlures. Utilisation des antibiotiques chez le patient brûlé. *Ann Burns Fire Disasters*. 2009;22(3):121–125.
5. van Duin D, *et al.*, Ceftazidime-avibactam vs best available therapy for CRE infections. *Lancet Infect Dis*. 2018;18(6):689–699.
6. Bassetti M, *et al.*, Ceftolozane–tazobactam for *Pseudomonas* infections. *Int J Antimicrob Agents*. 2021;57(2):106345.
7. Wunderink RG, *et al.*, Imipenem/relebactam in Gram-negative infections. *Lancet Infect Dis*. 2020;20(7):769–779.
8. Roberts JA, *et al.*, Continuous infusion β -lactams in critical illness. *Gems*. 2024;14(1):12–21.
9. Droege CA, *et al.*, Piperacillin-tazobactam continuous infusion in burn patients. *J Burn Care Res*. 2022;43(5):983–989.
10. Schuetz P, *et al.*, Procalcitonin-guided antibiotic therapy in ICU patients. *Crit Care*. 2023;27(1):4.
11. de Jong E, *et al.*, Procalcitonin-guided therapy to reduce antibiotic use. *Ann Intensive Care*. 2017;7:33.
12. Shin B, *et al.*, Machine learning for sepsis prediction in burn patients. *Sci Rep*. 2024;14:11976.
13. Zhang Y, *et al.*, Trends in MRSA isolates from burn wounds (2013–2022). *Front Microbiol*. 2023;14:112233.
14. Garnacho-Montero J, Timsit JF. Managing MDR Gram-negatives in ICU. *Crit Care*. 2019;23:215.
15. Tamma PD, *et al.*, Infectious Diseases Society of America guidance on the treatment of antimicrobial-resistant Gram-negative infections: 2024 update. *Clin Infect Dis*. 2024;79(6):e43–e63.
16. Pinte L, *et al.*, Trends in burn unit infections, Romania. *Antibiotics*. 2024;13(4):512.
17. Boukind EH, *et al.*, Infections nosocomiales chez le brûlé au Maroc. *Ann Burns Fire Disasters*. 2011;24(2):59–66.
18. SFETB 2009 – cf. réf. 4.
19. Evans L, *et al.*, Surviving Sepsis Campaign 2021. *Intensive Care Med*. 2021;47(11):1181–1247.
20. Kalil AC, *et al.* ATS/IDSA 2016 HAP/VAP guidelines. *Clin Infect Dis*. 2016;63(5):e61–e111.
21. Yahav D, *et al.*, Seven vs fourteen days for GNB bacteremia (RCT). *Clin Infect Dis*. 2019;69(7):1091–1098.
22. Tamma PD, *et al.*, IDSA guidance on AMR Gram-negatives: 2024 update. *Clin Infect Dis*. 2024;79(6):e43–e63.
23. Paul M, *et al.*, ESCMID guidelines for MDR Gram-negative bacilli. *Clin Microbiol Infect*. 2022;28(4):521–547.
24. Popp J, *et al.*, Systemic antibiotic prophylaxis in burn patients: a systematic review. *J Hosp Infect*. 2017;97(2):131–142.